SYLLABUS

VETERINARY PATHOLOGY DEPARTMENT WEDNESDAY SLIDE CONFERENCE 1989-1990

ARMED FORCES INSTITUTE OF PATHOLOGY
WASHINGTON, D.C. 20306-6000
1996

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DEPARTMENT OF VETERINARY PATHOLOGY
WEDNESDAY SLIDE CONFERENCE
1989-1990

129 Microslides
28 Kodachrome slides

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PREFACE

The Registry of Veterinary Pathology, Armed Forces Institute of Pathology, has conducted the Wednesday Slide Conference Program for more than three decades. The cases presented on 30 Wednesdays throughout the academic year are presently distributed to 135 active institutions employing civilian and military veterinary pathologists in the United States, Canada, Germany, Australia, Brazil, France, Northern Ireland, Switzerland, Finland, Israel, Japan, New Zealand, Norway, South Africa, and Taiwan. Throughout each year, histoslides, lantern slides, and histories for selected cases are distributed to the participating institutions and the AFIP's veterinary pathology staff and residents. Subsequent to completed conferences, case diagnoses, comments, and reference listings are forwarded to all participants. Unfortunately, the number of histoslides obtainable from many lesions limits the number of participating institutions.

This study set was assembled in an effort to make the material presented during the Wednesday Slide Conferences available to a wider circle of interested pathologists and other scientists.

This set, composed of 121 cases, 129 microslides, and 28 Kodachrome slides, was assembled from the cases studied during the 1989-1990 conferences. Assembly of this set was not accomplished until 1996; the following index of cases reflects any changes in etiologic agent nomenclature occurring subsequent to the case's original presentation.

We wish to thank each institution for their participation and each case submitter for the permission to use their case(s) in this study set. We also wish to give special thanks to the American Veterinary Medical Association and the American College of Veterinary Pathologists, which are co-sponsors of the Registry of Veterinary Pathology. The C.L. Davis Foundation also provides substantial support for the Registry.

LIST OF SLIDES

K= 35 mm Kodachrome

Slide	Animal	Tissue	Diagnosis / Etiology
1	Dog	Skin Kidney	Greyhound cutaneous and renal vasculopathy
2	Dog	Maxilla	Odontoma
3, K1	Ox	Oral mucosa	Bovine papular stomatitis Bovine parapoxvirus
4	Dog	Lip	Plasmacytoma
5	Dog	Skin	Plasmacytoma
6	Dog	Eye	Uveitis, phacoclastic
7	Kingsnake	Lung	Ophidian paramyxovirus
8	Ferret	Skin	Vitamin E deficiency; Fat necrosis
9	Ox	Brain	*Hemophilus somnus*
10	Cat	Intestine Bone marrow	Feline leukemia virus Feline retrovirus
11	Cat	Liver	Cholangiohepatitis, lymphocytic
12, K2-3	Dog	Kidney	*Leptospira sp.*
13	Cat	Stomach	*Ollulanus tricuspis*
14, K4	Horse	Heart	*Candida tropicalis*
15	Deer	Lung	*Mycobacterium bovis*
16	Cat	Skin	Phaeohyphomycosis *Exophiala jeanselmei*
17	Fish	Skin Muscle	Red spot disease Mycotic dermatitis and myositis
18	Sheep	Brain	Scrapie
19	Horse	Intestine	*Clostridium perfringens*

20	Pig	Kidney	*Salmonella cholerasuis*
21, K5	Horse	Spinal cord	Cauda equina syndrome
22	Dog	Skin	"Normal" Shar pei skin (Dermal mucinosis)
23	Ox	Skin	*Trichophyton verrucosum*
24	Cat	Skin	Xanthomatosis
25	Dog	Skin	Multinodular periadnexal granulomatous dermatitis
26	Dog	Stomach	Adenocarcinoma, gastric
27	Muskrat	Liver	*Cysticercus fasciolaris* *Capillaria hepatica*
28	Dog	Lung	*Pneumocystis carinii*
29, K6	Mouse	Colon	*Citrobacter freundii*
30, K7	Horse	Brain	Nigropalladial encephalomalacia Russian knapweed toxicity
31	Horse	Marrow, femur	Plasma cell myeloma
32	Horse	Pancreas Spleen	Combined immunodeficiency Equine adenovirus
33	Horse	Lung	*Aspergillus sp.*
34	Ox	Liver	Rift Valley fever Phlebovirus
35	Cat	Liver Lymph node	*Cytauxzoon felis*
36	Ovine	Brain	*Acanthamoeba sp.*
37	Horse	Skin	*Dermatophilus congolensis*
38	Dog	Eye	Panuveitis, Vogt-Koyanagi- Harada-like syndrome
39-40	Ox	Liver	*Listeria monocytogenes*
41	Dog	Kidney	*Borrelia burgdorferi* Lyme disease

42, K8	Ox	Muscle	*Cassia occidentalis* toxicity
43-44, K9	Ox	Esophagus Colon	Bovine pestivirus Bovine viral diarrhea virus
45	Goat	Skin	Capripoxvirus (Goatpox virus)
46	Ox	Liver	Sporidesmin toxicity
47	Pig	Spinal cord	Selenium toxicity Poliomyelomalacia
48, K10-11	Baboon	Heart	Encephalomyocarditis virus Cardiovirus
49	Rat	Lung	*Corynebacterium kutscheri*
50, K12	Owl monkey	Liver	*Blastomyces dermatitidis*
51	Rhesus monkey	Lung	*Pneumonyssus simicola*
52	Marmoset	Kidney	Nephritis, chronic interstitial
53	Ox	Brain	*Theileria sp.*
54	Raccoon	Gallbladder	Canine morbillivirus (CDV) *Cryptosporidium sp*
55	Dog	Kidney	Gentamicin toxicity
56, K13	Ox	Brain	*Solanum fastigiatum* toxicity
57	Dog	Kidney	Glomerulonephritis Possible ethylene glycol toxicity
58, K14	Heron	Esophagus	*Eustrongylides sp.*
59	Owl	Liver	Owl herpesvirus
60	Marmoset	Liver	Callitrichid hepatitis, Arenavirus (Lymphocytic choriomeningitis)
61	Dog	Kidney	Snake-bite mesangiolysis
62	Goat	Cord	Copper deficiency
63	Cockatoo	Heart	Avian papovavirus
64	Pig	Heart Lung	*Hemophilus parasuis* Glasser's disease

65	Turkey	Intestine Spleen	Avian adenovirus type II Hemorrhagic enteritis virus
66	Ox	Intestine	Bovine enteric coronavirus
67- 68	Pig	Lung	Pneumonia, foreign body
69	Rhesus monkey	Intestine	*Shigella flexneri*
70	Rat	Liver	Cholangiocarcinoma
71	Chimpanzee	Heart	*Hemophilus influnenzae*
72	Rat	Salivary gland	Cytomegalovirus
73	Rabbit	Liver	Lagomorph calicivirus Viral hemorrhagic disease
74	Minnows	Thyroid	Thyroid hyperplasia (Goiter)
75	Cockatoos	Skin	Psittacine circovirus (Dimunivirus) Psittacine beak & feather disease
76, K15	Cat	Muscle	Muscular dystrophy
77	Rhesus monkey	Lung	Simian lentivirus (SIV) *Pneumocystis carinii*
78, K16	Rat	Kidney	Carcinoma, renal cell
79	Guinea pig	Lung	Cavian adenovirus
80	Guinea pig	Maxilla	Odontoma, ameloblastic
81	Hamster	Kidney	Cadmium toxicity
82	Rat	Lung	CAR bacillus *Mycoplasma pulmonis*
83	Rat	Kidney	Nephropathy, chronic
84	Mouse	Lung	Aspiration pneumonia
85, K17	Cat	Intestine	Granulated round cell tumor
86	Rhesus monkey	Spleen	Simian hemorrhagic fever Simian arterivirus
87	Goat	Lung	Caprine arthritis-encephalitis virus, Caprine lentivirus

88-89	Dog	Bone, Spleen	Myeloproliferative disease
90, K18	Dog	Bone	Hypertrophic osteodystrophy
91, K19-20	Rhesus monkey	Rib	Vitamin C deficiency) Osteodystrophy
92, K21-22	Cat	Bone	Multiple cartilaginous exostosis
93	Guinea pig	Stifle	Osteoarthritis
94	Cat	Brain	GM_2 gangliosidosis
95, K23-24	Frog	Kidney Ovary	Herpesviral adenocarcinoma Microsporidial oophoritis
96	Dog	Liver	Acetaminophen toxicity
97-98	Dog	Lung	*Mycobacterium tuberculosis*
99	Patas monkey	Liver	Herpesvirus simiae (B-virus)
100	Rat	Heart	Endocardial schwannoma
101	Rat	Kidney	Nephroblastoma
102	Goat	Intestine	*Yersinia pseudotuberculosis*
103	Dog	Liver	Cirrhosis
104	Cat	Foot pad	Pododermatitis, lymphoplasmacytic
105, K25	Dog	Skull Lung	Multilobular Osteochondrosarcoma
106, K26	Dog	Ocular muscle	Polymyositis
107	Sheep	Placenta	*Coxiella burnetti*
108-109	Horse	Placenta	*Nocardia sp*
110	Giant panda	Lymph node	Lymphosarcoma
111	Dog	Uterus	Subinvolution of placental sites
112	Horse	Brain	Borna disease
113	Horse	Lung	*Streptococcus zooepidemicus*

114	Horse	Lung	Equine herpesvirus (Rhinopneumonitis)
115	Horse	Kidney	*Halicephalobus deletrix*
116, K27	Pig	Colon	*Balantidium coli* *Surpulina hyodysenteriae*
117	Dog	Brain	Bunyavirus (La Crosse virus)
118	Ox	Skin (Teat)	Poxviral mammillitis
119-120	Dog	Intestine Lymph node	*Neorickettsia helminthoeca*
121	Salmon	Kidney	Renibacteruim salmoninarium
122	Dog	Lung	Malignant histiocytosis
123	Cynomolgus monkey	Liver Lymph node	*Schistosoma sp.*
124, K28	Frog	Liver Kidney	*Aeromonas hydrophila*
125 126	Raccoon	Jejunum Tongue	Herpesvirus *Sarcocystis sp.* *Capillaria sp.*
127	Dog	Lung	Lymphomatoid granulomatosis
128	Ox	Kidney Oral mucosa	Malignant catarrhal fever Bovine gammaherpesvirus
129	Ox	Eye	Malignant catarrhal fever Bovine gammaherpesvirus

COMMENTARY ON SLIDES

MICROSLIDE 1

HISTORY This 2-year-old female greyhound was one of 200 greyhounds of which 8 became ill and 2 died. She was ill for one week with an acute onset of multifocal necrotizing dermatitis that started on the left forepaw and spread to most of both front limbs, the anterior chest and the medial right thigh. The animal became progressively depressed and anorectic and began vomiting three days prior to death. She became icteric the day before she died. She was treated symptomatically with antibiotics, steroids, fluids and Nolvasan scrub baths.

GROSS PATHOLOGY There were large ecchymotic hemorrhages on the upper forelegs, the anterior chest and the right medial thigh. The gallbladder was 4 times normal size. The animal was jaundiced. There were ecchymotic hemorrhages in the lungs. The kidneys were enlarged and covered by petechiae.

LABORATORY RESULTS

WBC 33.7 x 10^3
Segs 87
Bands 1
Lymphs 10
Mono 2
RBC 6.41 x 10^6
Hematocrit 47.9
Hemoglobin 15.9
Platelets adequate

Glucose 111 mg/dl
BUN 302 mg/dl
Creatinine 17.2 mg/dl
Potassium 6.9 mg/dl
Chloride 87 mg/dl
Sodium 159 mEq/L
Total bilirubin 6.7 mg/dl
ALT 424 U/L
AST 976 U/L

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Idiopathic cutaneous and renal vasculopathy of greyhounds.

This disease is presently only observed in greyhounds. The layman's term for this condition is "Alabama crud" or "Greenetrack disease" after the race track in Eutaw, Alabama where the disease was first noted. The skin lesions are described as distinctive. The skin lesion is initially an erythematous and tender cutaneous swelling on the limbs that progresses to a sharply demarcated ulcer. In dogs that survive, healing is slow.

AFIP DIAGNOSIS Haired skin: Dermatitis, chronic-suppurative, diffuse, moderate, with focally extensive ulceration, acute vasculitis and thrombosis, greyhound, canine.

Kidney, glomeruli and afferent arterioles: Glomerulitis/ vasculitis, acute, necrotizing, multifocal, moderate, with congestion, hemorrhage and thrombosis.

Kidney, tubules: Degeneration and necrosis, multifocal, moderate, with mineralization and proteinaceous casts.

CONFERENCE NOTE Conference participants agreed that, based on the breed, the clinical history and the histological lesions, this case most likely represents idiopathic cutaneous and renal glomerular vasculopathy of greyhounds as described by Carpenter, et al. The condition is characterized by hemorrhage, fibrinoid arteritis, thrombosis and infarction with deep ulceration in the skin. Renal lesions are primarily necrosis of the afferent arterioles and glomerular necrosis and thrombosis. Four clinical manifestations of the disease are seen: skin lesions without systemic illness, skin and renal lesions that develop about the same time, skin lesions with delayed onset of renal failure and skin lesions preceded by azotemia. The cause of this disease is unknown.

There was some variation in slides in that the characteristic vascular changes were more pronounced in some skin sections than in others.

CONTRIBUTOR

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REFERENCE

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MICROSLIDE 2

HISTORY This 5-month-old, male, Labrador retriever-cross puppy was presented for a rapidly growing right maxillary mass that was first noted 2 months previously. On clinical exam the mass was 7-8 cm in diameter, bony and nonpainful. Radiographs demonstrated a well-circumscribed, noninfiltrative calcified mass originating around and encompassing the right canine tooth and causing destruction of nasal turbinates and deviation of the nasal septum. The tumor was surgically debulked and the maxilla reconstructed with an implant. After two months the implant was removed. Four months after surgery the dog was in excellent health with no evidence of recurrence.

GROSS PATHOLOGY The surgical specimen consisted of an 8 cm diameter mass that was partially encapsulated by the right maxilla. On cut surface the mass had alternating solid and cystic segments. Some of the cystic spaces contained dense aggregates of small denticles; the solid segments were composed of numerous small mineralized foci embedded within firm, tan, fibrous tissue.

LABORATORY RESULTS Not available.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Compound odontoma, maxilla, dog.

Classification systems for odontomas in domestic animals vary slightly and often have overlapping categories making diagnosis somewhat arbitrary. In this tumor ameloblastic tissue is present but not abundant, and there are areas where odontogenic tissues are arranged in an orderly pattern resembling developing teeth. Additionally, there are cystic spaces that contain denticles, which are characteristic of compound odontomas in most schemas. Odontomas are generally benign tumors, and surgical excision can be curative as in this dog.

AFIP DIAGNOSIS Maxilla: Odontoma, Labrador retriever-cross, canine.

CONFERENCE NOTE Tumors of odontogenic origin are generally classified by the degree of differentiation of their component parts. Ameloblastomas contain epithelial components. Ameloblastic odontomas are predominantly epithelial with foci of dentin, enamel and/or cementum. Complex odontomas are disorderly arrangements of dentin, enamel, pulp, ameloblasts, or odontoblasts. Compound odontomas have similar components but in orderly arrangements. Some consider the well-differentiated odontomas as seen in young animals to be dysplasias or hamartomas rather than true neoplasms.

While all conference participants diagnosed an odontoma, there was considerable discussion concerning classification. Most favored a complex or compound odontoma, although sections varied somewhat and a number of participants felt there was a sufficient ameloblastic component to diagnose ameloblastic odontoma. Many authors comment that separating these neoplasms can be arbitrary; conference participants agreed on this point.

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Walsh, K. M., Denholm, L. J., and Cooper, B. J.: Epithelial odontogenic tumours in domestic animals. Journal of Comparative Pathology, 97: 503-521. 1987.

MICROSLIDE 3, 35 MM SLIDE 1

HISTORY This 8-week-old, female, Holstein calf was one of 17 calves in a 122 cow dairy herd. The calf had a sudden onset of dyspnea and coughing. Tachypnea and harsh lung sounds were evident on clinical examination. Because of the owners wish to establish a diagnosis on which the remaining animals could be treated, the animal was killed by lethal intravenous injection of sodium pentobarbital.

GROSS PATHOLOGY The animal was small for its age and only weighed 130 lbs. The cranioventral portion of both lungs was dark red and firm. The lung lesion involved approximately 5-10% of lung tissue. The lesion submitted was from a solitary, 0.7 cm diameter area of ulceration/erosion present on the dental pad.

LABORATORY RESULTS Pasteurella multocida was cultured from the lungs.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Stomatitis, proliferative, subacute, focal, mild, associated with epithelial cytoplasmic vacuolation and occasional intracytoplasmic inclusions. The morphology of this lesion is consistent with a diagnosis of bovine papular stomatitis.

Electron Microscopy: Many epithelial cells contained intracytoplasmic inclusions filled with oblong parapoxvirus particles measuring 270 x 120 nm.

This case represents a rather mild manifestation of bovine papular stomatitis, a common parapoxvirus disease of calves. The disease is of little clinical importance but has a worldwide incidence and can be a problem in regions where foot and mouth disease is prevalent since it may mimic early stages of this disease. Lesions usually develop on the muzzle, lip margins, buccal mucosa and occasionally esophageal mucosa. Papular lesions may be solitary to multifocal with areas of confluence. The disease is usually brief, often going unnoticed as would likely have been the case in this animal were it not for the development of coincidental pneumonia. In other instances successive crops of lesions may take a course of several months duration. Immunity is of short term duration and animals can be infected repeatedly throughout life. Animals under one year of age are most commonly affected. Bovine papular stomatitis virus shares some antigenic similarity with contagious ecthyma virus of sheep and similar to this latter agent can cause mild cutaneous infection in man.

AFIP DIAGNOSIS Dental pad (per contributor): Stomatitis, lymphohistiocytic, focally extensive, moderate, with ballooning degeneration of epithelial cells and eosinophilic intracytoplasmic inclusions, Holstein, bovine.

CONFERENCE NOTE In addition to foot and mouth disease, as mentioned by the contributor, participants also considered bovine viral diarrhea, infectious bovine rhinotracheitis, malignant catarrhal fever, bluetongue, and rinderpest in the differential diagnosis. The presence of the characteristic intracytoplasmic inclusions allows the specific diagnosis of papular stomatitis.

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REFERENCES

Baker, I. K., and Van Dreumel, A. A.: The alimentary system. In Pathology of Domestic Animals, Volume 2, Edition 3. Jubb, K. V. F., Kennedy, P. C., and Palmer, N., editors. Orlando, Academic Press, pages 90-114. 1985.

Fenner, F., Bachmann, P. A., Gibbs, E. P., Murphy, F. A., Studdert, M. J., and White, D. O.: Veterinary Virology, Orlando, Academic Press, pages 387-405. 1987.

MICROSLIDE 4

HISTORY This 10-year-old female poodle had a mass on the upper lip.

GROSS PATHOLOGY A 4mm x 5mm round mass was surgically removed.

LABORATORY RESULTS Not available.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Plasma cell tumor of the lip.

Plasma cell tumors made up of typical plasma cells have occasionally been seen in canine skin. Recently another group of round cell neoplasms has been shown to be of plasma cell origin. These tumors, previously called reticulum cell sarcoma by many, are represented by this tumor.

Cells are pleomorphic with abundant eosinophilic cytoplasm and distinct borders. Multinucleate cells are seen in all tumors but are especially numerous in this tumor. Cells tend to packet in some areas and may form chains along the edge. Cells having typical plasmacytoid features may be seen along the edges.

Plasma cell tumors occur most commonly in older dogs on the feet, ears, lips and in the oral cavity. Tumors are usually single but may be multiple. Although the degree of cellular differentiation varies markedly, the clinical behavior of nearly all tumors is benign.

Plasma cell tumors must be differentiated from Merkel cell tumors with which they share many features in common including location, age, clinical behavior, and gross and microscopic appearance. Merkel cell tumors have a distinct endocrine pattern, and the cells are basophilic with indistinct cytoplasmic borders. The two tumors can also be differentiated using electron microscopy and possibly immunohistochemistry.

AFIP DIAGNOSIS Haired skin, lip (per contributor): Plasmacytoma, poodle, canine.

CONFERENCE NOTE This tumor was considered poorly differentiated based on the numerous pleomorphic cells and relatively infrequent cells with plasmacytoid differentiation. Immunohistochemical techniques demonstrated the presence of lambda light chains in scattered neoplastic cells.

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REFERENCES

Baer, K. E., Patraik, A. K., Gilbertson, S. R., and Hurwitz, A. I.: Cutaneous plasmacytomas in dogs: A morphologic and immunohistochemical study. Veterinary Pathology, 26: 216-221. 1989.

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MICROSLIDE 5

HISTORY This 6-year-old male cocker spaniel had a mass on the ear.

GROSS PATHOLOGY A 2cm x 3cm firm, white mass was surgically removed.

LABORATORY RESULTS Not available

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Merkel cell tumor (neuroendocrine tumor) of the skin.

The Merkel cell tumor occurs most commonly on the ears, feet, lips and in the oral cavity. Tumors are solitary but may be multiple. Excision is generally curative but occasional recurrence is noted and rarely metastasizes.

Microscopically the Merkel cell tumor has an endocrine pattern of packets of cells separated by a fine fibrovascular stroma. Cells have small to moderate amounts of basophilic to amphophilic cytoplasm with poorly defined borders. Mitoses vary from few to many, and multinucleate cells are seen in most neoplasms.

The Merkel cell tumor must be differentiated from the plasma cell tumor, another recent addition to the group of cutaneous round cell tumors of the dog. These two neoplasms share many features in common including gross and microscopic appearance, location, age, and clinical behavior. The plasma cell tumor lacks a distinct endocrine pattern and the cells have abundant eosinophilic cytoplasm with distinct borders.

AFIP DIAGNOSIS Skin, ear (per contributor): Plasmacytoma, cocker spaniel, canine.

CONFERENCE NOTE While this case has an organoid pattern suggestive of neoplasms described in the literature as Merkel cell tumors, the AFIP diagnosis of plasmacytoma is based on uniform positivity of neoplastic cells for lambda light chains. In a recent study (Kyriazidou, et al), canine plasmacytomas are invariably monoclonal and usually produce lambda light chains.

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AFIP DIAGNOSIS Skin, ear (per contributor): Plasmacytoma, cocker spaniel, canine.

CONFERENCE NOTE While this case has an organoid pattern suggestive of neoplasms described in the literature as Merkel cell tumors, the AFIP diagnosis of plasmacytoma is based on uniform positivity of neoplastic cells for lambda light chains. In a recent study (Kyriazidou, et al), canine plasmacytomas are invariably monoclonal and usually produce lambda light chains. In the experience of the moderator, this case and case IV represent good examples of the variation that can be seen in the canine solitary plasmacytomas. The variation may be a function of differentiation. The moderator believes that, although occasional Merkel cell tumors occur in dogs, most of the canine round cell tumors diagnosed as Merkel cell tumors over the past few years are probably plasmacytomas.

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REFERENCES

Glick, A. D., Holscher, M. A., and Cunshaw, L. D.: Neuroendocrine carcinoma in the skin in a dog. Veterinary Pathology, 20: 761-763. 1983.

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MICROSLIDE 6

HISTORY This 5-year-old, female, mixed breed dog often roamed the desert near the owner's home. Five days before presentation to an ophthalmologist, one eye suddenly became cloudy. According to the owner there was no known history of trauma.

GROSS PATHOLOGY The ophthalmologist's report is as follows: The left eye appeared buphthalmic with markedly engorged episcleral vessels. The cornea was edematous centrally with peripheral neovascularization. Fundoscopic examination was not possible. There was the lack of a consensual reflex from the left to the right eye. The intraocular pressure was 60 mm Hg by application. In addition, periocular cactus spines were noted.

LABORATORY RESULTS The WBC, differential and biochemical profile were within normal limits. The PCV was 37.5.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Phacoclastic uveitis, etiology undetermined.

The epithelium is thin over the central cornea. The central stroma contains a few erythrocytes and neutrophils and is edematous. Near the limbus there is neovascularity and invasion by neutrophils and lymphocytes. The anterior and posterior chambers contain fibrinocellular exudate and hemorrhage. Rupture of the capsule of the cataractous lens is present at anterior polar site. Globules of lens material in the posterior chamber are surrounded by many neutrophils and a few macrophages. The posterior equatorial lens and the vitreal cavity contains exudate similar to that in the anterior chamber. The retina is detached and occurs as a tangled mass in the vitreal cavity.

AFIP DIAGNOSIS Eye: Endophthalmitis, fibrinosuppurative, diffuse, severe, with lens rupture and retinal detachment, mixed breed, canine.

CONFERENCE NOTE The term "phacoclastic uveitis" has been suggested to describe inflammation secondary to the rupture of the lens capsule and release of lens proteins in dogs. Characteristic findings in phacoclastic uveitis are perilenticular inflammation ranging from acute fibrinopurulent to chronic fibrinoproliferative, lymphoplasmacytic iridocyclitis, intralenticular neutrophils, secondary cataracts, and glaucomatous changes. It has been suggested that phacoclastic uveitis results from a breakdown of lens antigen tolerance as a result of the sudden exposure of lens proteins to the immune system.

"Phacolytic uveitis" is a second form of lens induced uveitis, and occurs when lens protein leaks across the intact lens capsule, as in hypermature, resorbing cataracts.

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REFERENCES

Anderson, G. A., and Buyukmihci, N.: Phacoanaphylactic endophthalmitis in an owl. Veterinary Pathology, 20: 776-778. 1983.

McCalla, T. L., Moore, C. P., and Collier, L. L.: Phacoclastic uveitis with secondary glaucoma in a cat. Comp. Anim. Pract., 2: 13-17. 1988.

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Wilcock, B. P., and Peiffer, R. L., Jr.: The pathology of lens-induced uveitis in dog. Veterinary Pathology, 24: 549-553. 1987.

MICROSLIDE 7

HISTORY This adult female king snake was one of 6 of a private collection of 250 snakes that developed vomiting and runny yellow feces over a period of 6 months. Three snakes died.

GROSS PATHOLOGY The trachea, bronchi and mesobronchus contain excessive fluid with foamy to seromucous material noted throughout the mesobronchus. The lung is plum-red, moist, firm and sinks in 10% buffered formalin. The air sac appears unremarkable.

LABORATORY RESULTS Imprints of the lungs show numerous gram negative rods that are sometimes in macrophages. Providencia rettgeri was cultured pure from the lungs and heart blood.

Electron microscopy is pending.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Syndrome, paramyxovirus infection.

Lung, pneumonia, giant cell with inclusion bodies.

Pancreas, pancreatitis, necrotizing, moderate.

Kidney, necrosis, tubular, acute, marked.

Syndrome, septicemia, Providencia rettgeri.

Liver, fatty change, mild.

Multiple sections of lung show hyperplastic changes of the surface epithelium lining septa with loss of basilar nuclear orientation, cytoplasmic swelling and occasional eosinophilic intracytoplasmic inclusion bodies that appear somewhat pleomorphic. Septa are thickened and edematous and air spaces contain masses of heterophils, many of which are degenerating. There are also numerous bacterial rods within the exudate. Occasionally syncytia, presumably from surface alveolar cells, are formed and there are increased numbers of mononuclear cells within the septa.

The splenopancreas shows areas of pancreatic acinar necrosis with accumulations of both heterophils as well as areas of interstitial lymphocytic infiltration. Some of the pancreatic ducts contain masses of degenerating heterophils; the splenic portion is within normal limits. The liver shows mild diffuse fatty change. The kidney shows extensive degeneration and necrosis of the proximal tubules with glomeruli and distal portions of the nephron remaining intact.

The pathologic picture of proliferative pneumonia with syncytia and intracytoplasmic inclusion bodies is characteristic of paramyxovirus infection as described in snakes. Pancreatic lesions have also been described which are present in this case. The kidney lesions are not typical and may be the result of gram negative sepsis which occurred secondarily in this case associated

with Providencia rettgeri which was cultured pure from the lungs and heart blood. Another possibility for the kidney lesion is acute toxicity if any of the aminoglycoside antibiotics were used in this snake.

AFIP DIAGNOSIS Lung: Pneumonia, proliferative, subacute, diffuse, severe, with intracytoplasmic eosinophilic inclusions and syncytial cells, king snake (Lampropeltis getulus), reptilian.

Lung: Pneumonia, heterophilic, multifocal, moderate, with bacilli.

CONFERENCE NOTE The moderator pointed out that even if one is unfamiliar with snake respiratory diseases, the changes seen in this case, including eosinophilic intracytoplasmic inclusions and syncytial cells, are comparable to those of mammalian paramyxoviruses and can lead to a presumptive diagnosis. The moderator and most participants considered the bacterial infection secondary to the viral pneumonia.

Dr. Richard Montali of the National Zoological Park commented that paramyxoviral disease is an emerging problem in snakes of many families and not just Viperidae, as initially reported in the literature.

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REFERENCES

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MICROSLIDE 8

HISTORY This 14-week-old, female, ferret (Mustella putorius furo) was one of 15 rapidly growing farmed kits 13-15 weeks of age that were found dead. Another 35 were depressed and reluctant to move. Diffuse, firm swellings were present beneath the skin; hard subcutaneous lumps were present bilaterally in inguinal areas and affected animals cried in pain when palpated in the lower abdominal region. The diet consisted of 40% squid offal, 20% other fish, 25% slink meat and 15% poultry layer pellets. A commercial vitamin/ mineral supplement was

added to the blended feed.

GROSS PATHOLOGY Subcutaneous and omental adipose tissue was yellow-brown, firm and had a coarse granular texture. The spleen was enlarged and pale.

LABORATORY RESULTS Hemograms from several affected animals revealed a massive neutrophilia with left shift and a mild microcytic, normochromic anemia.

The mean liver vitamin E level in ferrets with steatitis was 14 mg/kg compared to 40 mg/kg in healthy ferrets on another property.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Morphologic Diagnosis: Acute, diffuse, pyogranulomatous steatitis and fat necrosis. Etiology: Dietary deficiency of vitamin E and excess polyunsaturated fat.

Nutritional steatitis has been reported in farmed mink in association with dietary Vitamin E deficiency and polyunsaturated fat (PUFA) excess, but this was the first reported outbreak of the disease in ferrets.

The diet contained 7.7% PUFA (DMB), largely due to the very high levels of PUFA in squid offal (17.9% DM). This presumably triggered the widespread peroxidative damage to adipose tissues in ferrets that had inadequate protective levels of vitamin E.

AFIP DIAGNOSIS Subcutis: Fat necrosis, diffuse, severe, with suppurative and granulomatous steatitis, Highland ferret (Mustella putorius furo), mustelid.

CONFERENCE NOTE A number of attendees commented on the unstained refractile material in adipocytes. The Department of Chemical Pathology identified this material as calcium salts of fatty acids.

The discussion centered on mechanisms of free radical injury and the protective effects of antioxidants.

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REFERENCES

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MICROSLIDE 9

HISTORY This 12-month-old shorthorn steer had a sudden onset of depression, pyrexia, ataxia, and recumbency rapidly followed by death (within 24 hours). It was from a group of 30 feedlot cattle and was the only animal affected.

GROSS PATHOLOGY The brain had distinct, randomly dispersed, 0.5 to 2 cm, dark, friable, red areas of hemorrhage and necrosis scattered throughout the midbrain, rostral brain stem, and both cerebral cortices.

LABORATORY RESULTS Bacterial cultures were negative.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Severe, acute, multifocal to locally extensive, hemorrhagic, fibrinosuppurative meningoencephalitis with necrotizing vasculitis and intralesional bacterial thromboemboli.

Microscopically, the hemorrhagic necrosis within affected brain regions was characterized by well delineated, pale staining areas of the neuropil that had intense hemorrhage and infiltration of neutrophils oriented around blood vessels, and frequent vasculitis and thrombosis. Affected blood vessels had diffuse fibrinoid necrosis of the vessel wall, dense infiltration of neutrophils into the vessel lumen, vessel wall, and perivascular neuropil, and thrombosis. Many thrombi contained small colonies of basophilic gram-negative coccobacilli. There was moderate accumulation of neutrophils and fibrin in the overlying meninges, as well as vasculitis and thrombosis of occasional meningeal blood vessels. Although microbial cultures of the brain were negative, the gross and microscopic lesions, and clinical history were highly suggestive of Hemophilus somnus septicemia (thromboembolic meningoencephalitis (TEME). Acute, multifocal, necrosuppurative lesions with vasculitis were also present in the kidneys and heart.

AFIP DIAGNOSIS Brain: Encephalitis, hemorrhagic and suppurative, multifocal, severe, with vasculitis and associated gram negative bacilli, shorthorn, bovine.

CONFERENCE NOTE Attendees agreed that the lesions and bacterial morphology are consistent with Hemophilus somnus induced meningoencephalitis. Malignant catarrhal fever, sporadic bovine encephalitis, listeriosis, systemic bacterial or mycotic infections and rabies were also discussed.

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REFERENCES

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MICROSLIDE 10

HISTORY This 3-year-old female domestic shorthair cat had a history of ulcerative stomatitis, gingivitis, diarrhea and severe anemia for several months before it died. A feline leukemia virus (FeLV) test was negative one year previously.

GROSS PATHOLOGY Severe anemia, a moderately fatty liver and moderate catarrhal enteritis were present at necropsy.

LABORATORY RESULTS The bone marrow, spleen and intestine were immunohistologically positive for FeLV.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Enteritis, characterized by crypt depletion, crypt epithelium degeneration, villous atrophy and crypt dilatation.

Bone marrow, hypercellular with prominence of immature nuclear forms, lesions consistent with FeLV infection.

The intestine exhibits a diffuse infiltration with mononuclear cells in the mucosa. In parts of the intestine vanishing of crypts, crypt epithelium degeneration, crypt dilatation and villous atrophy are visible. Bone marrow is hypercellular with proliferation of poorly differentiated blasts. Histological alterations are suggestive for FeLV-associated enteritis, which is confirmed by immunohistology. Anemia and fatty liver are also FeLV-associated conditions. Cats with FeLV-associated enteritis are usually older (mean age 2 ½ years) than cats with parvovirus-induced enteritis (mean age
6 months) and have a longer history of disease with intermittent phases of diarrhea. Lymphoid tissues and bone marrow are either normal or hypercellular with prominence of immature nuclear forms in FeLV-associated enteritis, which allows differentiation of this condition histologically from parvovirus infection.

AFIP DIAGNOSIS Small intestine: Enteritis, necrotizing, diffuse, moderate, with villous atrophy and crypt loss, domestic shorthair, feline.

Bone marrow: Hypercellularity with myeloid hyperplasia.

CONFERENCE NOTE In addition to FeLV-associated enteritis and feline parvoviral enteritis, attendees also included radiation exposure and treatment with radiomimetic drugs in the differential diagnosis.

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Hardy, W. D.: Feline leukemia virus non-neoplastic diseases. Journal of The American Animal Hospital Association, 17: 941-949. 1981.

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MICROSLIDE 11

HISTORY This 9-year-old American domestic shorthair female cat had a history of weight loss and distended abdomen of several months duration. A clinical diagnosis of feline infectious peritonitis was made and the owner requested euthanasia.

GROSS PATHOLOGY The mucous membranes were mildly icteric. The abdomen contained approximately 200 ml of clear yellow fluid. The liver was distorted due to marked, diffuse periportal and capsular fibrosis. There was moderate splenomegaly with marked lymphoid hyperplasia and thickening of the splenic capsule. The mesenteric lymph nodes were moderately enlarged.

LABORATORY RESULTS Hematology: No significant changes.

Blood Chemistry:	Tot Prot 9.9 g/dL	Alb 1.7 g/dL
	Glob 8.2 g/dL	GGT 5 IU/L
	Alk Phos 117 IU/L	AST 125 IU/L
	ALT 260 IU/L	Tot Bil 0.6 mg/dL
Abdominal Fluid:	Tot Sol 7.0 g/dL; Sp Grav 1.022; many neutrophils, moderate numbers of macrophages, and a few lymphocytes and RBC's.	

Tests for feline leukemia virus and feline infectious peritonitis were negative.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Liver: Severe, diffuse, chronic lymphocytic cholangitis with bile duct proliferation and biliary fibrosis.

Chronic cholangitis and cholangiohepatitis of ill-defined origin occur in mature cats. The inflammatory infiltrate is dominated by lymphocytes, with varying degrees of bile duct proliferation and portal fibrosis. Cholangitis and cholangiohepatitis in cats are often accompanied by some degree of pancreatic inflammation, possibly due to the anatomic association of the common bile duct and the pancreatic duct in this species. There are several reports in the literature describing inflammatory hepatobiliary disease in cats, and although different terminology is used in individual reports, these diseases share many common features suggesting they may not be separate entities but possibly different stages in the progression of a single disease, from suppurative cholangitis or cholangiohepatitis, to chronic, nonsuppurative cholangiohepatitis, and finally, biliary cirrhosis. Other reports describe a chronic, lymphocytic cholangitis in cats, characterized by lymphoid follicle formation, marked bile duct proliferation and concentric periductal fibrosis, which has been compared to primary biliary cirrhosis of man, as well as primary sclerosing cholangitis of man, and thus may represent a specific disease entity.

AFIP DIAGNOSIS Liver: Cholangiohepatitis, lymphocytic, chronic, bridging, diffuse, moderate with biliary hyperplasia, domestic shorthair, feline.

Liver: Capsulitis, chronic, diffuse, moderate, with edema.

CONFERENCE NOTE A number of participants remarked they had seen cases similar to this one but without the prominent capsulitis. Feline coronavirus and lymphosarcoma were the primary differentials.

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REFERENCES

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MICROSLIDE 12, 35 MM SLIDES 2 & 3

HISTORY This 8-year-old, collie-mix dog developed acute renal failure that was originally thought to possibly be due to ethylene glycol poisoning. The dog was a household pet with opportunity to run in the local area. The dog had been routinely immunized for canine pathogens including leptospirosis.

GROSS PATHOLOGY The kidneys were markedly swollen, soft, mottled tan to light brown with light gray-white areas throughout.

LABORATORY RESULTS BUN - 280, Creatinine - 6.5.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Generalized necrotizing pyogranulomatous nephritis due to a Leptospira infection.

Three more dogs in the neighborhood developed acute renal failure which presumably was due to leptospirosis. Serologic testing of these dogs showed the highest titer to L. grippotyphosa. The L. grippotyphosa titers of these 3 dogs were 1:3200 or greater. The L. canicola and L. icterohemorrhagiae titers were less than 1:200 for all 3 dogs. Evidence of L. grippotyphosa infection in dogs has previously been confirmed in foxhounds in Georgia.

AFIP DIAGNOSIS Kidney: Nephritis, interstitial, lymphoplasmacytic to pyogranulomatous, diffuse, moderate, collie-mix, canine.

CONFERENCE NOTE The moderator and many participants commented that the pattern of inflammation in this kidney was atypical of most cases of canine leptospirosis. While the multifocal to coalescing, predominantly lymphoplasmacytic, interstitial inflammation in the cortex is more typical, the diffuse predominantly pyogranulomatous inflammation in the medulla is not. The moderator suggested that a second disease process may be present; Encephalitozoon cuniculi infection was considered as a possibility, although special stains failed to demonstrate organisms.

Leptospira interrogans serovar grippotyphosa has rarely been reported in dogs. Serovars canicola, with the dog as the primary host, and icterohemorrhagiae, with the rat, are more commonly encountered.

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Baldwin, C. T., and Atkins, C. E.: Leptospirosis in dogs. Compendium on Continuing Education, 9: 499-507. 1987.

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MICROSLIDE 13

HISTORY The submitted tissue was obtained from the necropsy of a 21-month-old male Persian cat. The cat had a history of a watery ocular discharge with intermittent periods of anorexia and vomiting.

GROSS PATHOLOGY Significant gross findings were confined to the gastric mucosa which was thickened and had an increased prominence of rugal folds.

LABORATORY RESULTS Multiple complete blood counts and serum chemistry panels were within the normal range. Tests for feline leukemia virus and feline infectious peritonitis were negative.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Stomach, gastritis, fibrosing, chronic, due to Ollulanus tricuspis, feline, Persian.

Ollulanus tricuspis is a minute trichostrongyle nematode parasitizing the stomach of cats. Only recently has it been described in cats in the United States. The parasite is unusual in that the entire life cycle may be completed in one host. Route of exit from the host is via vomitus which may contain infective larvae and/or adults. Ingestion of the vomitus by an uninfected animal results in transmission of the parasite. Diagnosis of the infection can be difficult and requires examination of the vomitus as eggs and/or larvae are not passed in the feces. Concentration methods (i.e. Baermann technique) yield better results. Features helpful in identifying the parasite include its small size (less than or equal to 1 mm in length) and multiple longitudinal cuticular ridges. Adult females have three major and two minor tail cusps while adult males lack cusps but have a well developed terminal bursa with split copulatory spicules.

AFIP DIAGNOSIS Stomach: Gastritis, chronic, proliferative, diffuse, moderate with mucosal fibrosis and trichostrongylid nematodes, Persian, feline.

CONFERENCE NOTE A presumptive diagnosis of Ollulanus tricuspis was based on the small size and prominent cuticular ridges of the parasite and its location on the mucosal surface and in crypts. The platymyarian musculature and strongylid intestine of trichostrongylids were difficult to see in this section. Histologically, an increase in mucosal fibrous connective tissue and lymphoid aggregates have been reported to be characteristic of Ollulanus infection. This parasite can be differentiated from Strongyloides tumefaciens, a rabditid associated with intestinal nodules in cats, by the presence of both males and females in tissue sections; swallowed larvae of Aelurostrongylus abstrusus or other nematodes should also be considered in the differential diagnosis.

Participants compared this lesion in the cat to proliferative gastric or abomasal lesions caused by trichostrongylids in other species: Ostertagia ostertagia in cattle, Ostertagia circumsincta in sheep and goats, Molineus torulogus and Nochtia nochti in non-human primates, and Trichostrongylus axei in horses.

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MICROSLIDE 14, 35 MM SLIDE 4

HISTORY This neonatal thoroughbred colt was delivered prematurely by C-section. Soon thereafter it became comatose and seizured. Supportive therapy included sedatives, antibiotics and ventilation. Clinical examination revealed renal disease, ruptured urinary bladder secondary to urethral obstruction, pneumonia and oral candidiasis. The ruptured bladder was repaired surgically, but the animal died two days later.

GROSS PATHOLOGY Gross examination revealed multiple foci of inflammation, notably in the heart, kidneys, upper gastrointestinal tract, and peritoneal surfaces. Pulmonary atelectasis, polyarthritis, a urachal tear, and a small thymus were also present.

LABORATORY RESULTS

Hematology: RBC: 5.51 X 10^6/ul
WBC: 2 X 10^3/ul

Differential: Neutrophils 1780/ul (89%)
Lymphocytes 180/ul (9%)
Monocytes 40/ul (2%)

Plasma proteins: 5.5 g/dl
Creatinine: 6.3 mg/dl
BUN: 70 mg/dl
Glucose: 179 mg/dl

A swab submitted from the epicardium and other tissues resulted in moderate to heavy growths of yeast ultimately identified as Candida tropicalis.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Myocarditis and epicarditis, necrotizing, acute, multifocal, moderate, heart. Etiology: Candida tropicalis.

This animal was immunocompromised as evidenced by lymphoid depletion in organs such as thymus, spleen and visceral lymph nodes. Lymphoid depletion of both T-lymphocyte and B-lymphocyte zones, in conjunction with prematurity, resulted in systemic dissemination of secondary opportunistic agents, in this case Candida tropicalis. Candida organisms were present in the umbilicus, urachus, kidneys, urethra, tongue, esophagus, stomach, lymph nodes, spleen, liver, lung, brain and skeletal muscles. It is of interest to reflect on the mechanism of infection, e.g. in-utero vs post-natal infection, and the route of entry, e.g. umbilical vs. oral. No information was available relative to the breeding history or health status of the mare. Serum electrophoresis of the foal was not done.

AFIP DIAGNOSIS Heart: Myocarditis, necrotizing, multifocal, moderate, with intralesional yeast and pseudohyphae, thoroughbred, equine.

CONFERENCE NOTE The presence of characteristic poorly staining 3-5 um round to oval budding yeasts (blastospores) mixed with pseudohyphae and hyphae are usually sufficient to make a diagnosis of candidiasis, although fungal stains may be necessary to adequately demonstrate candidal morphology. The moderator recommended a Gomori's methenamine silver stain counterstained with hematoxylin and eosin if only one slide is available for examination.

Systemic candidiasis is generally characterized by suppuration and necrosis. Due to a compromised inflammatory response, the severely immunosuppressed host may have predominantly necrotic lesions and lack the usual abscessation. In this case the lack of prominent inflammation is further evidence that this foal was immunocompromised.

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REFERENCES

Gross, T. L., and Mayhew, I. G.: Gastroesophageal ulceration and candidiasis in foals. Journal of The American Veterinary Medical Association, 182: 1370-1373. 1983.

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MICROSLIDE 15

HISTORY This tissue is from a young adult female sika deer.

GROSS PATHOLOGY Several raised irregular yellow-white plaques were present on the pleural surface of the thoracic wall. The lungs were diffusely reddened and contained numerous, randomly distributed, yellow-white nodules varying in diameter from 0.5 to 1.5 cm. On sectioning, the nodules were firm and homogeneous, with a coarse, gritty texture.

LABORATORY RESULTS Mycobacterium bovis was isolated from lung and lymph node submitted to the National Veterinary Services Laboratory, APHIS, USDA, Ames, Iowa.

Intracellular acid-fast bacilli were not demonstrated using either Ziehl-Neelsen or Fite techniques for acid-fast organisms.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Severe, chronic, multifocal, granulomatous bronchopneumonia.

Multifocal coalescing granulomas are often centered around bronchioles and efface alveoli. The granulomas have necrotic, often mineralized centers encircled by large numbers of foamy vacuolated macrophages and variable numbers of multinucleate Langhans' giant cells, lymphocytes, and plasma cells. Alveolar septa throughout the lung are frequently thickened by moderate to large numbers of macrophages. Large numbers of lymphocytes and plasma cells are occasionally present adjacent to or surrounding bronchioles and blood vessels.

This animal was killed during eradication of a small privately owned herd following culture of M. bovis from a herdmate. All deer examined had similar microscopic lesions varying only in number and severity. M. bovis is the most frequently isolated mycobacterial species reported in ungulates in captivity as well as from deer in the wild. Failure to demonstrate intralesional acid fast organisms is not an uncommon finding in cases of M. bovis. Numerous intra- and extracellular acid fast bacilli were identified in sections of lung from a more severely affected herdmate.

AFIP DIAGNOSIS Lung: Granulomas, multiple, coalescing with mineralization, sika deer (Cervus nippon), cervine.

CONFERENCE NOTE The moderator was able to demonstrate small numbers of extracellular acid-fast bacilli in caseous debris within the lumen of a single airway using a Fite-Faraco acid-fast stain on an unstained section submitted by the contributor. The identification of these organisms was fortuitous as no bacilli were found in the rest of the section.

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REFERENCES

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MICROSLIDE 16

HISTORY This domestic house cat was presented to a veterinary clinic with a mass of prolonged duration on one foot. The mass was surgically removed and submitted for histopathologic examination.

GROSS PATHOLOGY At trimming the mass was found to be intensely black, with no other remarkable features.

LABORATORY RESULTS Following histopathologic examination, the surgical excision site was opened and a microbiologic culture specimen collected. Growth on a mycologic culture medium resulted in growth of deeply pigmented colonies. The culture growth was submitted to Dr. J. Kwong-Chung at the National Institutes of Health. The growth was identified as Exophiala jeanselmei.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Granulomatous inflammation, severe, skin, associated with pigmented fungal elements, etiology Exophiala jeanselmei.

Chromomycosis, severe, skin. Etiology: Exophiala jeanselmei.

Chromomycotic granulomas are seen occasionally in cats and tend to occur most commonly on the feet. Typical cases are presented as masses for histopathologic evaluation, therefore identification of the causative agent is limited to microscopic features of the organism. The present case represents a fortunate occasion in which cultural identification was possible.

AFIP DIAGNOSIS Haired skin (dermis) and subcutis: Dermatitis and panniculitis, focally extensive, severe, with associated dematiaceous fungal elements, domestic, feline.

CONFERENCE NOTE Phaeohyphomycosis is the term given to systemic or cutaneous infection by opportunistic dematiaceous (pigmented) fungi; a wide variety of genera, including Phialophora, Cladosporium, Drechslera, Dactylaria and Exophiala, have been reported. Histologically, fungal elements are heavily-pigmented yellow-brown septate hyphae that are often constricted at their septations and frequently have thick-walled vesicular swellings resembling chlamydospores. The various genera of pigmented fungi appear similar in tissue

and specific identification can only be made by isolating the organism. The moderator commented that deparaffinized, unstained sections are useful in demonstrating the morphology of pigmented fungi.

Phaeohyphomycosis should not be confused with chromoblastomycosis, a disease of humans and amphibians. While also caused by opportunistic pigmented fungi, chromoblastomycosis is characterized by large (6-12 um) thick-walled, dark brown muriform cells (sclerotic bodies).

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MICROSLIDE 17

HISTORY This adult sea mullet (Mugil cephalus) was collected from the Richmond River estuary in northeast New South Wales 2 weeks after moderate local flooding.

GROSS PATHOLOGY A single pale grey to pink raised ovoid lesion 2 cm x 3 cm with macerated surface was present on the skin. Scales over the lesion were missing. Skeletal muscle underlying the lesion was pale yellow, soft and wet.

LABORATORY RESULTS Large numbers of broad, non-septate, sparsely branching hyphae were visible in squash preparations of skin and skeletal muscle.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Necrotizing granulomatous dermatitis and myositis. There is locally extensive invasion of skin and muscle by unidentified fungi, consistent with class Oomycetes.

This lesion is consistent with red spot disease, a syndrome of cutaneous ulceration which affects several species of estuarine fish in New South Wales and Queensland, Australia. Lesions range from a mild to severe, chronic active dermatitis without fungal involvement (erythematous dermatitis) to a severe, locally extensive granulomatous lesion with dermal and skeletal muscle invasion by numerous broad (15-30 um), branching, non-septate fungal hyphae encircled by macrophages (necrotizing dermatitis). The invasive fungi have not been identified.

The pathogenesis of red spot disease is unknown. Damage to fish skin may be necessary before fungal invasion occurs. Outbreaks typically begin two weeks after heavy rainfall as flood waters recede, leading to the speculation that associated water quality changes, notably falls in dissolved oxygen concentrations to sub-lethal levels, may predispose fish skin to fungal invasion. Damage to skin from viral infection has also been proposed.

AFIP DIAGNOSIS Skin, hypodermis and muscle: Dermatitis, panniculitis and myositis, necrotizing, granulomatous, diffuse, severe with intralesional fungal hyphae, sea mullet (Mugil cephalus), piscine.

CONFERENCE NOTE Many conference participants considered zygomycotic fungi as a primary differential based on the very broad, branching, infrequently septate, hyaline hyphae present. The moderator compared this lesion to "kunkers", a necrotizing cutaneous lesion in horses caused by either Basidiobolus haptosporus, a zygomycete of the order Entomophthorales, or Pythium sp., an organism now considered a protista in the phylum Oomycetes. In some sections, protozoal organisms consistent with a myxosporidian parasite were present in the skin.

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REFERENCES

Callinan, R. B., Frasser, G. C., and Virgona, J. L.: Pathology of red spot disease in sea mullet Mugil cephalus Linnaeus from eastern Australia. Journal of Fish Diseases. (In Press.)

Mackenzie, R. A., and Hall, W. T. K.: Dermal ulceration of mullet (Mugil cephalus). Australia Veterinary Journal, 52: 230-231. 1976.

MICROSLIDE 18

HISTORY This 3-year-old Suffolk ewe from Maryland was hospitalized after a 5-day history of ataxia. She would fall and later was unable to stand. She was bright and alert and ate well but was pruritic. After five more days her condition worsened and she was euthanatized.

GROSS PATHOLOGY Not available.

LABORATORY RESULTS Not available.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Spongiform encephalopathy with gliosis, astrocytosis and neuronal vacuolation due to infection with the scrapie agent.

Based on a histological examination of brain sections, a diagnosis of scrapie was confirmed by Dr. A. J. Davis of the National Veterinary Services Laboratory in Ames, Iowa. Astrocytosis and neuronal vacuoles were present in several brain stem nuclei. Microcavitation was more pronounced in some nuclei than in others.

Scrapie infection appears to be caused by a transmissible filterable agent, a prion, a low molecular weight protein (27-30 kDa) containing no nucleic acid. Scrapie associated fibrils are aggregates of the prion protein (PrP 27-30) and can be demonstrated by electron microscopy.

AFIP DIAGNOSIS Brain stem, gray matter: Neuronal vacuolation, multifocal, minimal, with gliosis, Suffolk, ovine.

CONFERENCE NOTE Vacuolated neurons characteristic of scrapie were infrequent in many sections; several conference participants diagnosed essentially normal tissue. The moderator commented that sheep brain tissue should be carefully examined for lesions of scrapie when there are no apparent histologic lesions at low magnification and there is a history of neurological symptoms.

In comparison to scrapie of sheep, the moderator showed a section of bovine spongiform encephalopathy (BSE), a disease of adult cattle recently identified in Great Britain. The predominant change seen histologically in BSE is bilaterally symmetrical neuronal vacuolation very similar to that of scrapie. On electron microscopy scrapie associated fibrils have been detected in BSE. It is speculated that BSE is caused by scrapie or a scrapie-like agent that was introduced by feeding cattle ruminant-derived protein, possibly of sheep origin. Food-born cross-species transmission of scrapie is not unprecedented; transmissible mink encephalopathy has been attributed to feeding scrapie-infected sheep offal to mink.

CONTRIBUTOR CIBA-GEIGY Pharmaceuticals Corporation
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REFERENCES

Brunon, M., et al.: The scrapie agent and the prion hypothesis. Trends in Biochem. Science, 13: 309-313. 1988.

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Wells, G. A. H., et al.: A novel progressive spongiform encephalopathy in cattle. Veterinary Record, 121: 419-420. 1987.

Wilesmith, J. W., et al.: Bovine spongiform encephalopathy: epidemiological studies. Veterinary Record, 123: 638-644. 1988.

MICROSLIDE 19

HISTORY A day old quarterhorse colt appeared impacted and was given a phosphate enema. He was presented to the veterinarian with diarrhea, increased heart rate, decreased gut sounds and signs of abdominal pain. The colt rapidly became moribund and died.

GROSS PATHOLOGY A large segment of the ileum was distended with blood tinged fluid. The mucosal surface was quite congested and hemorrhagic.

LABORATORY RESULTS Small and large intestine cultures were positive for abundant numbers of Clostridium perfringens and Escherichia coli. Additional studies to characterize enterotoxins were not performed.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Acute necrotizing hemorrhagic enteritis with denuded and bacilli-lined villi and rapid mucosal autolysis. Clostridium perfringens.

There is marked autolysis and necrosis of the luminal half of the mucosa. The villi are denuded and lined by massive numbers of large basophilic bacilli. Focal hemorrhage of the mucosa and submucosa with variable edema of the submucosa is present. The cause of death is attributed to acute enterotoxemia.

AFIP DIAGNOSIS Small intestine: Enteritis, necrohemorrhagic, diffuse, moderate, with myriads of large bacilli, quarterhorse, equine.

CONFERENCE NOTE Most participants agreed with the contributor's diagnosis. Shock was discussed as a possible cause of similar lesions.

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REFERENCES

Barker, I. K., and Van Dreumel, A. A.: The alimentary system. In: Pathology of Domestic Animals, Third Edition, Volume 2, pages 149-155. 1985. Edited by: Jubb, K. V. F., Kennedy, D. C., and Palmer, N. Academic Press, Orlando.

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Ochoa, R., et al.: The effects of Clostridium perfringens Type A enterotoxin in Shetland ponies. Veterinary Pathology, 17: 738. 1980.

MICROSLIDE 20

HISTORY This 8-day-old piglet died suddenly. A field necropsy was performed by the referring veterinarian and kidneys were submitted fresh and in formalin.

GROSS PATHOLOGY There were multifocal ecchymotic and petechial hemorrhages on the surface of kidney and extending into the parenchyma of the cortex ("turkey egg" kidney).

LABORATORY RESULTS Salmonella cholerasuis (kunzendorf) was isolated from the kidneys.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Hemorrhage and necrosis with fibrinous thrombosis of glomerular capillaries, acute, multifocal, moderate, kidney. Etiology: Salmonella cholerasuis (kunzendorf).

Although only kidneys were submitted in this case, this animal was probably septicemic. Fibrinous thrombi in glomerular capillaries are suggestive of D.I.C. In some glomerular capillaries, bacterial colonies can also be seen. The "turkey egg" kidney has been described previously with Salmonella septicemia, but is not seen in all such cases.

AFIP DIAGNOSIS Kidney, glomerular capillaries: Fibrin thrombi and bacterial emboli, multifocal, mild, with hemorrhage and tubular necrosis, breed unspecified, porcine.

CONFERENCE NOTE Most conference participants listed Erysipelothrix rhusiopathiae as the likely etiology. Hog cholera, African swine fever, and other bacterial septicemias such as Hemophilus or Streptococcus should also be included in the differential diagnosis.

The prominent cortical glomerulopoiesis is indicative of a very young animal.

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REFERENCES

Barker, I. K., and Van Dreumel, A. A.: The alimentary system. In: Pathology of Domestic Animals, Third Edition, Volume 2, pages 140-143. 1985. Edited by: Jubb, K. V. F., Kennedy, P. C., and Palmer, N. Academic Press, Orlando, Florida.

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MICROSLIDE 21, 35 MM SLIDE 5

HISTORY This 6-year-old Morgan horse gelding had slowly developed posterior ataxia over the last few months and recently had been unable to defecate. At presentation, the tail and anus were flaccid. The hindquarters, tail, and tail head were anesthetic.

GROSS PATHOLOGY The urinary bladder was distended and filled with a mass of firm light yellow "clay-like" material. Ventral coccygeal muscles were pale and mildly edematous. The cauda equina was swollen, hemorrhagic, and indurated.

LABORATORY RESULTS Not available.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Lymphoplasmacytic neuritis of the cauda equina, equine. Etiology: unknown.

In sections of cauda equina, there is marked perineural fibrosis with multifocal perineuronal hemorrhages. Low to moderate numbers of lymphocytes and plasma cells are present throughout the perineural connective tissue and nerve bundles with numerous focal perineural aggregates of lymphocytes and plasma cells.

This is a disease of horses that has been recognized in Europe and the United States. No etiology has been confirmed. However, the disease has been circumstantially associated with preceding respiratory disease. Lesions similar to those seen in equine cauda equina syndrome have been reported in cranial nerves as well as other spinal nerves.

AFIP DIAGNOSIS Cauda equina: Polyneuritis, lymphoplasmacytic, fibrosing, diffuse, moderate to severe, with hemorrhage, axonal degeneration and Schwann cell proliferation, Morgan, equine.

Spinal cord, white matter: Axonal degeneration, multifocal, mild.

CONFERENCE NOTE The clinical history and gross and microscopic lesions in this case are classic for chronic neuritis of the cauda equina in the horse.

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REFERENCES

deLahunta, A.: Veterinary Neuroanatomy and Clinical Neurology. Second Edition, pages 80-81. 1983. Saunders, Philadelphia.

Sullivan, N. D.: The nervous system. In: Pathology of Domestic Animals, Third Edition, Volume 1, page 309. 1985. Edited by: Jubb, K. V. F., Kennedy, P. C., and Palmer, N. Academic Press, Orlando, Florida.

Martens, R., Stewart, J., and Eicholtz, D.: Clinico-Pathologic Conference - University of Pennsylvania. Journal of The American Veterinary Medical Association, 156: 478-487. 1970.

MICROSLIDE 22

HISTORY This 2-year-old male Shar pei dog's skin began to thicken and shed at 4 months of age, but he did not have excessive folds for this breed. The skin condition and severe body odor continued to worsen up to the time of biopsy in spite of an early course of corticosteroids and continuing varied antibiotic therapy for 1-1/2 years. Neutering at one year of age had no effect. Others in the litter are affected with demodecosis. After biopsy, the dog was placed on 40 mg prednisone (with antibiotics) and has improved markedly.

GROSS PATHOLOGY There is a rather sharp line of demarcation along the lateral trunk with the head, neck and ventral body involved severely. The ventrum and legs are severely lichenified and thickened with hyperpigmentation, complete alopecia and rancid greasy seborrhea oleosa. The dorsal thorax and abdomen seem more normal although scaling is becoming evident along the back.

LABORATORY RESULTS Numerous skin scrapings and three separate thyroid panels, as well as ANA titer, were normal. RAST testing revealed reactivity to most every allergen but there was no clinical response to hyperimmune serum injections.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Idiopathic mucinosis.

Multifocal spongiotic superficial perivascular dermatitis, consistent with seborrhea.

These slides were cut from wedge skin biopsies from three sites on the shoulder, upper leg and back at the alopecic interface, and thus show variable changes. All show epidermal and follicular hyperkeratosis and acanthosis associated with the severe separation of dermal collagen fibers by basophilic mucinous ground substance. Sebaceous glands are prominent but not hyperplastic. A certain degree of dermal mucin is "normal" in the Shar pei but this degree should be regarded as pathologic. The epidermal crusting, spongiosis, pigmentary incontinence and multifocal perivascular inflammation with neutrophilic exocytosis reflect concurrent seborrhea, also common in this breed and probably responsible for most of the patient's signs. The pitting edema appearance often resolves ("deflates") at sexual maturity or possibly under corticosteroid therapy. Some slides look more "deflated" and have the deep dermis and adnexa artefactually cut off but the superficial dermis is characteristic. In some sections, the stratum corneum is separated giving the impression of vesicle formation as has been described clinically.

AFIP DIAGNOSIS Haired skin: Dermal mucinosis, diffuse, moderate, with acanthosis and follicular keratosis, Shar pei, canine.

CONFERENCE NOTE In the experience of the moderator, the examined sections are typical of "normal" Shar pei skin. Idiopathic mucinosis in the Shar pei is characterized by large lakes of dermal mucin that widely separate preexisting collagen; in this case there is much less mucin and more collagen than one would expect in idiopathic mucinosis. The moderator commented that mucinosis is often best diagnosed grossly by the abundant viscous mucin "blobs" seen when the biopsy specimen is trimmed in.

Conference participants considered focal mucinosis, mucopolysaccharidosis and mucinosis secondary to hypothyroidism, hypersomatotropism, or systemic lupus erythematosus in the differential diagnosis if this skin section was not from a Shar pei.

CONTRIBUTOR Comparative Medical and Veterinary Services
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REFERENCES

Dillberger, J. E., and Altman, N. H.: Focal mucinosis in dogs: Seven cases and review of cutaneous mucinoses of man and animals. Veterinary Pathology, 23: 132-139. 1986.

Muller, G. H., Kirk, R. W., and Scott, D. W.: Small Animal Dermatology. Fourth Edition, pages 842 and 952. 1989. W. B. Saunders, Philadelphia.

MICROSLIDE 23

HISTORY This 6-month-old, female Holstein calf was to be used as a laboratory animal during chronic cardiovascular studies. A skin condition was diagnosed during the quarantine period. Another calf was substituted for the intended study and this calf was maintained in isolation and treated.

GROSS PATHOLOGY There were multiple raised grey-white firm nodules ranging from 1-3 cm diameter in the head, neck, and thoracic skin.

LABORATORY RESULTS Trichophyton verrucosum was isolated from the lesions.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Focal purulent subacute dermatitis - Dermatomycosis due to Trichophyton verrucosum.

AFIP DIAGNOSIS Haired skin: Folliculitis and furunculosis, suppurative, diffuse, severe, with chronic hyperplastic dermatitis, intracorneal pustules and endothrix and ectothrix arthrospores and hyphae, Holstein, bovine.

CONFERENCE NOTE Trichophyton verrucosum is the most frequently isolated dermatophyte in cattle. Calves are more commonly infected than adults and the head, neck and perineum are sites of predilection. Gross lesions are circumscribed, alopecic, up to 5 cm in diameter, and covered with a thick gray-white crust. Histologically, there is marked hyperplasia of the surface epidermis and proximal root sheath with rete ridge formation, ortho- and parakeratotic hyperkeratosis, and intracorneal microabscesses. Suppurative to pyogranulomatous folliculitis and furunculosis are often seen in severe cases. Branching septate hyphae and arthrospores colonize the keratinized layers of the epithelium and the hair shafts. Trichophyton spp. are both endothrix (within the hair shaft) and ectothrix (on the hair surface) compared to Microsporum spp., which are usually ectothrix.

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REFERENCES

Fox, J. G., Cohen, B. J., and Loew, F. M.: Laboratory Animal Medicine, page 632. 1984. Academic Press, Orlando, Florida.

Yager, J. A., and Scott, D. W.: In: Pathology of Domestic Animals, Volume 1, page 483. 1985. Edited by: Jubb, K. V. F., Kennedy, P. C., and Palmer, N. Academic Press, Orlando, Florida.

MICROSLIDE 24

HISTORY This 6-year-old neutered male Siamese cat developed multiple slow-growing subcutaneous nodules behind both ears that varied in size from 1-10 mm. There was temporary response to corticosteroids and antibiotics.

GROSS PATHOLOGY Multiple, soft, yellow-white, intradermal nodules were surgically removed.

LABORATORY RESULTS Not available.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Xanthogranulomas, multiple, dermal and subcutaneous, head, feline (xanthomatosis).

The disease has been associated with diabetes mellitus and hypertriglyceridemia. Other cases are idiopathic. Most cases in the cat are secondary to megesterol acetate (Ovaban) therapy.

A follow-up on this case showed no evidence of diabetes but the referring veterinarian reported that the cat had been on Ovaban for 4 years.

AFIP DIAGNOSIS Haired skin: Dermatitis and panniculitis, histiocytic, focally extensive, severe, with acicular clefts, consistent with xanthomatosis, Siamese, feline.

CONFERENCE NOTE Densely packed large foamy histiocytes that stain positive for lipid are characteristic of xanthomatosis. Associated granulomatous inflammation and extracellular lipid deposits are occasionally seen, as in this case.

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REFERENCES

Fawcett, J., and Demaray, S. Y.: Multiple xanthomatosis in a cat. Feline Practice, 7: 31-33. 1977.

Jones, B. R., et al.: Cutaneous xanthomata associated with diabetes mellitus in a cat. Journal of Small Animal Practice, 26: 33-41. 1985.

Kwochka, K. W., and Short, B. G.: Cutaneous xanthomatosis and diabetes mellitus following long-term therapy with megesterol acetate in a cat. Compendium on Continuing Education, 6: 185-191. 1984.

Muller, G. H., Kirk, R. W., and Scott, D. W.: Small animal dermatology. Fourth Edition, page 945. 1989. W. B. Saunders, Philadelphia.

MICROSLIDE 25

HISTORY This 3-year-old mixed breed spayed female dog developed nodular eruptions over the bridge of the nose and around the nostrils. There was no response to antibiotics.

GROSS PATHOLOGY Not available.

LABORATORY RESULTS Not available.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Multinodular granulomatous perifolliculitis (idiopathic periadnexal multinodular granulomatous dermatitis).

Distinctive features characteristic of idiopathic periadnexal multinodular granulomatous dermatitis (Carpenter, et al) are apparent in most sections. Nodular masses of cells oriented around hair follicles extend from just below the infundibulum to encompass the hair bulb and consist of macrophages, lymphocytes and plasma cell with rare neutrophils. Where the plane of section includes the hair follicle, the outer root sheath and hair shaft are unaffected by the inflammation and apocrine glands are similarly preserved. In contrast, sebaceous glands are effaced in affected pilosebaceous units, while they are present in adjacent unaffected units. Special stains (PAS, Acid-Fast, Toluidine Blue and Gram's stain) failed to reveal an etiologic agent.

Lesions typically begin on the face and clinically exhibit resistance to antibiotic therapy but respond to systemic corticosteroids. The pathogenesis of this condition is unknown, but may be immune-mediated.

AFIP DIAGNOSIS Haired skin: Dermatitis, granulomatous, periadnexal, nodular, multifocal to coalescing, moderate, mixed-breed, canine.

CONFERENCE NOTE The differential diagnosis includes necrobiotic granuloma, canine cutaneous histiocytoma, cutaneous histiocytosis, sterile nodular panniculitis, familial systemic histiocytosis of Bernese mountain dogs and sebaceous adenitis of standard poodles.

CONTRIBUTOR Experimental Pathology Laboratories, Inc.
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Research Triangle Park, North Carolina 27709

REFERENCES

Calderwood-Mays, M. B., and Bergeon, J. A.: Cutaneous histiocytosis in dogs. Journal of The American Veterinary Medical Association, 188(4): 377-381. 1986.

Carpenter, J. L., Thornton, G. W., Moore, F. M., and King Jr., N. W.: Idiopathic periadnexal multinodular granulomatous dermatitis. Veterinary Pathology, 24: 5-10. 1987.

Rosses, E. J., et al.: Sebaceous adenitis with hyperkeratosis in the standard poodle: a discussion of 10 cases. Journal of The American Animal Hospital Association, 23: 341-345. 1987.

MICROSLIDE 26

HISTORY This 9-year-old female Norwegian elkhound developed signs of intestinal obstruction with vomiting which lasted intermittently for 7 to 8 weeks. An exploratory laparotomy revealed a gastric mass. The animal was euthanatized.

GROSS PATHOLOGY In addition to the gastric tumor, necropsy findings included a mottled, friable liver and rough, granular gallbladder mucosa.

LABORATORY RESULTS Not available.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Cystic, mucinous adenocarcinoma, stomach, with metastasis to omentum, spleen, liver, gallbladder, mesenteric and pancreatic lymph nodes, dog.

Glomerulointerstitial nephritis, membranoproliferative, chronic, plasmacytic, multifocal to diffuse, moderate, with amyloidosis and tubular protein casts, kidney.

Cystic mucinous hyperplasia, diffuse, severe, gallbladder.

Cytoplasmic vacuolization, diffuse, severe, hepatocytes, liver.

Congestion, edema, thrombosis, multifocal, moderate, with focal vascular mineralization, lung.

This was a highly malignant neoplasm with lymphatic invasion, and widespread metastasis. The marked smooth muscle hypertrophy and desmoplasia most likely caused pyloric stenosis and subsequent vomiting.

AFIP DIAGNOSIS Stomach: Adenocarcinoma, Norwegian elkhound, canine.

CONFERENCE NOTE Gastric adenocarcinoma is the most common neoplasm of the canine stomach, but is rare (less than 1%) among all canine malignant neoplasms. As in humans, tumors are most often seen in males and usually arise in the lesser curvature or the pylorus. A recent study from Europe (Fonda, et al.) reports a breed predilection for Belgian shepherd dogs. Human and canine gastric adenocarcinomas have been classified as either intestinal, with distinct glandular structures lined by well polarized cells, or diffuse, with random infiltration by neoplastic cells; an intermediate type with characteristics of both has also been described. Some authors further divide the intestinal type into tubular or papillary, and the diffuse type into mucinous, signet ring or undifferentiated. Studies vary in reporting which type is most common in the dog.

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REFERENCES

Fonda, D., Gualtieri, M., and Scanziani, E.: Gastric carcinoma in the dog: a clinicopathological study of 11 cases. Journal of Small Animal Practice, 30: 353-360. 1989.

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Patnaik, A. K., Hurvitz, A. I., and Johnson, G. F.: Canine gastric adenocarcinoma. Veterinary Pathology, 15: 600-607. 1978.

MICROSLIDE 27

HISTORY This tissue is from a wild muskrat (Ondatra zibethicus).

GROSS PATHOLOGY Not available.

LABORATORY RESULTS Not available.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Liver: Cysticercosis, muskrat, Rodentia, due to Cysticercus fasciolaris.

Liver: Nematodiasis, due to Capillaria hepatica.

Cysticercus fasciolaris is the larval stage of Taenia taeniaformis. Adults are found in the intestine of domestic dogs and cats and some free living carnivores such as wolves, lynx, and racoons. Infective eggs are passed in feces. Larvae develop in the gut of the rodent, and then migrate to internal organs, i.e. liver, where the cysticercus forms. The cycle is completed when the rodent is consumed by a carnivore.

The primary definitive host for Capillaria hepatica seems to be a rodent, but it is reported in a variety of mammals including the rat, muskrat, shrew, mouse, vole, rabbit, hare, squirrel, beaver, woodchuck, cavy, prairie dog, peccary, pig, dog, cat, horse, capuchin, chimpanzee and man.

Capillaria hepatica inhabits the liver, where adult nematodes copulate and die soon after the female worm has deposited a large number of eggs. These eggs mature in the liver tissue only to a stage of a 4 to 8 cell morula. In order to sporulate further the eggs require the high oxygen tension and humidity of soil.

This is achieved in two ways: (1) the egg bearing host is eaten by an omnivore or carnivore, where the eggs merely pass through the intestines and reach the environment. This passing of eggs in the feces is called spurious infection or (2) the egg bearing host dies and the eggs are freed from the carcass by autolytic processes and end up in water or soil. After the infective eggs are ingested by an animal the first stage larva hatches in the small intestine and reaches

the liver via the portal circulation. This process is believed to be a direct migration, but there is data suggesting a possibility of passive larval migration to the lungs and kidneys.

The hepatic pathology of liver capillariasis varies greatly depending on the species and the immunological status of the host. There might be extensive liver damage with multifocal necrosis and foreign body granuloma formation with liver fibrosis and death of the host, or there may be no acute response to the adult and the eggs, which are gradually removed by the host. Examination of the eggs of Capillaria hepatica is important, because their morphology is often the only means to establish the diagnosis on a liver biopsy. Eggs are described as 35x65 um, barrel shaped and double operculated with a double shell having striae between the layers and pits in the outer shell.

AFIP DIAGNOSIS Liver: Strobilocercus with associated chronic inflammation, muskrat (Ondatra zibethicus), rodent, etiology--Cysticercus fasciolaris.

Liver: Hepatitis, granulomatous, multifocal, random, mild, with associated Capillaria sp. eggs.

CONFERENCE NOTE The moderator commented that he has seen Cysticercus fasciolaris in isolation-reared laboratory rodents that were presumably infected from feed contaminated with cat feces. The moderator also mentioned that Cysticercus fasciolaris has been linked to hepatic sarcomas.

The larval stage of Taenia taeniaformis is considered a strobilocercus at about 42 days post infection when the scolex evaginates and becomes connected to the bladder by the segmented strobila.

Adult Capillaria with characteristic bacillary bands were present in some sections. The degree of inflammation associated with the Capillaria eggs varied from section to section.

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MICROSLIDE 28

HISTORY A 1-year-old miniature dachshund had a history of severe weight loss, tachypnea and dyspnea. There was a generalized patchy alveolar pattern in the lungs on thoracic radiographs. The dog was euthanatized.

GROSS PATHOLOGY The lungs did not collapse when the thoracic cavity was opened.

LABORATORY RESULTS Bacterial cultures were negative.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Diffuse interstitial pneumonia.

According to the literature, pneumocystis occurs sporadically in dogs, but there are several reports of the condition in young miniature dachshunds. The morphologic lesions are typical and similar to those seen in other species. The possible existence of an inherited immune deficiency has been suggested.

AFIP DIAGNOSIS Lung: Pneumonia, interstitial, diffuse, severe, with type II pneumocyte hyperplasia, intra-alveolar histiocytosis and eosinophilic flocculent material, miniature dachshund, canine.

CONFERENCE NOTE Most participants diagnosed pulmonary pneumocystosis based on the presence of the characteristic amorphous foamy exudate in the alveoli. Organisms were demonstrated by GMS impregnation. Interstitial pneumonia, alveolar histiocytosis and hypertrophy of type II pneumocytes are also typical lesions. Participants agreed that histiocytes and type II pneumocytes are often difficult to distinguish by light microscopy. The taxonomical position of Pneumocystis carinii remains unclear; it has been considered as either a protozoan or as a fungus of the class Ascomycetes. Pneumocystosis usually occurs in immune suppressed animals.

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REFERENCES

Arean, V. M.: Pulmonary pneumocystosis. In: Pathology of Protozoal and Helminthic Diseases, pages 291-317. 1971. Edited by: Marcial-Rojas, R. A.

Botha, W. S., and Van Rensburg, I. B. J.: Pneumocystosis: A chronic respiratory distress syndrome in the dog. Journal of the South African Veterinary Association, 50: 173-179. 1979.

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Yoshida, Y.: Ultrastructural studies of Pneumocystis carinii. Journal of Protozoal, 36(1): 53-60. 1989.

MICROSLIDE 29, 35 MM SLIDE 6

HISTORY This tissue is from one of several 3-month-old female AKR/J mice within an established mouse colony in which many young mice developed prominent perianal hyperemia and mild rectal prolapse.

GROSS PATHOLOGY At necropsy, affected animals had thickened, rigid, corrugated colons, especially in the terminal/distal region.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Hyperplasia, mucosal, diffuse, marked.

Colitis, acute to subacute, patchy/segmental, mild to moderate (some sections) -- consistent morphologically with transmissible murine colonic hyperplasia. Etiology: Citrobacter freundii.

The gross and microscopic lesions in these cases are consistent with those described (Barthold, et al.) for transmissible murine colonic hyperplasia, a naturally occurring infectious disease of mice caused by a specific strain of Citrobacter freundii (C. freundii 4280). The principle microscopic lesion is marked mucosal hyperplasia, with increased crypt height, prominent crowding and pseudostratification of crypt epithelium, increased mitotic activity, and a dramatic decrease or absence of goblet cells. Inflammation is variably present, and ranges from a few scattered mixed inflammatory cells within the mucosa to a more severe, extensive reaction which in some segments extends transmurally with an associated involvement of the serosa and adjacent mesocolon. Focal mild superficial mucosal ulceration is present in several sections.

Citrobacter freundii was isolated in bacterial cultures from colons of all affected mice (frequently in pure culture), and disease was subsequently reproduced by inoculating these culture suspensions back into naive animals.

Transmissible murine colonic hyperplasia may cause high mortality in suckling mice, but is frequently inapparent clinically in adults. Bacterial attachment, which is an essential part of the pathogenicity of this organism, occurs on surface mucosa within 4 days of infection. Subsequent cellular changes may be related either to endotoxin effects, or to the sequelae of distortion created in the surface epithelium brush border. Marked variation in the severity of the disease is associated with dietary influences and strain/genetic differences. NIH Swiss mice are the most susceptible and C57BL/6J and DBA/2J the least, according to one study. As was the case in this epizootic, neomycin or tetracycline therapy is generally effective for treatment, however; some strains can harbor C. freundii in a carrier state for 9 months, thus making eradication difficult.

AFIP DIAGNOSIS Colon: Hyperplasia, diffuse, severe with multifocal submucosal and mesenteric subacute inflammation, AKR/J mouse, rodent.

CONFERENCE NOTE Participants compared this lesion to the Campylobacter-related proliferative ileitis of hamsters and pigs. The combination of circumstances required to produce colonic hyperplasia (strain of Citrobacter freundii, strain of host mouse, feed) was discussed.

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REFERENCES

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MICROSLIDE 30, 35 MM SLIDE 7

HISTORY This 5-month-old female thoroughbred foal and its mother were on a pasture in northern New Mexico. The owner noticed that the foal was losing weight. The foal and mare were put in a dry lot so that the foal could be watched more closely. The foal continued to lose weight over the next 10 days. The foal had difficulty swallowing that developed into a complete inability to swallow. The foal had a normal body temperature. Because of a poor prognosis for recovery, the foal was euthanatized.

GROSS PATHOLOGY Lymph nodes throughout the body are slightly enlarged. There is mild diffuse reddening of the mucosa of the cecum and large colon. Formed feces are present in the small colon. No gross lesions are observed in the mouth, pharynx or larynx. No lesions are observed externally in the brain.

LABORATORY RESULTS There is a moderate elevation in the WBC count.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Morphologic diagnosis: Focal encephalomalacia, chronic (nigropalladial encephalomalacia). Etiology: Russian knapweed (Centaurea repens) toxicosis.

The focal areas of aseptic necrosis in the substantia nigra are considered pathognomonic for Centaurea sp. toxicosis in horses. Yellow star thistle (C. solstitialis) is the cause of this condition on the west coast of the United States and Russian knapweed (C. repens) is the cause in the western mountain region. In the present case, the horses had access to abundant Russian knapweed. No yellow star thistle was present in the pasture.

AFIP DIAGNOSIS Brain stem: Encephalomalacia, focally extensive, severe, thoroughbred, equine.

CONFERENCE NOTE Nigropalladial encephalomalacia has only been reported in horses and ponies. Young horses seem particularly sensitive, with foals as young as 4 months affected and over half of reported cases in horses under 2 years. Clinical signs include hypokinesia and difficulty in prehension and mastication. The usual cause of death is dehydration and/or starvation. Signs have been related to dopamine deficiency in the nigrostriatal tract. The characteristic early histological lesions are non-progressive pannecrosis of the globus pallidus and/or the substantia nigra with sharply defined margins between living and necrotic tissue and little or no hemorrhage. Later lesions are predominantly phagocytic. The onset of clinical signs and lesions is abrupt, although Centaurea sp plants may have been ingested for some time, suggesting a threshold must first be reached. The toxin remains unidentified.

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REFERENCES

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MICROSLIDE 31

HISTORY Eight months prior to death this 16-year-old thoroughbred mare developed right hind leg swelling which was treated symptomatically. The attending veterinarian noted increased total protein level at that time. At terminus the right hind leg became acutely swollen and painful, and the BUN was acutely elevated.

GROSS PATHOLOGY The diaphyseal fatty zone of both femurs had a central zone of dark red tissue.

LABORATORY RESULTS Clinicopathologic studies at a referral equine hospital revealed IgG-T gammopathy.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Plasma cell myeloma with IgG-T gammopathy.

Histopathologic examination revealed plasmacytic infiltrates in the kidneys, liver, spleen and bone marrow. The kidneys had massive involvement, with a large amount of protein and cellular debris in tubular lumina. Renal glomerular capsules were thickened and fibrotic, and some glomerular tufts were mildly sclerotic.

AFIP DIAGNOSIS Bone marrow: Plasma cell myeloma, thoroughbred, equine.

CONFERENCE NOTE Diagnosis of plasma cell myeloma requires the presence of at least two of the following conditions: neoplastic plasma cells (present in this case in marrow, spleen, liver and kidneys), a monoclonal gammopathy (present), osteolytic lesions (present on radiographs presented by the moderator), and Bence-Jones proteinuria (not reported).

The Department of Hematolymphatic Pathology concurred with the contributor/moderator's diagnosis of myeloma based on the hypercellular marrow with predominantly plasmacytoid cells. There is a diffuse lack of erythropoietic cells, but scattered normal myelopoietic elements remain in varying amounts from section to section.

Many conference participants included equine infectious anemia in the differential diagnosis before acquiring the information on the monoclonal gammopathy.

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REFERENCES

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MICROSLIDE 32

HISTORY This 7-week-old male Arabian foal was "normal" at birth. At 1 week of age, the foal developed respiratory disease.

GROSS PATHOLOGY Lungs fail to collapse. Multiple, coalescing dark red areas are scattered over the pleural surface of each lobe. The caudodorsal parenchyma is hyperinflated. Ventral aspects of the cranial lobes are firm and on section have dark red, soft areas that are

occasionally peribronchial. Airways in the ventral aspect of the cranial lung lobes and the cranioventral aspects of the caudal lung lobes contain soft tan material. Adipose tissue occupies the cranial mediastinal space normally occupied by the thymus. Scattered in the adipose tissue are multifocal islands of pink to gray tissue compatible with thymic tissue. The spleen is small even though the foal was euthanatized with barbiturates. Lymph nodes are small and difficult to identify. The pancreas is markedly small and firm.

LABORATORY RESULTS

At 24 hours of age:	IgG	900
	IgM	35
At 5 weeks of age:	IgG	400
	IgM	0
	lymphopenia (lymphocytes	500)

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Lymphoid hypoplasia, marked, thymus and spleen.

Acinar atrophy, exocrine pancreas, with necrosis and hyperplasia of exocrine ducts.

The lymphoid tissue was hypoplastic. The severe lymphoid hypoplasia coupled with lymphopenia and absence of serum IgM are compatible with combined immunodeficiency. Pancreatic changes are compatible with adenovirus infection.

AFIP DIAGNOSIS Spleen; thymus: Lymphoid hypoplasia, diffuse, severe, Arabian, equine.

Pancreas, ducts: Necrosis, multifocal, moderate, with hyperplasia, periductal fibrosis and basophilic intranuclear inclusions.

Pancreas, acini: Degranulation and atrophy, focally extensive, moderate.

CONFERENCE NOTE Combined immunodeficiency in Arabian foals is due to an autosomal recessive trait that is expressed as a defect in the lymphopoietic system at a level that involves both bone marrow-derived and thymic-dependent systems. Histologically, there is hypocellularity of all lymphoid tissue, including the thymus and spleen, as in this case. Affected foals are clinically normal at birth but develop a variety of opportunistic diseases within the first ten days of life. Adenoviral infections are particularly common in these foals and typically cause degeneration and necrosis of epithelial cells of the respiratory tract, the urinary tract, and lacrimal, salivary and pancreatic glands.

While all participants agreed there was necrosis of pancreatic ducts in this case, there was lack of unanimity in interpreting the focally extensive acinar pallor and disorganization. Some considered the lesion to be degenerative while others thought it to be regenerative. The Department of Endocrine Pathology diagnosed degranulation and atrophy as the predominant acinar change.

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MICROSLIDE 33

HISTORY This 8-year-old quarterhorse mare became ill after returning from one week of showing at a local fair. The animal developed severe diarrhea and laminitis. No clinical evidence of respiratory disease was evident at that time. Treatment consisted of phenylbutazone, flunixin meglumine, trimethoprim, and intestinal protective agents. The horse later died.

GROSS PATHOLOGY Large amounts of yellow to tan, turbid fluid was obtained from the pleural cavity. Occasional fibrin tags were adherent to the pleural surface of the lung. Numerous friable nodules, measuring 0.5 cm. in diameter and containing dark tan caseous material, were evident throughout the lung. The fecal contents of the small intestine and large colon were thin in consistency.

LABORATORY RESULTS Lung Cultures: Heavy growth - Aspergillus sp.

Fecal Cultures: Negative.

Immunofluorescence (lung): Aspergillus sp. positive. (Performed at CDC, Atlanta, GA).

Serum antibody titer Ehrlichia risticii: 1:2,560.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Severe multifocal to coalescing fibrinosuppurative necrotizing mycotic pneumonia with pleuritis. Aspergillus sp.

Sections of lung are included. Marked linear pleural accumulations of fibrin intermixed with bacterial colonies and necrotic debris are evident. Moderate to marked subpleural fibrosis is also seen. Numerous large multifocal to coalescing areas of necrosis are found throughout the sections. The necrotic areas are characterized by massive accumulations of neutrophils, cellular debris, fibrin, foci of mineralization, and lesser numbers of macrophages. Within many of the necrotic sites are large accumulations of fungal organisms characterized by basophilic, septate hyphae with parallel walls of a uniform width and dichotomous branching at acute angles. Numerous intravascular fibrin thrombi are seen within and adjacent to the necrotic foci. Intraluminal accumulations of variable numbers of red blood cells and cellular debris are evident in many of the bronchi and bronchioles. Variable amounts of fibrin and cellular debris are noted within alveolar spaces adjacent to the necrotic foci. Mild to moderate interstitial mineralization is also seen. Moderate growth of Aspergillus was obtained from the lung.

Pulmonary aspergillosis is relatively uncommon in the horse. This agent is more commonly associated with fetal abortions and guttural pouch infections in this species of animal. Circumstances that may have predisposed this animal to the fungal infection include exposure to

large numbers of infective fungal particles, immunosuppression, immunodeficiency and antibiotic suppression of normal bacterial flora. As association between enterocolitis and pulmonary aspergillosis has been reported (Slocombe, et al). It is felt that the enterocolitis contributes to invasive pulmonary aspergillosis by facilitating mycotic invasion from the gut and subsequent embolic mycotic lung disease.

AFIP DIAGNOSIS Lung: Pneumonia, necrotizing, multifocal to focally extensive, moderate, with infarction and intralesional fungal hyphae, quarterhorse, equine.

Lung, alveolar septa: Mineralization, multifocal, moderate.

Lung, pleura: Pleuritis, chronic, fibrinous, diffuse, severe.

CONFERENCE NOTE Although the contributor did not specifically state it, conference participants assumed that, based on the single high Ehrlichia risticii titer, the clinically diagnosed enterocolitis was attributed to Potomac horse fever.

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REFERENCES

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MICROSLIDE 34

HISTORY An adult Hereford cow was experimentally inoculated with Rift Valley Fever virus. She was euthanized two days post-inoculation.

GROSS PATHOLOGY Both on capsular and cut surface, the liver had multifocal, slightly depressed, hemorrhagic foci with irregular margins.

LABORATORY RESULTS None given.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Morphologic diagnosis: Multifocal hepatic necrosis, with hemorrhage. Etiology: Rift Valley Fever virus.

Rift Valley Fever is a mosquito-borne infection of significant morbidity and mortality in sheep, cattle, and humans in Africa. The etiologic agent is an RNA virus of the genus Phlebovirus, family Bunyaviridae. The disease is most severe in young animals, especially lambs, where mortality may reach 100%. In adults, infection usually results in a short febrile illness. Abortion storms are common.

In animals, the characteristic lesion is focal hepatic necrosis. Petechial to ecchymotic hemorrhages may be present on mucosal and serosal surfaces of many organs.

The disease in humans is seen where people have contact with infective mosquitoes. An outbreak in West Africa in 1987 is thought to be linked to a water reclamation project on the Senegal River which served to increase the number of mosquitoes in the region.

Currently, the principal protection against animal disease is the use of vaccines for livestock. Similarly, vaccination of livestock serves to reduce human exposure since sheep and cattle are amplifier hosts.

AFIP DIAGNOSIS Liver: Necrosis and hemorrhage, centrilobular and random, multifocal, moderate, Hereford, bovine.

CONFERENCE NOTE Participants considered the differential diagnosis to include toxic hepatoses, Wesselsbron disease, infectious bovine rhinotracheitis, blue-green algae intoxication, parasitic migration, and bacterial infections by Salmonella sp, Clostridium sp, Bacillus piliformis or Fusobacterium necrophorum.

The moderator commented that the Wister-Firth rat is very susceptible to the Rift Valley Fever virus and is an excellent animal model. The Lewis rat, on the other hand, is very resistant.

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REFERENCES

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MICROSLIDE 35

HISTORY This adult male domestic longhaired cat was recently adopted as a stray. On presentation to a veterinarian the cat was lethargic and had a temperature of 106.8, mild epistaxis and bulbar conjunctival petechiae. Within 24 hours the cat died while having a seizure.

GROSS PATHOLOGY There was marked splenomegaly, petechiation of the lung, epi- and endocardium, and generalized lymph node enlargement and hemorrhage.

LABORATORY RESULTS Rabies exam: negative. FeLV: negative.

Bacterial cultures of lung, liver, spleen, lymph node: no bacteria isolated.

CONTRIBUTOR'S DIAGNOSIS Liver, sinus histiocytosis, erythrophagocytosis.

Lung, hemorrhage/edema.

Lung, pneumonia, interstitial, acute.

Lymph node, hemorrhage, erythrophagocytosis.

Syndrome, cytauxzoonosis.

Vessel, parasitemia.

AFIP DIAGNOSIS Lung, blood vessels; liver, blood vessels and sinusoids; lymph node, blood vessels and sinuses: Protozoal schizonts, intrahistiocytic, multifocal, moderate, domestic longhair, feline.

Lung: Hemorrhage and edema, multifocal, mild.

CONFERENCE NOTE Cytauxzoonosis in the domestic cat has been reported primarily in the south central and southeastern United States. Domestic cats are thought to be accidental hosts for Cytauxzoon felis because of the rapid fatal course of natural infections. The primary host and complete life cycle are yet to be determined, but bobcats have been demonstrated to be asymptomatic carriers. Ticks are vectors. At necropsy infected cats show pallor, icterus, splenomegaly, and scattered petechial and ecchymotic hemorrhages. C. felis has an intraerythrocytic stage of 1.0 - 1.5 um ring forms and a schizont stage within mononuclear phagocytic cells. Schizonts may have characteristic multinucleated cytomeres or mature merozoites. Diagnosis is made on finding the schizonts in histiocytes within the vascular channels of various organs, including the lung, lymph nodes, liver, spleen and kidney. Parasitized erythrocytes are usually absent, and when present must be differentiated from Haemobartonella felis; this stage is best diagnosed on a blood smear. Haemobartonella felis rickettsial organisms are coccoid or rod-shaped bodies on the external periphery of the

erythrocyte membrane, while Cytauxzoon felis organisms are single signet-ring forms within erythrocytes.

Several participants observed numerous refractile circular structures within red cells in the lung hemorrhages and wondered if these may represent the intraerythrocytic form. The staff parasitologist commented that he did not think so as this form is non-refractile and rarely seen in tissue section. The structures may be Heinz bodies.

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MICROSLIDE 36

HISTORY This adult Döhne merino ewe became anorectic and developed nervous signs including ataxia and walking in circles with the head tilted to one side. It was necropsied 5 days after the onset of clinical signs.

GROSS PATHOLOGY Significant macroscopic changes were evident only in the central nervous system. The meninges covering the cerebrum and cerebellum were diffusely thickened and brownish-grey. On incision, almost the entire cerebrum and cerebellum appeared necrotic with a friable consistency and a brown discoloration.

LABORATORY RESULTS None given.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Meningoencephalitis, necrogranulomatous, subacute, diffuse, severe with vasculitis, thrombosis and amoebic trophozoites and cystic forms compatible with Acanthamoeba sp., Döhme merino, ovine.

The morphological characteristics and staining of the trophozoites and cysts in this animal conformed to the description for free-living amoebae of the genus Acanthamoeba. Differentiation of species in the genus Acanthamoeba on a morphological basis is difficult, if not impossible, and for this purpose immunoperoxidase and immunofluorescent staining techniques must be employed. The organisms in this case could be differentiated from Naegleria, since, in Naegleria infections, cysts are not formed in tissues of the host.

The nervous lesions resembled that of granulomatous amoebic encephalitis (GAE) caused by Acanthamoeba in man. This is in contrast to the more acute lesions of hemorrhagic necrosis with a minimal inflammatory reaction described in primary amoebic meningoencephalitis (PAM) caused by Naegleria. Infection with Naegleria usually occurs in healthy individuals via penetration through the cribriform plate. Acanthamoeba is usually seen in chronically ill or immunosuppressed individuals and is spread primarily intravascularly.

AFIP DIAGNOSIS Brain: Meningoencephalitis, necrotizing, chronic, diffuse, severe, with necrotizing vasculitis, protozoal trophozoites and cysts, Döhme merino, ovine.

CONFERENCE NOTE Most participants correctly identified the tissue as brain based on fragments of choroid plexus and scattered remnants of recognizable neuropil within most sections.

In many sections there were clearly delineated areas of necrosis and inflammation suggesting infarction. Trophozoites and cysts were demonstrated with a PAS reaction and were often clustered around necrotic vessels. Many participants remarked on the difficulty in differentiating neurons, gitter cells and amoebic trophozoites and cysts in this section.

Amebic meningoencephalitis caused by Lepto-mixed sp. has been recently reported in a sheep.

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MICROSLIDE 37

HISTORY This 21-year-old thoroughbred stallion had a long shaggy hair coat and was suspected to have a pituitary adenoma.

GROSS PATHOLOGY The hair coat was long and the skin of the extremities dry and crusty (hyperkeratotic). An adenoma was present in the pituitary gland. There was bilateral adrenal cortical hyperplasia.

LABORATORY RESULTS None given.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Epidermitis, suppurative, chronic, diffuse, moderately severe with acanthosis, parakeratosis, and branching filamentous bacterial organisms compatible with Dermatophilus sp., skin, thoroughbred, equine.

Dermatophilus congolensis is a keratinophilic actinomycete that causes epidermitis or dermatitis in a wide range of animal species. Occasionally the squamous mucosa of the oral cavity or esophagus will be affected. Lymph node granulomas and subcutaneous infections have also been reported. The organism invades and proliferates within the epidermis as branching filaments which divide in characteristic multi-dimensional fashion, often giving rise to multiple rows of coccoid organisms which develop into motile zoospores. Dermatophilus stimulates an acute inflammatory response in the epidermis which inhibits deeper penetration of the organisms. As the epidermis is regenerated, the bacteria invade the new tissue, thus creating the characteristic pattern of alternating inflammation and hyperkeratosis.

AFIP DIAGNOSIS Haired skin: Epidermitis, hyperplastic, diffuse, severe, with mild subacute dermatitis and intralesional filamentous bacteria, thoroughbred, equine.

CONFERENCE NOTE Discussion centered on pituitary adenomas in horses; adenomas of the pars intermedia are the most commonly reported tumors. The clinical syndrome of polyuria, polydipsia, increased appetite, muscle weakness, somnolence, hyperpyrexia, hyperhidrosis and hirsutism is associated with compression of the hypothalamus from the expanding tumor; adenomas of the pars intermedia expand dorsally in the horse due to the incomplete diaphragma sellae. Plasma cortisol is reported only modestly elevated in these horses, however, it is not suppressed by either high or low dose dexamethasone.

What role, if any, the pituitary adenoma played in the development of dermatophilosis in this horse is speculative.

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MICROSLIDE 38

HISTORY This 2-year-old male Akita dog was presented because of red eyes and gradual vision loss followed by progressive periocular and nasal alopecia and depigmentation. Ocular and dermal changes were observed, but not treated, 4 months prior to euthanasia.

GROSS PATHOLOGY The globes were buphthalmic and the cornea was roughened. Cross sections revealed a thick grey-white uvea, iris bombe, a corneal stria and a firm red-white vitreous.

LABORATORY RESULTS None given.

CONTRIBUTORS DIAGNOSIS AND COMMENT Vogt-Koyanagi-Harada like syndrome or uveitis-depigmentation syndrome.

Chronic diffuse severe granulomatous panuveitis.

Uveal depigmentation.

Chronic keratitis, posterior corneal scarring.

Cataractous changes.

Retinal necrosis and detachment.

The cornea is markedly thickened and there are focal areas of mild corneal epithelial hyperplasia. The substantia propria is thickened due to fluid accumulation and neovascularization. Descemet's membrane is ruptured and the break is covered with fibrous connective tissue. The entire uvea is markedly thickened by accumulations of macrophages, lymphocytes and fewer plasma cells that surround most of the pigmented uveal cells. Some of the melanocytes are necrotic and partially degranulated. There is a diffuse subretinal exudate with complete retinal detachment. The lens contains numerous bladder cells near the equator. Hyaloid bodies and focal areas of hemorrhage are present in the vitreous.

AFIP DIAGNOSIS Eye: Panuveitis, necrotizing, granulomatous, chronic, diffuse, severe, with retinal necrosis, cataractous change, vitreal hemorrhage and chronic keratitis, Akita, canine.

CONFERENCE NOTE Miguel N. Burnier, Jr., M.D., of the Sao Paulo (Brazil) School of Medicine and currently a staff fellow in the Department of Ophthalmic Pathology, gave a presentation comparing Vogt-Koyanagi-Harada (VKH) syndrome in humans with similar conditions seen in dogs. VKH is seen in young Oriental, black or Hispanic adults and is characterized by uveitis, chorioretinitis, poliosis (whitening of the hair), vitiligo (depigmentation of the skin), hearing loss and meningitis. VKH syndrome is believed to be an immune-mediated disease in which melanocytes are the target cells. The veterinary literature reports a few cases of a VKH-like syndrome in dogs. The condition was initially described in Akitas from Japan, although it has now been seen in other breeds. In dogs the syndrome is also characterized by uveitis, poliosis and vitiligo; meningitis and hearing disturbances, however, have not been described. Histologically, ocular lesions are as seen in this case, with prominent granulomatous uveitis, pigment-laden cells, retinal detachment and destruction of the retinal pigmented epithelium. The characteristic skin lesion is lichenoid interface dermatitis with decreased or absent melanin in the epidermis and hair follicles.

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MICROSLIDES 39 and 40

HISTORY This 1-week-old, male, mixed breed beef calf was born without incident and nursed. It was less active than normal since birth but no other clinical signs were observed. It was found dead.

GROSS PATHOLOGY Fecal material was adhered to the tail and perineum, and the contents of the intestine and colon were yellow and watery. The liver was mottled with alternating tan and dark red foci. The ventricular myocardium contained several pale foci 5-10 mm in diameter. Small pale foci were also found within the cortex of the left kidney. The lungs were diffusely

congested and edematous.

LABORATORY RESULTS Listeria monocytogenes was isolated from liver and lung. ELISA test, feces: positive for rotavirus.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Hepatitis, necrotizing, subacute, multifocal to coalescing, severe, with gram-positive bacterial rods. Etiology: Listeria monocytogenes.

The liver had extensive multifocal to coalescing areas of necrosis rimmed by dense accumulations of degenerating neutrophils and mononuclear inflammatory cells. Numerous small rod-shaped bacteria could be seen, particularly among the inflammatory cells surrounding the areas of necrosis. Similar though less extensive lesions were in the kidneys and myocardium.

AFIP DIAGNOSIS Liver: Hepatitis, necrotizing, acute, random, multifocal to coalescing, with gram-positive bacilli, mixed-breed, bovine. Etiology: consistent with Listeria monocytogenes.

CONFERENCE NOTE The pathogenesis and epidemiology of listeriosis are poorly understood. Infection is manifested by abortion, septicemia, or encephalitis; each appears to be a separate syndrome. A localized uterine infection is thought to be hematogenous following oral exposure of the pregnant animal; abortion occurs late in pregnancy. Septicemia in ruminants is primarily a disease of neonates, most likely acquired in utero. Hepatic abscessation is the principal lesion in the septicemic form with variable involvement of the heart and other viscera. Encephalitis affects the brainstem; ascending neuritis of the trigeminal nerve has been implicated in the pathogenesis. Outbreaks have been associated with feeding of silage; the organism will multiply in incompletely fermented silage with a pH of greater than 5.5. Listerial mastitis has also been reported. This has important zoonotic implications as there is some concern that organisms may survive some types of pasteurization within leukocytes.

The staff of the Department of Infectious and Parasitic Diseases did not hesitate to diagnose Listeria monocytogenes based on the uniform population of numerous, small gram-positive pairs of rods. Other gram-positive rods included in the differential diagnosis are Actinomyces pyogenes, which is larger and more pleomorphic than Listeria, and Erysipelothrix, which, while similar in size to Listeria, is more pleomorphic and usually present in smaller numbers.

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MICROSLIDE 41

HISTORY This 2-year-old male poodle was anorectic and lethargic for several weeks. The dog had a Borrelia burgdorferi titer greater than 1:1000. One year prior the dog also had an elevated Borrelia titer and difficulty opening its mouth. The dog responded to tetracycline given at that time.

GROSS PATHOLOGY Kidney is pale tan and the cortex has a fine granular appearance.

LABORATORY RESULTS

WBC 23.1	In Phos 12.7	Urinalysis
Segs 84%	BUN 159	S.G. 1.021
Lymphs 6%	SGOT 186	pH 6.5
Monos 7%	Creatinine 6.7	Protein 952 mg/dl
Bands 1	Cholesterol 340	Creatinine 73.0 mg/dl
Eos 2%	T.P. 5.1	Blood 3+
	Alb 2.1	

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Glomerulonephritis, subacute to chronic, mesangioproliferative, marked, generalized and diffuse with proteinuria and crescent formation.

Tubular degeneration and necrosis, moderate, multifocal, acute.

The prior history of arthritis associated with a persistently elevated Borellia burgdorferi titer is compatible with a diagnosis of Lyme disease. Glomerular lesions have been reported in association with Lyme disease in the dog and may be due to immune complex deposition.

AFIP DIAGNOSIS Kidney: Glomerulonephritis, mesangioproliferative, diffuse, moderate to severe, with crescent formation, mineralization and secondary tubulointerstitial changes, poodle, canine.

CONFERENCE NOTE Lyme disease is an immune-mediated disease of people initiated by the spirochete Borrelia burgdorferi and characterized by a primary skin lesion (erythema chronicum migrans) that may be followed by cardiac, neurologic or arthritic complications. The organism is transmitted by Ixodes sp hard ticks. The natural host of the larval and nymph stages is the white-footed mouse while the host of the adult stage is the white-tailed deer. Borreliosis has also been reported in dogs; clinical signs in dogs include arthritis, arthralgia, fever, and anorexia. Recently there have been reports in dogs associating glomerulonephritis with positive titers for B. burgdorferi.

In this case, the glomerulonephritis is considered mesangioproliferative based on the prominent cellularity, accumulation of mesangial matrix and lobularity of the glomerular tufts. Crescent formation, indicative of severe glomerular damage, is pronounced. Secondary changes include

tubular ectasia, tubular necrosis and regeneration, and chronic interstitial nephritis.

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MICROSLIDE 42, 35 MM SLIDE 8

HISTORY This 2-year-old mixed-breed steer was in a group of cattle receiving sorghum as feed supplementation. Thirty-nine animals died with a clinical disease characterized by initial diarrhea followed by progressive muscular incapacitation, recumbency and death. Dark brown discoloration of the urine was also a consistent clinical finding. Animals, in spite of recumbency, kept their appetite until approximately 24 hours before death. Mainly mature cattle were affected. The clinical course varied from 4 days to 2 weeks. Some animals recovered. The sorghum which was fed to these animals revealed a contamination by Cassia occidentalis (coffee senna) seeds in the range of 10%.

GROSS PATHOLOGY There were pale areas of discoloration of the skeletal muscles mainly of the heavy muscles of the upper hind limbs, shoulders and diaphragms. Some of these areas had a focal distribution, others were diffusely distributed along the muscle groups as white-tan streaks. The heart was pale. The liver was enlarged with a nutmeg aspect in the cut surface. A mild degree of pulmonary edema was evident.

LABORATORY RESULTS There was marked elevation in CPK and SGOT levels in the serum. No determination for myoglobin was carried out in the urine.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Degenerative myopathy, toxic. Etiology: Cassia occidentalis poisoning, bovine, skeletal muscle.

Cassia occidentalis (coffee senna) is a well known poisonous plant which induces mainly myopathy and cardiomyopathy in cattle and other species. The characteristic lesion of hyaline degeneration with swollen, eosinophilic, occasionally ruptured, myofibers can be observed in the sections. The elevated serum enzymes (CPK, SGOT) found in this case are common findings in this disease. The dark-brown discoloration of the urine is described as being imparted by myoglobin from muscle breakdown. Other histological lesions observed in this case were centrilobular congestion of the liver with vacuolation of the cytoplasm of hepatocytes, fine vacuolation of myocardial fibers and mild lung edema. The source of coffee senna in this case was the contamination of the sorghum by the seeds of the poisonous plant which was then fed to the animals. The seeds of coffee senna are very similar in shape and density to the sorghum seeds. The contamination occurs during mechanical harvest of the sorghum crops. The disease in this case was later reproduced in calves using the contaminated ration. The lesions of this toxic myopathy are similar to those found in nutritional myopathy of calves (Se/vit. E deficiency). Important points in the differential diagnosis are that nutritional myopathy occurs in young calves, the muscle lesions have marked mineralization and the heart lesions are usually more marked.

AFIP DIAGNOSIS Skeletal muscle: Degeneration and necrosis, acute, diffuse, severe, mixed-breed, bovine.

Skeletal muscle: Sarcocystis, multiple.

CONFERENCE NOTE In addition to C. occidentalis toxicity, the differential diagnosis for acute myodegeneration and necrosis in cattle includes ionophore (monensin) toxicity, gossypol toxicity, coyotillo toxicity, metal toxicity, exertional myopathy, nutritional (vitamin E/selenium deficiency) myopathy and trauma.

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MICROSLIDES 43 and 44, 35 MM SLIDE 9

HISTORY This 18-month-old crossbred bull was born and raised at the National Animal Disease Center. At 5 months of age, a preinoculation blood sample was collected and he was inoculated with bovine immunodeficiency-like virus (BIV) as part of an experimental protocol. He subsequently became seropositive to BIV and BIV was reisolated. Later, he was turned out with a group of heifers for breeding. He suddenly developed bloody diarrhea and a slight serous nasal discharge and died three days later.

GROSS PATHOLOGY At necropsy, there were linear esophageal erosions and multifocal hemorrhagic ulcers of the abomasum, ileum and proximal colon. The distal colon contained casts of fibrinohemorrhagic and necrotic material.

LABORATORY RESULTS Noncytopathic bovine viral diarrhea (BVD) virus was isolated from a thawed serum sample collected from the bull before BIV inoculation. At necropsy cytopathic BVD virus mixed with noncytopathic BVD virus were isolated from the esophagus. Attempts to isolate BIV from tissues collected at necropsy were unsuccessful. No salmonella could be isolated from ileum, colon or mesenteric lymph node. The bull's serum contained antibodies that reacted with several viral induced proteins of the cytopathic virus. Immunohistochemistry using monoclonal antibodies demonstrated BVD viral antigen in colonic crypts, GALT and myenteric ganglion cells and in esophageal mucosal cells.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Esophagitis, subacute, multifocal, moderate, with erosions and superficial bacterial colonies, esophagus, etiology bovine viral diarrhea virus, and secondary bacterial colonization.

Colitis, subacute, segmental, moderate, with multifocal erosions, distension of glands and crypts and lymphocytic depletion, proximal colon, etiology bovine viral diarrhea virus.

The bull died of the acute mucosal disease form of bovine viral diarrhea. Mucosal disease is caused by cytopathic BVD viral superinfection of a bovine persistently infected with noncytopathic BVD virus. Persistent infections result from early in utero infection with noncytopathic virus. The bull appeared healthy and was not known to be persistently infected when he was entered into the experimental protocol for BIV. After he died, he was retrospectively diagnosed as having been persistently infected when a thawed serum sample yielded noncytopathic BVD virus This case points out the havoc that can be caused by undetected concurrent or intercurrent viral infections in research animals.

AFIP DIAGNOSIS Esophagus: Esophagitis, ulcerative, subacute, multifocal, moderate, crossbred, bovine.

Colon: Colitis, necrotizing, subacute, diffuse, moderate, with crypt abscesses.

CONFERENCE NOTE Bovine viral diarrhea (BVD) virus infection causes a wide spectrum of disease depending largely on the age and immune status of infected cattle. Infections are usually subclinical in susceptible immunocompetent adult cattle. Less commonly, bovine viral diarrhea, an acute disease characterized by mild depression, inappetence, oculonasal discharge, oral erosions and diarrhea occurs. Morbidity may be high, but mortality is very low. When a susceptible cow or heifer is infected during pregnancy, transplacental infection is

common. The fetal response is primarily determined by the age of the fetus. Fetal infection from 50-100 days of gestation may result in fetal death, followed by abortion or mummification. Fetal infection between 100 and 150 days often results in a variety of teratogenic lesions. Infection with noncytopathic BVD virus early in fetal development, prior to immunocompetence (which occurs at 150-200 days) may result in immunotolerance to the infecting strain of BVD virus. After birth, these cattle are persistently infected, viremic, constantly shed virus and may appear healthy. If such cattle are subsequently exposed to cytopathic BVDV, acute or chronic mucosal disease may result.

Bovine immunodeficiency-like virus is a recently recognized lentivirus of cattle. The effects of infection with this virus have not yet been fully determined.

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MICROSLIDE 45

HISTORY This adult female crossbred goat was experimentally inoculated with goatpox virus. She was euthanized two weeks post-inoculation.

GROSS PATHOLOGY There were raised, occasionally ulcerated nodules over large areas of the skin surface. The lungs contained several sub-pleural foci of consolidation and failed to collapse normally.

LABORATORY RESULTS None given.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Dermatitis, proliferative, necrotizing, subacute, severe. Etiology: Goatpox virus.

The capripox genus of the Poxviridae family consists of three closely related viruses - goatpox, sheeppox, and bovine lumpy skin disease. All cause similar clinicopathologic syndromes in their respective species, with generalized cutaneous eruptions often accompanied by dissemination to internal organs, especially lung.

Goatpox is endemic in Africa, the Middle East and Asia. Transmission occurs by aerosol, contact, and arthropods. The virus is relatively stable in the environment and can survive in scab material for three to six months. Control is achieved through restricted animal movement, reduction of insects, and vaccination.

A characteristic histologic feature of all three viruses of the capripox genus is the presence of variable numbers of "sheeppox" cells. These are histiocyte-like cells with large vacuolated nuclei with marginated chromatin and often eosinophilic intracytoplasmic inclusions.

AFIP DIAGNOSIS Haired skin: Dermatitis, necrotizing, subacute, focally extensive, severe, with epidermal hyperplasia, ballooning degeneration and microvesiculation, vasculitis and eosinophilic intracytoplasmic inclusions in epidermal and mesenchymal cells, crossbred, caprine.

CONFERENCE NOTE In addition to the capripox genus, severe systemic disease also occurs in monkeypox, fowlpox and ectromelia. Poxvirus infections associated with hyperplastic and neoplastic lesions include Shope fibroma virus of rabbits, squirrel fibroma, Yaba disease of African monkeys, myxomatosis of European rabbits and molluscum contagiosum.

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MICROSLIDE 46

HISTORY Ten percent of a mob of heifers were depressed, reluctant to stand in direct sunlight and had yellow mucus membranes. This 8-month-old Friesian heifer died and was necropsied.

GROSS PATHOLOGY The liver was firm, orange/tan in color and had a prominent lobular pattern.

LABORATORY RESULTS None given.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Subacute hepatopathy with cholestasis, partial fibrosis, biliary ductular hyperplasia, fibrous replacement and recanalization of large and medium-sized intrahepatic bile ducts, multifocal hepatocellular necrosis and bile pooling. Etiology: Sporidesmin toxicity.

Sporidesmin is a potent mycotoxin produced by Pithyomyces chartarum and is the cause of facial eczema, a serious production-limiting disease of ruminants in New Zealand and to a lesser extent in Southern Australia and South Africa. The fungus proliferates on pasture litter when conditions are favorable (warm and moist) and spores containing sporidesmin are ingested by grazing animals. The primary hepatic lesion is a necrotizing cholangitis involving the large and medium-sized intrahepatic and extrahepatic bile ducts. Reparative changes characterized by fibrosis and recanalization are a feature of subacute and chronic cases and portal triads become expanded with fibrous tissue and bile ductules.

Multifocal necrosis and bile pooling is an inconsistent but well-recognized lesion of sporidesmin toxicity.

AFIP DIAGNOSIS Liver: Fibrosis, portal and bridging, diffuse, with bile duct proliferation, biliary stasis and random hepatocellular necrosis, Friesian, bovine.

CONFERENCE NOTE Conference participants included Lantana camara intoxication, aflatoxicosis and pyrrolizidine alkaloid toxicosis in the differential diagnosis. Hepatocellular megalocytosis with fine cytoplasmic vacuolization is seen in Lantana poisoning. Megalocytosis is also a prominent feature in pyrrolizidine alkaloid toxicosis. Acute cholangitis or cholangiohepatitis are considered characteristic lesions of acute sporidesmin toxicosis. As this case illustrates, multifocal hepatocellular necrosis and bile pooling are characteristic chronic lesions.

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MICROSLIDE 47

HISTORY An acute outbreak of afebrile paresis in feeder pigs between 8 to 10 weeks of age was diagnosed. Experimental reproduction of the disease was attempted in 5 week old pigs, using a compound similar to that found in the feed ration of the field outbreak. This tissue is from a 5-week-old female mixed breed pig.

GROSS PATHOLOGY Spinal cord segments C6-C8 and L5-L7 contained focal, well delineated areas of discoloration within the ventral grey horns.

LABORATORY RESULTS Whole blood selenium at day 10 post inoculation was 2.556 ppm. (Controls were 0.23 ppm.)

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Severe, focal, symmetrical poliomyelomalacia - C6-C8, L5-L7.

The sections submitted were from 5 week old pigs orally inoculated with 2.86 mg/kg sodium selenite and euthanized 10 days later.

The lesions within the spinal cord were restricted to the cervical and lumbar spinal cord segments. Similar lesions were also seen within the brain (fastigial cerebellar nuclei, and interpositional cerebellar nuclei). The lesions within the spinal cord were well delineated and restricted to the ventral grey horns. Severe microcavitation was evident, with marked endothelial cell proliferation also seen. Neuronal degeneration and necrosis, evidenced by increased cytoplasmic acidophilia and chromatolysis was also present. Severe neuronal loss was found in many of the sections. Mild to moderate microgliosis intermixed with small foci of hemorrhage were found in the majority of the sections. Occasional eosinophils and mononuclear inflammatory cells were found scattered throughout the lesions. The lesions found within these experimentally inoculated pigs were similar to those found in the natural disease found in the field outbreak described.

AFIP DIAGNOSIS Spinal cord: Poliomyelomalacia, bilaterally symmetrical, focally extensive, moderate, with endothelial proliferation, mixed-breed, porcine.

CONFERENCE NOTE In addition to selenium toxicity, 6-aminonicotinamide (an antimetabolite of niacin) has been reported to produce similar lesions in swine. Acute selenium poisoning in herbivores is reported to cause an acute congestive and enteric disease, characterized by hemorrhagic enteritis and proctitis, passive congestion of the lungs and abdominal viscera, acute toxic hepatitis and acute tubular necrosis. Death is caused by respiratory and myocardial failure. Chronic selenium poisoning in herbivores has been described under two distinct syndromes, "blind staggers" and "alkali disease".

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MICROSLIDE 48, 35 MM SLIDE 10 and 11

HISTORY This 11-year-old female baboon was one out of approximately 92 baboons that died during an eight month epizootic at a primate facility. Clinical disease, when detected, was consistent with acute congestive heart failure.

GROSS PATHOLOGY The salient necropsy findings were pulmonary congestion and edema, hydropericardium, hydrothorax, ascites and pale white to tan mottled hearts.

LABORATORY RESULTS No significant bacteria were isolated.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Myocarditis, nonsuppurative, necrotizing, heart, baboon, etiology encephalomyocarditis virus (Picornavirus).

Diagnosis was confirmed by light microscopy, transmission electron microscopy, viral culture and serology. Feral rats were the source of the virus and their control along with improved sanitation stopped the epizootic.

The 2x2 color slide illustrates the typical pale myocardium, congested, edematous lungs and excess fluid in the pericardial sac. The EM slide illustrates the EMCV in the myocardium of a baboon.

AFIP DIAGNOSIS Heart: Myocarditis, necrotizing, subacute, multifocal, mild, with myocardial mineralization, baboon, non-human primate.

CONFERENCE NOTE Encephalomyocarditis virus (EMCV) belongs to the family Picornaviridae and the genus Cardiovirus. Rats are the most common reservoir host with EMCV persisting in the gut for extended periods. Target host species include primates, mice, squirrels, elephants, cattle, raccoons, and swine. The mechanism of transmission is not clear, but ingestion of sick or dead rodents and fecal/urine contamination of food and water are the likely sources of infection.

Participants discussed the various target organs of this virus. Most animals develop myocardial lesions, but in some species (mice and owl monkeys) encephalitis also occurs. In the squirrel monkey mild myocardial and CNS changes occur while more pronounced lesions are observed in the exocrine pancreas.

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MICROSLIDE 49

HISTORY This is tissue from an adult male Sprague-Dawley rat that developed chromodacryorrhea and respiratory rales. The rat was thin, hunched, dyspneic and its hair coat was ruffled.

GROSS PATHOLOGY The lungs failed to collapse and were consolidated in the anteroventral area. The consolidated regions were discolored red and the pleural surface was dull and granular.

LABORATORY RESULTS Corynebacterium kutscheri was cultured from the lungs.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Pneumonia, necrotizing, acute, multifocal, severe. Etiology: Corynebacterium kutscheri.

Microscopic lesions consist of multifocal to coalescing areas of coagulation necrosis. The necrotic regions contain numerous degenerated neutrophils admixed with prominent aggregates

of bacteria. Some necrotic foci include necrotic bronchioles plugged with degenerated inflammatory cell debris. Associated vessels are thrombosed. Surviving alveoli contain moderate amounts of protein-rich edema fluid.

Gross lesions of pseudotuberculosis, caused by Corynebacterium kutscheri, occur most often in the lung. However, pleuritis, pericarditis, hepatitis and nephritis may accompany pulmonary lesions.

Infections with Corynebacterium kutscheri in rats often are subclinical; fulminating disease occurs after experimental manipulations or stresses which lower the host's resistance. No inciting stressor was identified to account for this epizootic.

AFIP DIAGNOSIS Lung: Pneumonia, necrosuppurative, multifocal to coalescing, severe, with large bacterial colonies, rat, rodent.

CONFERENCE NOTE Corynebacteria are highly pleomorphic gram-positive bacilli which range from straight or slightly curved rods to club-shaped and filamentous forms. Near completion of cell division a movement designated as "snapping" occurs and the bacilli may remain attached at sharp angles to one another in a palisade or picket fence arrangement; groups of these cells are said to resemble Chinese letters. This disease is usually one of low morbidity and mortality with inapparent infection more common in mice than rats. However, outbreaks with high morbidity and mortality do occur, usually following or in association with stress, cortisone administration, irradiation, dietary deficiencies (i.e. pantothenic acid or biotin) and with other infections (i.e. ectromelia, salmonella, or mycoplasma). Cases have been reported without any known predisposing factors.

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MICROSLIDE 50, 35 MM SLIDE 12

HISTORY This adult wild caught male owl monkey was experimentally infected with Plasmodium falciparum and treated successfully 3 years previously. No other procedures had been performed since that time. The animal was found dead one morning with no history of any clinical illness.

GROSS PATHOLOGY Mild dehydration was evident. Little subcutaneous or abdominal body fat was present. Generalized muscle atrophy was noted. The liver was slightly yellowish brown and mildly friable. No other gross lesions were apparent.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Liver, hepatopathy, diffuse, moderate to severe, with intrasinusoidal and intracellular yeast-like organisms.

Hemosiderosis, moderate.

Lipidosis, multifocal, mild to moderate.

Etiology: Blastomyces dermatitidis.

Numerous 2-6 micron diameter budding yeast-like organisms were present in the sinusoids and within the cytoplasm of hepatocytes and Kupffer cells. Organisms were also prominent within all other tissues examined including the interstitium of the kidneys, adrenal glands, lung, heart and sinusoids of the spleen. The organisms had thick cell walls, central basophilic structures (retracted cytoplasm) and formed single broad-based buds. Direct immunofluorescence performed at the CDC was positive for Blastomyces dermatitidis. Small forms (2-6u) of Blastomyces, usually admixed with a majority of larger forms, have been reported in man and nonhuman primates. This case was unusual in that the small form predominated and there was no demonstrable host reaction. Differential diagnosis included Histoplasma capsulatum var. capsulatum, Torulopsis glabrata, and Cryptococcus neoformans. Mucicarmine stains were repeatedly negative. Histoplasma and Torulopsis have thinner cell walls and narrow based buds.

AFIP DIAGNOSIS Liver: Myriads of intrahepatocellular, intrahistiocytic, and extracellular yeast, owl monkey, Aotus trivirgatus.

Liver: Hemosiderosis, diffuse, moderate.

CONFERENCE NOTE Conference participants had difficulty in identifying the organism based on histomorphology; Blastomyces and Histoplasma were the fungi most commonly diagnosed. The staff of the Department of Veterinary Pathology believes that this fungus is histomorphologically most consistent with Histoplasma capsulatum. Morphologic justification for this diagnosis includes: the predominance of narrow-based budding; the presence of only one nucleus; a tendency for the yeasts to form pseudohyphae; and, the presence of host cells packed with yeasts. Obviously, a definitive diagnosis cannot be made without culturing the organism.

In some sections the yeasts are strongly anisotropic, a feature occasionally observed with yeasts and algae.

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MICROSLIDE 51

HISTORY This aged female rhesus monkey (Macaca mulatta) was culled from the primate colony.

GROSS PATHOLOGY There were multifocal gray nodules in pulmonary parenchyma.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Bronchitis/bronchiolitis, granulomatous, chronic, multifocal, moderate. Etiology: Pneumonyssus simicola (presumptive).

Lung mite infestation is relatively common in wild-caught rhesus monkeys. It is localized to bronchi/bronchioles and adjacent alveoli, and sections of the mite are commonly observed. The respiratory epithelium is usually denuded or attenuated. The wall of the lung mite cyst consists of lymphocytes, plasma cells, eosinophils, and pigmented macrophages. Foci of pigmented macrophages, also containing anisotropic spicules, can be observed at sites distant from the principal lesion, and oftentimes, they are the only indication of current or previous infestation.

AFIP DIAGNOSIS Lung: Bronchiolitis and parabronchiolitis, eosinophilic and granulomatous, chronic, multifocal, moderate, with bronchiectasia and intraluminal arthropods, rhesus monkey, non-human primate. Etiology: consistent with Pneumonyssus sp.

CONFERENCE NOTE Respiratory mites, including nasal mites, have been found in at least 22 species of Old World monkeys. New World monkeys are relatively free of these parasites, although they have been reported in Lagothrix (woolly monkey) and Alouatta (howler monkey) species. Wild macaques and baboons have a particularly high rate of infestation by these parasites with incidence levels often in the range of 80-100%. Many feel that all wild-caught rhesus monkeys should be considered infested until proven otherwise. Because of this and

associated tissue changes, some researchers do not consider rhesus monkeys suitable for inhalation studies. Respiratory mites in primates are classified in the order Mesoshigamata, family Halarachinidae and species Pneumonyssus. Several species of mites in the genus Pneumonyssus infect the respiratory system of Old World primates as do at least two known species of Rhinophagia (may cause nasal polyps). Although several of these mites are considered host specific, P. simicola can naturally infect the lungs of at least four species of macaques, and is the most common mite in the rhesus monkey. Concurrent infestation by more than one species of respiratory mite is common in baboons.

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MICROSLIDE 52

HISTORY This adult male common marmoset (Callithrix jacchus) was one of two marmosets found dead in cage following 3 days of transport and 2 days of extensive handling.

GROSS PATHOLOGY The kidneys were bilaterally swollen and white, with multifocal depressions on the capsular surface.

LABORATORY RESULTS Culture/sensitivity of blood and visceral organs was negative.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Inflammation, tubulointerstitial, chronic, diffuse, severe, kidney.

Glomerulonephritis, chronic, diffuse, marked, kidney.

Severe microscopic glomerulonephritis/tubulointerstitial nephritis is commonly seen in New World primates. A high incidence of IgM mesangial glomerulopathy in marmosets has recently been reported. While our facility lacks the capability to confirm the diagnosis via immunohistochemical staining for IgM and complement, the microscopic features present to support the diagnosis include:

Glomerular changes: Thickened Bowman's capsule; hypertrophic parietal epithelial cells; expanded Bowman's space; mesangial hyperplasia with increased mesangial matrix; glomerular synechia; occasional sclerotic glomeruli.

Tubular changes: Dilatation with attenuated epithelium; tubular epithelial degeneration and necrosis; mineralization; loss of tubules; regeneration/hyperplasia; proteinaceous casts.

Interstitial changes: Fibrosis; heavy lymphocytic infiltrate with lesser plasma cells and eosinophils.

The stress of transport and handling most likely accounted for decompensation and death due to renal failure.

AFIP DIAGNOSIS Kidney: Nephritis, interstitial, chronic, diffuse, severe, with marked fibrosis and multifocal mineralization, common marmoset (Callithrix jacchus), non-human primate.

Kidney, interstitium: Hemosiderosis, multifocal, mild.

CONFERENCE NOTE While several attendees believed the lesions to be consistent with the reported IgM-mesangial nephropathy of callithricids, others believed that the relatively mild glomerular lesions could be secondary to the interstitial nephritis. Immunocytochemical techniques were employed in an attempt to demonstrate immunoglobulins within mesangial cells and matrix, but the results were .noncontributory. In the opinion of the Division of Nephropathology, the glomerular changes are nonspecific; they might have been mediated by IgM accumulation or they could be secondary changes.

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MICROSLIDE 53

HISTORY This tissue is from either of 2 adult Zebu-breed cattle. The cattle were reared in the northern Transvaal region (bushveld area) of South Africa. Both suddenly developed nervous signs, in particular circling to one side, and died within 3 weeks of the development of clinical signs.

GROSS PATHOLOGY No significant gross pathology was noted except for the brain which showed focal areas of encephalomalacia with associated hemorrhage or yellow discoloration. The superficial meningeal vessels were greatly thickened and thrombosed.

LABORATORY RESULTS Examination of a smear from a thrombosed blood vessel of the brain revealed the presence of numerous schizonts within large lymphoblasts or loose among the cells.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Cerebral infarction, subacute, focally extensive, with focal hemorrhage, thrombosis and vascular proliferation and numerous schizonts compatible with Theileria spp. within large lymphoblasts in blood vessels, bovine.

Theileria are tick-transmitted parasitic protozoa of the family Theleiriidae. The taxonomy of the genus at species level is still the subject of much controversy. In southern Africa several Theileria species and subspecies have been described in cattle. These include Theileria parva parva - the cause of East Coast Fever; T parva lawrencei - Corridor Disease; T. parva bovis - Zimbabwe theileriosis; T. mutans - which is generally nonpathogenic; T. (Cytauxzoon) taurotragi - which is generally nonpathogenic but has been associated with brown tick toxicosis and cerebral theileriosis and T. (Haematoxinus) velifera which is also nonpathogenic.

In southern Africa cerebral theileriosis (turning sickness) is an aberrant form of T. taurotragi infection characterized by the accumulation of parasitized lymphoblasts in the cerebral, spinal and meningeal arteries with resultant thrombosis and infarction and the development of nervous signs characterized in particular by circling. Other symptoms such as head pressing, blindness, ataxia, paralysis and opisthotonos can occur. Death usually occurs within 2-21 days of the development of symptoms. Parasitization of splenic vessels may occasionally occur. It is not known why the cerebral vessels should be preferentially affected but the condition generally arises after massive infection of a partially immune animal and is thought to be related to intercurrent infection or stress in a chronically infected animal.

Diagnosis is based on the region of origin of the affected animal and the typical clinical signs and pathological lesions. The diagnosis is confirmed by the demonstration of large numbers of

schizonts, loose or within lymphoblasts in smears from thrombosed vessels or in histological sections from cerebral lesions. In chronic cases schizonts may be difficult to find.

AFIP DIAGNOSIS Cerebrum: Encephalomalacia, multifocal, severe, with vasculitis, thrombosis and intravascular lymphoblasts containing protozoa, Zebu, bovine.

CONFERENCE NOTE Sporozoite of tick origin invade and infect host lymphocytes. Infected lymphocytes are transformed into lymphoblastic cells. Macroschizonts develop in the cytoplasm of the blastic cells and divide in synchrony with the cells to infect progeny cells.

Lymphoblastic lymphoid leukemia is the primary histologic differential.

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MICROSLIDE 54

HISTORY This 12-week-old raccoon (Procyon lotor) had been raised at a wildlife referral center since infancy. There was a several week history of dyspnea and sneezing. Clinical signs were unresponsive to antibiotic therapy and the animal progressively lost weight and became increasingly lethargic. The animal died naturally.

GROSS PATHOLOGY Necropsy findings included muscular and visceral pallor, noncollapsed lungs, and an enlarged gallbladder.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Cholecystitis, eosinophilic and lymphohistiocytic, severe, widespread, subacute, with apical epithelial protozoal forms and intranuclear/intracytoplasmic inclusion bodies, gallbladder, raccoon (Procyon lotor). Etiology: Cryptosporidium sp. and canine distemper virus.

Cryptosporidiosis has previously been documented in the raccoon. In that report, there was no

clinical disease but marked histopathologic changes were reported in the small intestine -- involvement of the gallbladder was not mentioned. Cryptosporidial cholecystitis has been described in man and the organisms have been noted in the biliary tract of humans and animals with gastrointestinal cryptosporidiosis. In one description of cryptosporidial infection in a juvenile rhesus macaque, there was striking involvement of the hepatobiliary tract with lesser involvement of the gallbladder. The typical proprial infiltrates associated with cryptosporidial infection are mononuclear and neutrophilic. The prominent eosinophilic component in the current case is unusual in this respect - interestingly, the above mentioned report in a raccoon describes similar eosinophilic infiltrates associated with intestinal cryptosporidiosis. Certainly it must be considered that lesions in the gallbladder may have been due to an agent not apparent in the specimen examined and that the cryptosporidia are concomitant or secondary invaders. The prominent eosinophilic intranuclear/intracytoplasmic inclusion bodies are typical of canine distemper viral infection (CDV) and such inclusions can often be noted in gallbladder and ductular epithelia. The prominent signs of respiratory embarrassment in this case were due to a severe interstitial pneumonia that is characteristic of CDV infection. This paramyxovirus is well-known for its immunosuppressive effects and its association with cryptosporidiosis has been reported in the canine. The association of CDV and cryptosporidiosis in this case is uncertain as both diseases are noted to occur separately in this species.

AFIP DIAGNOSIS Gallbladder: Cholecystitis, subacute, eosinophilic, diffuse, mild, raccoon, procyonid.

Gallbladder, luminal mucosal surface: Protozoa--etiology consistent with Cryptosporidium sp.

Gallbladder, mucosal epithelium: Eosinophilic intranuclear and intracytoplasmic inclusion bodies.

CONFERENCE NOTE Three separate morphologic diagnoses were made because the relationship of the inflammatory changes to the infectious agents was not clear. In the experience of some attendees, eosinophils are commonly present in inflammatory lesions of various causes in raccoons. Giemsa is useful for demonstrating small numbers of Cryptosporidium organisms; modified acid-fast procedures differentiate red staining oocysts from similarly sized and shaped green staining yeasts in smears and floatation procedures. Canine distemper is a relatively common fatal disease in raccoons.

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MICROSLIDE 55

HISTORY This 5-month-old male Beagle presented with vomiting, anorexia, and fever (105F). The animal was treated for 7 days with ampicillin and gentamicin. The dog responded to treatment, but was readmitted 3 days later for anorexia. The animal was re-started on ampicillin, gentamicin and fluids. No response was seen and the animal died 7 days after readmittance to the hospital.

GROSS PATHOLOGY At gross necropsy, the kidneys were pale and the lungs were moderately congested.

LABORATORY RESULTS

BUN	244 mg/dl	TP	=	3.7 gm/dl
Creatinine	13.3 mg/dl	Alb	=	2.1 gm/dl
P	15.4 mg/dl	K+	=	7.6 mM/1
Ca	8.5 mg/dl	C1	=	90.0 mM/1
		Na+	=	139 mM/1

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Necrosis, acute, diffuse, severe with mineralization, proximal tubules, kidney, dog.

These renal lesions reflect typical manifestations of gentamicin nephrotoxicity. Several similar cases of gentamicin nephrotoxicity occurred during a recent outbreak of parvovirus enteritis in a closed beagle colony. Multiple clinicians were treating dogs, and initial signs of renal failure were mistaken for continued clinical signs of parvoviral enteritis. Hence, gentamicin therapy was continued in some animals for more than 10 days. Although the intestine showed evidence of a moderate subacute enteritis, consistent for a resolving parvoviral enteritis, the cause of death in these animals was nephrotoxicity from prolonged gentamicin toxicity.

AFIP DIAGNOSIS Kidney: Tubular necrosis, acute, diffuse, severe, with regeneration and mineralization, beagle, canine.

CONFERENCE NOTE As with most nephrotoxins, gentamicin primarily affects proximal convoluted tubular epithelium. Lesions progress from hyaline droplet degeneration with loss of

brush border and dilated lumina to necrosis. Ultrastructurally, cytosomes containing myelin figures and clumps of flocculent material are seen prior to necrosis. The intralysosomal myelin figures are probably remnants of membranes and organelles; the myelin figures are believed to form at an increased rate because of inhibition of protein synthesis by gentamicin.

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MICROSLIDE 56, 35 MM SLIDE 13

HISTORY This 2-year-old steer was affected as part of an outbreak of a neurologic disease in cattle in Southern Brazil. Clinical signs were transient seizures which affected animals of all ages older than 1 month and all breeds. The seizures were manifested by a rigid neck, pendular movements with the head, muscular tremors and falling over on one side or backwards. While on the ground the animals had opisthotonos.

The seizures were triggered when the animals were disturbed or frightened. The animals which did not fall showed ataxia, a wide base stance and dysmetric (hypermetric) gait. The duration of the seizures were from a few seconds to 1-2 minutes after which the animals, if left undisturbed, returned to apparent normality. The morbidity was 2% of the herd and the mortality was very low. Fatalities were usually due to accidents during the seizures.

In the pasture where the disease occurred abundant growth of the plant Solanum fastigiatum var. fastigiatum was observed. The animal of this report was killed for necropsy after showing the clinical signs described above.

GROSS PATHOLOGY No specific gross lesions were observed. The animal of this report had subcutaneous ecchymoses and a fractured jaw due to trauma inflicted during the seizures.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Cerebellar cortex, primary neuronal degeneration, bovine. Etiology: poisoning by Solanum fastigiatum var. fastigiatum.

The spontaneous poisoning by S. fastigiatum var. fastigiatum is a neurological disease of cattle which has been described in Southern Brazil. The disease was experimentally reproduced by feeding this poisonous plant to cattle. Similar diseases in cattle with similar histopathological changes and caused by the ingestion of related plants (S. dimidiatum and S. kwebense) have been described respectively in the United States and South Africa. These plants induce a primary neuronal degeneration, mainly of the Purkinje cells (PC) of the cerebellar cortex. In the Brazilian cases EM studies of the lesions suggest an induced lysosomal storage disease, probably a lipidosis. The sections in the present case reveal the typical changes of the disease. There is depopulation of PCS, many of the remaining PCS have fine vacuolation of perikaryon; occasionally large vacuoles are observed. These vacuoles were negative with Sudan staining of frozen sections and on PAS staining. Secondary axonal swelling (spheroids) are seen in the cerebellar white matter. In toluidine stained sections and EM preparations lipid inclusions similar to those described in the inherited and induced lipidoses of animals and man are observed.

AFIP DIAGNOSIS Cerebellum, Purkinje cells: Degeneration and loss, diffuse, moderate, breed unspecified, bovine.

CONFERENCE NOTE Discussion centered on the pathogenesis; it is thought the accumulation of abnormal lipid results in the primary histopathological changes.

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MICROSLIDE 57

HISTORY This previously healthy 7-year-old male Labrador retriever had a 3 month course of illness involving lethargy, weakness, partial anorexia, polyuria, polydipsia and lingual ulcerations. The dog had a fixed urine specific gravity, extremely elevated BUN, extremely elevated creatinine, extremely elevated serum phosphates and mild anemia. Another dog in the household was similarly affected; persons in the household were normal. Both dogs had direct access to waters in the Chesapeake Bay which was about 1/2Km from 3 large marinas. The Labrador was euthanized in an attempt to make an etiologic diagnosis and possibly save the other dog.

GROSS PATHOLOGY Uremic ulceration, severe, tongue.

Parathyroid hyperplasia, bilateral, severe, parathyroid glands.

Gastric ulcers, hemorrhagic, multifocal, mild, stomach.

Nephrosis, chronic, diffuse, severe, bilateral, kidneys.

Enteropathy, cestodal, disseminated, mild, small intestines. Etiology: Dipylidium sp.

LABORATORY RESULTS

BUN:	247	mg/dl
Creatinine:	8	mg/dl
Phosphates:	28.9	mg/dl
Calcium:	8.7	mg/dl

PVC: 26%

Bacterial culture results: No pathogenic bacteria were cultured from the kidneys, liver, lung, spleen, urine, urinary bladder, prostate and gastrointestinal tract.

Serologic titers to 5 serotypes of Leptospira were less than 1:100.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Glomerulonephrosis, oxalate, chronic, diffuse, severe, kidneys.

Edema, diffuse, moderate, kidney. Etiologic diagnosis: Chronic oxalate nephrosis. Etiology: Repeated sublethal ingestion of ethylene glycol.

About 48 hours after this Labrador was euthanized, its cagemate also died. Both dogs had similar renal, lingual, gastric and parathyroid lesions. The etiologic diagnosis was established by the histologic examination of ethanol-fixed, unstained sections of the kidneys. Massive amounts of intensely birefringent crystals, consistent with oxalates, were present in the kidneys of both dogs. Ethylene glycol was the presumed toxicant. The source of the ethylene glycol was the Chesapeake Bay. During the spring, large numbers of pleasure boats are removed from dry-docks and launched in the 3 neighboring marinas. The ethylene glycol which is present in the pipes of the boats is generally flushed into the marina. One marina owner estimated that over 10,000 gallons of ethylene glycol had been flushed from boats each spring in the past 10 to 15 years. Each spring the waters of the Bay around marinas have a distinct greenish sheen; the owner of the dogs reported seeing such greenish waters along his property, and that the dogs avidly drank this water. The onset of symptoms in both dogs was April 1989, corresponding to the launching of pleasure craft. The dogs survived for about 3 months, and one only by receiving intensive therapy, including peritoneal dialysis.

Oxalate crystals are very rare in the formalin-fixed, H&E stained sections. The principal renal lesions are those of intense interstitial edema and fibrosis, dilation and atrophy of tubules, atrophy, degeneration and necrosis of tubular epithelium and various glomerular alterations. In the unstained alcohol-fixed sections, nearly every cortical tubule contains oxalate crystals within the atrophied epithelial cells; only a very few clusters of crystal were present free in the tubular lumina. This distribution of crystals is consistent with chronic recovering stages rather than acute ingestion of ethylene glycol.

AFIP DIAGNOSIS Kidney: Glomerulonephritis, mesangioproliferative, diffuse, moderate, with crescents, interstitial nephritis, nephrocalcinosis, and oxalate deposition, Labrador retriever, canine.

CONFERENCE NOTE Most conference participants diagnosed a mesangioproliferative or membranoproliferative glomerulonephritis as the primary lesion in this case; the tubulointerstitial changes, nephrocalcinosis and oxalate crystal deposition were considered secondary changes. Oxalate crystal deposition is not uncommonly observed in cases of nephritis with tubular obstruction in the dog; deposition is less than expected in cases of acute ethylene glycol toxicity.

We agree with the contributor/moderator that the tubulointerstitial changes, taken by themselves, are compatible with the chronic, recovering phase of ethylene glycol toxicity; however, the glomerulonephritis is the predominant lesion and indicates an alternate pathogenesis. The staff of the Division of Nephropathology concurred that the principle lesion is a glomerulonephritis, and in a human, would be suggestive of lupus or hypersensitivity.

Oxalate crystals are not water soluble and should not require alcohol fixation for preservation. In our experience in cases of acute ethylene glycol toxicity, tubules contain large numbers of oxylate crystals, while in more chronic cases the crystals are fewer.

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MICROSLIDE 58, 35 MM SLIDE 14

HISTORY This adult female great blue heron (Ardea herodias) was found injured and was taken to a wildlife rehabilitator for care. The bird had a compound fracture of the left proximal metatarsus. The bird was treated with fluids and the leg was bandaged, albeit inadequately. The bird died within 24 hours of admission. At necropsy, numerous lesions were found, but cause of death was attributed to gangrene of the limb distal to the fracture.

GROSS PATHOLOGY Compound fracture of left metatarsus with gangrene, severe.

Verminous fibrosing peritonitis and air sacculitis, focal, severe, abdominal cavity; etiology consistent with Eustrongylides sp. (probably E. ignotus).

Verminous stomatitis, multifocal, mild to moderate, mouth. Etiology: flukes.

Atrophy and degeneration of muscles and fat, generalized, moderate to severe. Etiology: suggestive of starvation/inanition.

LABORATORY RESULTS Parasite Examination of Feces & Blood: Fluke and ascarid eggs were present in the feces in mild numbers.

Leukocytozoan-like protozoa were present in white blood cells.

Dioctophymata-like eggs, mild, feces.

Bacteria Culture Results: Edwardsiella tarda was cultured from all internal organs.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Chronic-active, verminous, peritonitis, diffuse, severe, esophageal-proventriculus junction. Verminous peritonitis (peritoneal eustrongylidiasis). Eustrongylides sp. (probably E. ignotus).

The immediate cause of death in this heron was attributed to trauma (compound fracture of the left leg with gangrene). The bird was found in very poor condition (emaciated). It is not clear whether the poor body condition was due solely or partially to the chronic verminous peritonitis, but the bird was clearly unable to feed as a result of the limb fracture.

Eustrongylidiasis is common in herons and egrets in the mid-Atlantic region (Locke, 1960). At necropsy the proventriculus, gizzard and most of the upper intestines formed a solid fibrous mass in which numerous large (4-7 mm diameter) bright red nematodes were present. Despite several hours of dissection of the fresh and formalin-fixed mass, no intact worms could be removed from their dense fibrous "tubes". Incidental necropsy lesions included numerous flukes attached to the oral, esophageal and proventricular mucosas and generalized emaciation.

Histologically, the nematodes are embedded in dense fibrous inflammatory tissue; a few dead worms with suppuration are present. Key features in identifying the nematodes are size, prominent thick cuticle, prominent hypodermis, uniformly tall coelomyarian muscles, large ventral cord, numerous small chords, pseudocoelomic membranes, prominent intestine with tall columnar cells lined by long microvilli and the presence of pigment in the intestinal cells. The eggs are dioctophyma-like, having thick knobby shells and are non-larvated. In some sections, worms are present transmurally in the esophagus or proventriculus.

A few sections also contain unidentified nematodes within the esophageal lumen; some slides also have sections of flukes in the lumen of the esophagus.

The life cycle of this parasite has not been fully studied. Larvae probably infect microcrustaceans initially, then various fresh-water fish, such as minnows (Fundulus spp) or killifish. It is not clear whether fish are intermediate hosts or paratenic hosts. Some species of Eustrongylides require an aquatic oligochaete as an intermediate host. The definitive host of the adult worms are usually piscivorous birds, especially herons, egrets and ducks. Mammals are rarely infected, but larval Eustrongylides have been reported from muskrats, otter and man. In one study, as many as 48% of minnows were found infected with fourth-stage larvae of Eustrongylides. Human infections have occurred as the result of fishermen swallowing uncooked minnows. Two fishermen developed intense abdominal pain 24-48 hours after swallowing minnows; both men had laparotomies performed and the worms were removed surgically. In a group of 25 garter snakes which were fed banded killifish caught in the Chesapeake Bay, 20 snakes died or developed subcutaneous nodules; larval Eustrongylides were surgically removed from these nodules. Hence, this parasite has the potential for infecting a wide variety of species from at least 4 orders of vertebrates. Clearly, this parasite has significant public health concerns.

AFIP DIAGNOSIS Proventriculus: Granulomas, multiple, transmural, with peritoneal fibrosis and intralesional nematodes and eggs, great blue heron, (Ardea herodias) avian. Etiology: Eustrongylides sp.

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REFERENCES

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MICROSLIDE 59

HISTORY This juvenile male great horned owl (Bubo virginianus) was hit by a car and treated for trauma. Necrotic plaques were observed in the oral cavity. The bird did not respond to therapy and died the next day.

GROSS PATHOLOGY Traumatic lesions were present on the head. Necrotic plaques were found on the oral mucosa especially the laryngeal prominences and palatal folds. The liver was swollen and multiple, white-tan, 1-2 mm diameter foci were on the capsular and cut surfaces. Similar foci were present in the spleen and bone marrow.

LABORATORY RESULTS Cultures were negative for pathogenic bacteria. Herpesviruses were detected in spleen and liver by electron microscopy.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Liver: Hepatitis, necrotizing, acute, multifocal to coalescing, severe, with intranuclear inclusion bodies in hepatocytes, great-horned owl, avian. Etiology: Owl herpesvirus.

The herpesvirus causing fatal disease in owls and falcons is widely distributed in North America and Europe and has been recognized as a cause of mortality of great horned owls since at least 1932. The virus is serologically related to pigeon herpesvirus and pigeons may serve as the source of the virus for owls and falcons. The impact of this disease on populations of owls and falcons is not known, but the disease appears to be sporadic.

The lesions in this case are typical of herpesvirus infections in other birds including pigeons and psittacines (Pacheco's disease). Clinical course of disease is short and lesions are acute with little opportunity for inflammatory cell response. Inclusion bodies are usually prominent.

AFIP DIAGNOSIS Liver: Hepatitis, necrotizing, acute, multifocal to coalescing, severe, with hepatocellular eosinophilic intranuclear inclusion bodies, great horned owl, (Bobo virginianus) avian.

CONFERENCE NOTE The following viruses are known to cause hepatocellular intranuclear inclusion bodies in birds: adenovirus in chickens, goslings and bobwhite quail; herpesvirus in parrots, pigeons, owls, hawks and ducks; parvovirus in geese; papovavirus in budgerigars. Additionally, adenoviral hepatitis has been reported in a merlin.

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REFERENCES

Graham, D. L., Mare, D. J., Ward, F. P., and Peckham, M. C.: Inclusion body disease (herpesvirus infection) of falcons (IBDF). Journal of Wildlife Diseases, 11: 83-91. 1975.

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Wednesday Slide Conference No. 29, Case II. May 18, 1988. Contributor: Tufts University School of Veterinary Medicine, Department of Pathology.

MICROSLIDE 60

HISTORY This tissue is from one of three 6-year-old common marmosets (Callithrix jacchus) that were inoculated with frozen liver tissue taken from an emperor tamarin and 7 golden lion tamarins (Leontopithicus iosalia).

GROSS PATHOLOGY There was hepatomegaly, serous cavity effusion, occasional serosal hemorrhages and jaundice.

LABORATORY RESULTS AST greater than 2000/UL (60-200 U/L, normal range).

SAP 1760 U/L (4-76 U/L, normal range).

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Acute hepatitis, diffuse, with acidophilic body formation. Etiology: Viral, enveloped RNA virus as of yet unclassified.

AFIP DIAGNOSIS Liver: Hepatitis, necrotizing, subacute, multifocal, mild to moderate, with vacuolar change and acidophilic bodies, common marmoset, (Callithrix jacchus) non-human primate.

CONFERENCE NOTE In 1981 and 1982 acute fatal hepatitis was reported in tamarins and marmosets at two zoos. Ten additional outbreaks have been reported at nine other U.S. zoos and animal parks since 1982. Experimentally, infected livers exhibited hepatocellular swelling and necrosis with inflammation and acidophilic bodies. Enveloped virus particles, 85-105 nm, were present within vesicles of the rough endoplasmic reticulum and Golgi apparatus. The particles were negative for human hepatitis A, B, D, and non-A, non-B antigens as well as for GB agent. The source of this disease and its pathogenesis is unknown.

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MICROSLIDE 61

HISTORY This 5-month-old male sheepdog was bitten by a viper (Vipera aspis). Local swelling of the limb developed quickly but disappeared after 3 days. Vomiting and apathy continued, however, and the dog was destroyed 6 days after the incident.

GROSS PATHOLOGY There were hemorrhages in the lung, thymus, kidneys.

LABORATORY RESULTS BUN 860 mg%.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Cystic dilatation of glomerular tufts caused by mesangiolysis, caused by snake venom (V. aspis).

A second case of snake-bite in a dog we necropsied showed similar lesions (after 1 day).

Vipera aspis is found mainly in the alpine region of Switzerland. More widespread in Switzerland and northern Europe is Vipera berus (Kreuzotter). Identical lesions were found in Japan caused by another viper, the Habu snake.

AFIP DIAGNOSIS Kidney: Glomerular necrosis and thrombosis, segmental to global, generalized, severe, with mesangiolysis, mild tubular degeneration and necrosis, mineralization, and hyaline and hemorrhagic casts, sheepdog, canine.

CONFERENCE NOTE The renal glomerular changes observed in this case are typical of that seen in animals and man following bites by viperine snakes. The hemorrhagic cysts within the glomerular tufts have been studied in detail and are termed "mesangiolysis". In addition to its association with snake bite, mesangiolysis has been recognized in types of glomerulonephritis, diabetes mellitus, ischemia, and due to the toxic effects of miscellaneous chemicals. Cystic lesions in the glomerular tufts are due to sublethal endothelial damage that is followed by segmental ballooning of capillary loops, dissolution of the endothelium, dissolution of the mesangial matrix and the eventual formation of hemorrhagic cysts. The exact mechanism by which the initial injury leads to the characteristic cystic lesions is unclear, but may involve the release of granules from platelets. If the animal survives, there is segmental proliferation of mesangial cells, possibly induced by mitogenic agents known to exist in the products of platelet degranulation.

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REFERENCE

Morita, T., et al.: Mesangiolysis: sequential ultrastructural study of Habu venom-induced glomerular lesions. Laboratory Investigation, 38: 94-102. 1978.

MICROSLIDE 62

HISTORY This 4-month-old mixed-breed male goat developed progressive hind limb paresis starting at 1 month of age. Patellar reflexes were normal to hyperreflexic. Front limb reflexes appeared normal. He could not turn corners without stumbling.

LABORATORY RESULTS Serum copper was 0.27 ppm (normal 0.7-1.7 ppm).

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Vacuolative myelopathy, ventral and lateral funiculi spinal cord, due to copper deficiency.

Spinal cord: Numerous vacuolated spaces are present in the ventral funiculi with lesser number also located in the lateral funiculi. Some vacuolated spaces contain identifiable axons while others contain axonal or cellular debris. Scattered macrophages (gitter cells) are present in the neuropil and in some vacuoles.

The medulla had a localized area of swollen myelin sheaths with rare intrasheath macrophages in the fasciculus gracilis. Sections of sciatic nerve had multifocal myelin digestion chambers with segmental swollen myelin sheaths and swollen axons. Copper levels in the corn fed to the goats was 3 ppm which is below the total dietary requirement for small ruminants. Copper deficiency in ruminants may be influenced by the low availability of copper in the diet. Inhibitory effects are exerted on absorption of copper by small increases in herbage molybdenum and sulphur. High iron containing soils, if ingested, are antagonistic to copper absorption. There is also wide genetic variation in copper absorption between different breeds of small ruminants. In these cases, levels of molybdenum, sulphur and iron in the diet were not determined. Cell damage in copper deficiency is presumably caused by diminished activity of copper containing enzymes of the cytochrome oxidase system.

AFIP DIAGNOSIS Spinal cord: Axonal degeneration, multifocal, moderate, with swollen myelin sheaths, mixed-breed, caprine.

CONFERENCE NOTE Conference participants discussed the various clinical and histopathologic findings that are observed in copper deficiency. The two syndromes observed in young sheep and goats (swayback and enzootic ataxia) were discussed.

Copper is a cofactor in a variety of oxidative enzymes of diverse function. Deficiencies of the element may effect electron transport (cytochrome oxidase), absorption of iron and its utilization in hematopoiesis (ceruloplasmin), tyrosine degradation and pigmentation (tyrosinase), neurotransmitter metabolism (dopamine hydroxylase) or cross linkage of elastin and tropocollagen (lysyl oxidase).

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REFERENCES

Cordy, D. R., and Knight, H. D.: California goats with a disease resembling enzootic ataxia or swayback. Veterinary Pathology, 15: 179-185. 1978.

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MICROSLIDE 63

HISTORY A 2-year-old female Molluccan cockatoo was presented for necropsy with a history of severe ascites, acute dyspnea and death. The ascitic fluid was yellow, and analysis revealed a total protein of 36 g/dl and low cellularity (approximately 50% mesothelial cells and 50% heterophils). Additionally, radiographs revealed a possible mass in the left cranial air sac.

GROSS PATHOLOGY The carcass was in fair condition. There was feather loss over the dorsal neck, back and over the lateral surface of the legs. The abdomen was slightly distended and contained approximately 25-30 ml of yellow fluid. The heart was dilated and the pericardial sac was distended with approximately 20 ml of clear fluid. The liver was small, diffusely pale, had a thickened capsule and a prominent lobular pattern. The lungs were congested and edematous. The kidneys were swollen and congested. Multiple tissues were collected for histopathologic evaluation.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Marked diffuse subacute necrotizing arteritis and periarteritis, with mild multifocal necrotizing myocarditis, Cockatoo. Etiologic diagnosis: Papovaviral vasculitis and myocarditis.

The principal alterations in the cardiac muscle are marked diffuse subacute necrotizing arteritis and periarteritis with mild multifocal acute necrotizing myocarditis. The arteritis is characterized by necrosis of the tunica media and perivascular mononuclear cell infiltration involving the majority of arterioles. There is marked hydropic degeneration with Cowdry type B pale basophilic intranuclear inclusion bodies within hyalinized smooth muscle cells. The perivascular spaces are distended and infiltrated by low numbers of inflammatory cells, primarily lymphocytes. The myocarditis is characterized by occasional foci of myocyte swelling, hyalinization, sarcoplasmic coagulation, karyorrhexis and karyolysis.

Ultrastructural examination revealed that the intranuclear inclusions consist of 45 to 55 nm diameter virus particles with typical papovaviral morphology.

AFIP DIAGNOSIS Heart, arteries: Arteritis, necrotizing, diffuse, severe, with multifocal necrotizing myocarditis, cockatoo, avian.

CONFERENCE NOTE Papovavirus infection has been described in a wide variety of psittacine species (budgerigars, splendid parakeets, conures, macaws, cockatiels and cockatoos) and in finches. A papovavirus (genus Polyoma) is considered to be the etiologic agent of budgerigar fledgling disease.

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REFERENCES

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MICROSLIDE 64

HISTORY This Chester white pig died after onset of a disease characterized by fever, dyspnea and lethargy.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Pneumonia, fibrinous, subacute, multilobular, moderate, lung.

Meningoencephalitis, fibrinous, subacute, diffuse, severe, brain.

Epicarditis, fibrinous, subacute, diffuse, severe, heart.

Microscopic lesions are consistent with a diagnosis of Glasser's disease (Haemophilus parasuis).

AFIP DIAGNOSIS Lung: Bronchopneumonia, suppurative, focally extensive, moderate, with fibrinosuppurative pleuritis, Chester white, porcine.

Brain: Meningitis, suppurative, diffuse, severe.

Pericardium: Pericarditis, chronic, diffuse, moderate.

CONFERENCE NOTE Although the lesions present in this case are a good example of the disease, culture results were not available. Infection with Streptococcus suis produces similar lesions and was considered the primary diagnosis by many participants. Mycoplasma sp. infection would be another plausible etiology for polyserositis; however, it produces different morphologic tissue alterations.

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REFERENCE

Nicolet, J.: Hemophilus infections. In: Diseases of Swine, Sixth Edition, pages 426-436. 1986. Edited by Leman, A. D., et al. Iowa State University Press, Ames, Iowa.

MICROSLIDE 65

HISTORY These tissues are from an 8-week-old broad breasted white turkey hen. A commercial flock of 4,000 turkeys were ranged in a fenced field. Sudden death of 4-5 birds per day commenced abruptly and continued for approximately 10 days, when death loss ceased. No clinically ill birds were reportedly observed; however, dead birds were observed to have blood oozing from the vent.

GROSS PATHOLOGY The small intestines were distended, dark in color and filled with blood; the mucosa was congested. The spleens were enlarged, congested, friable and in some cases mottled.

LABORATORY RESULTS Random blood samples taken from the birds following the death loss episode were positive in AGID tests against the turkey hemorrhagic enteritis agent (Avian adenovirus type II).

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Spleen: Hyperplasia of the RE cells, occasional germinal center necrosis, numerous intranuclear inclusions in the RE cells; splenitis (Avian adenovirus type II).

Intestine: Degeneration and sloughing of the villous epithelial cells and severe mucosal congestion, hemorrhage into the lumen. Occasional intranuclear inclusions in RE cells in the basal lamina propria; hemorrhagic enteritis (Avian Adenovirus type II).

Hemorrhagic enteritis is characterized by a rapid onset and signs of the disease may include depression and bloody droppings as well as birds that are found dead. The disease episode usually subsides within 7-10 days. The pathological changes are most evident in the reticuloendothelial system and intestine. The virus seems to be produced in the RE cells that form virus containing intranuclear inclusions. Intestinal bleeding from damaged capillaries of the villi is the probable cause of death.

AFIP DIAGNOSIS Spleen: Hyperplasia, reticuloendothelial, diffuse, severe, with basophilic intranuclear inclusions, turkey, avian.

Small intestine: Congestion, diffuse, severe.

CONFERENCE NOTE Of the economically important gastrointestinal diseases of turkeys, Avian adenovirus type II infection shares an important level of consideration with that of transmissible enteritis, which is caused by a coronavirus. Hemorrhagic enteritis is characterized by, of course, bloody diarrhea, whereas the diarrhea seen in transmissible enteritis is frothy.

The splenic changes are similar to those of marble spleen disease in pheasants.

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REFERENCES

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MICROSLIDE 66

HISTORY This 2-week-old Charolais calf had diarrhea since it was 3-4 days old. This calf was from a 160 head herd with a neonatal diarrhea problem of 25% morbidity and 75% mortality despite extensive treatment.

GROSS PATHOLOGY The practitioner reported depletion of body fat stores and dehydration. The intestines were hyperemic and flaccid with watery to pasty (colon) contents.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Colon: subacute, necrotizing colitis with regenerative hyperplasia of crypt epithelium. Etiology: Enteric Coronavirus.

The direct F.A. on cryostat sections and avidin-biotin-immunoperoxidase studies on paraffin block sections with bovine enteric Coronavirus monoclonal antibodies confirmed the etiology.

This is an extremely common cause of diarrhea in beef calves in Western Canada. In our experience, the spiral colon is the tissue of choice for histopathologic, immunoperoxidase and virologic studies, since even in moderately autolyzed carcasses, the etiology can still be confirmed. The histopathologic changes are typical but by no means pathognomonic for coronavirus infection. Generalized lymphoid tissue atrophy (including GALT) and microscopic foci of abomasal mucosal necrosis are also typical of this disease. Unless the calf is submitted alive or fresh there is little value in histopathologic evaluation of the small intestine, except to rule out other causes of diarrhea. Gross evaluation of the intestinal tract alone is very misleading.

AFIP DIAGNOSIS Colon: Colitis, necrotizing, subacute, diffuse, mild to moderate, with crypt epithelial hyperplasia, Charolais, bovine.

CONFERENCE NOTE The pathogenesis of enteric viral diseases was discussed, and focused on altered enterocyte function, namely reduced absorption, as one of the main causes of clinical diarrhea. The resulting sodium loss and acidosis causes a release of intracellular potassium, which many times is a primary factor in death.

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REFERENCES

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MICROSLIDES 67 and 68

HISTORY This 5-month-old Landrace-Yorkshire cross pig was from a minimal disease herd. A marked increase in coughing and sneezing was noted in the herd following a period of unusually cold weather during which both the ventilating fan system and the automated feed system froze. On that occasion, the feed system was restarted first and ran for several hours before the ventilating system was back in operation. This caused the atmosphere in the barn to become very dusty. The feed in use was a pelleted ration that had been coated with starch powder in order to facilitate the flow of feed from the delivery trucks into the storage tanks. Coughing gradually subsided but sneezing persisted until slaughter, two and half weeks later.

GROSS PATHOLOGY The referring veterinarian indicated that lungs from five of 34 animals examined at slaughter had grossly visible lesions confined to the cranioventral regions. The only tissues available to the pathologist for gross and microscopic examination were small samples of lung. These showed multifocal areas of consolidation and atelectasis.

LABORATORY RESULTS Bacteriological culture of the lungs yielded no significant isolate.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Pneumonia, granulomatous, multifocal, secondary to aspiration of dust.

Pulmonary lesions had a lobular distribution. They included an infiltration of a large number of macrophages and multinucleated giant cells within alveoli and bronchiolar lumens, infiltration of mononuclear leukocytes within alveolar walls, and moderate interstitial fibrosis. Many macrophages and giant cells were closely associated with, or had phagocytized, particles of plant material and starch-like granules that were birefringent under polarized light and positive

on periodic acid-Schiff stain.

This outbreak of respiratory disease associated with a dusty environment was a relatively mild one which reflected the brief duration of the problem in the barn. More severe outbreaks including mortality may occur in association with a proportionally more severe and persistent dust problem.

AFIP DIAGNOSIS Lung: Bronchopneumonia, granulomatous, subacute, multifocal, moderate, with emphysema and intralesional granular birefringent particles, Landrace-Yorkshire cross, porcine.

CONFERENCE NOTE The development of pneumoconiosis is dependent on the amount of dust, size and shape of the particles, particle solubility, and presence of other irritants.

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REFERENCE

Jericho, K. W. F., and Harries, N.: Dusty feed and acute respiratory disease in pigs. Canadian Veterinary Journal, 16: 360-366. 1975.

MICROSLIDE 69

HISTORY This 2-year-old rhesus monkey was a control animal in a study to evaluate the efficacy of Shigella flexneri live oral vaccine. This animal received a placebo prior to being challenged with oral S. flexneri. This animal developed diarrhea and died within 24 hours of challenge in spite of antibiotics and supportive therapy.

GROSS PATHOLOGY The colon was diffusely edematous, but the mucosa was intact with scattered fibrinohemorrhagic tags on its surface. The ileum and distal jejunum were diffusely edematous and lined by a diphtheritic membrane.

LABORATORY RESULTS Pure cultures of Shigella flexneri were isolated from the ileum.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Acute, severe, diffuse, fibrinopurulent enteritis, with loss of villous epithelium. Etiology: Shigella flexneri.

Other lesions seen include focally extensive edema of the submucosa, crypt abscesses, epithelial erosions and the appearance of bacterial colonies in many sections. Shigellosis is seen only in primates, including man, and is generally thought to affect the colon. However, lesions are occasionally seen in the ileum, as in this case, and stomach. Early lesions in the colon include edema, congestion, and hemorrhage accompanied by a purulent exudate. A diphtheritic membrane is often seen grossly. The organisms invade the mucosa and can spread cell to cell but rarely invade the submucosa or blood. Differentials, particularly for small intestinal involvement, include Campylobacter, Salmonella, Yersinia and E. coli.

AFIP DIAGNOSIS Small intestine: Enteritis, fibrinosuppurative, necrotizing, diffuse, moderate, with villus loss and crypt abscesses, rhesus monkey (Macaca mulatta), non-human primate.

CONFERENCE NOTE Many attendees felt the loss of villi, the collapsed mucosa, and the lack of goblet cells made tissue identification somewhat difficult. Discussion centered around the proposed pathogenesis of this disease, in which the small intestine is colonized first and the bacteria produce a shigatoxin which is believed to induce jejunal secretion. Colonic mucosal damage depends on the organism's ability to penetrate enterocytes.

Some conferees had difficulty reconciling the known pathogenesis with the apparent short incubation time (and death) in this case.

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REFERENCES

Carlton, W. W., and Hunt, R. D.: Bacterial Diseases: Shigellosis. In: Pathology of Laboratory Animals, Volume 2. 1978. Edited by: Benirschke, K., et al. Springer-Verlag, New York.

Enurah, L. U., et al.: Fatal shigellosis in a chimpanzee (Pan troglodyte). Journal of Wildlife Diseases, 24(1): 178-179. 1988.

MICROSLIDE 70

HISTORY This 2-year-old female Sprague-Dawley rat was found moribund and killed after 678 days on study.

GROSS PATHOLOGY A firm tan mass 2.0 x 1.5 x 1.5 cm was present in the liver. Also present were a 2 mm diameter cyst in the uterus and a 4 mm red lesion in the pituitary.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Cholangiocarcinoma (with intestinal cell metaplasia).

This tumor is a rare spontaneous lesion in rats. The occurrence of cells with the appearance of intestinal epithelium is not unexpected since intestinal cell metaplasia can also occur with hepatic adenofibrosis.

AFIP DIAGNOSIS Liver: Cholangiocarcinoma, Sprague-Dawley rat, rodent.

CONFERENCE NOTE Conference discussion centered on the primary differentials for this neoplasm: cholangiocarcinoma, cholangiofibroma, and metastatic carcinoma from the gastrointestinal tract. Metastatic gastrointestinal neoplasia, considered in the differential diagnosis because of the presence of goblet cells and paneth-like cells, was considered unlikely as no primary neoplasm was observed in the gastrointestinal tract. In addition, and as mentioned by the contributor, intestinal epithelial metaplasia (differentiation) has been described

in areas of cholangiofibrosis. The difficulty in distinguishing between cholangiocarcinoma and cholangiofibroma in the rat liver is well known. In this particular case, the primary criterion used for diagnosing cholangiocarcinoma (versus cholangiofibroma) was the piling-up of neoplastic cells. This case was reviewed by a group of pathologists at the National Institute of Environmental Health Sciences (NTP); the group's consensus diagnosis for this controversial lesion was cholangiocarcinoma.

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MICROSLIDE 71

HISTORY This 2-month-old female chimpanzee was raised in a nursery. A chronic nasal discharge began at one month of age and persisted despite treatment with Keflex and decongestants. She developed dyspnea and cyanosis with moist rales 18 hours before dying; the body temperature was only mildly elevated during this time.

GROSS PATHOLOGY Atelectasis of right cranial lung lobe. Pericardium greatly thickened by fibrin. Fibrin tags present in meninges of cerebrum.

LABORATORY RESULTS CSF, lung, pericardial fluid and blood all had positive cultures for Haemophilus influenzae.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Acute, severe, diffuse fibrinopurulent pericarditis.

Acute, mild, diffuse, purulent epicarditis. Etiology: Haemophilus influenzae.

Haemophilus influenzae is a serious infection of humans and is particularly severe in children. It can be seen clinically as meningitis, epiglottitis resulting in airway obstruction, pneumonia, septic arthritis, pericarditis and bacteremia.

Encapsulated H. influenzae type B is implicated in 95% of systemic infections in children. Non-human primates have been shown to be susceptible as a result of a study by Scheifle in which 17 out of 19 neonatal animals exposed intranasally developed bacteremia, septic arthritis, pericarditis, and meningitis. The latter appears to be the most frequent lesion seen with systemic infections. Other bacterial causes of pericarditis in neonatal chimpanzees include: Neisseria meningitidis, Streptococcus pneumoniae, Klebsiella pneumoniae, and Mycobacterium species.

AFIP DIAGNOSIS Heart: Pericarditis, fibrinosuppurative, diffuse, severe, chimpanzee, (Pan troglodytes), non-human primate.

Heart, myofibers: Degeneration, multifocal, mild, with swelling and vacuolation.

CONFERENCE NOTE Attendees discussed the various conditions associated with Hemophilus influenzae type B infection. Disease caused by H. influenzae was compared and contrasted with disease caused by H. parasuis (Glasser's disease). Both organisms cause a fibrinous inflammatory reaction in the meninges, pericardium, pleura and synovial membrane of many joints.

Many conference participants also identified degeneration and swelling of myocytes. The staff of the Department of Cardiovascular Pathology commented that this lesion resembled the changes observed in human myocardium following toxic insult (such as seen with adriamicin toxicity).

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REFERENCES

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MICROSLIDE 72

HISTORY This tissue is from an 8-week-old female BALB/c mouse.

GROSS PATHOLOGY No gross lesions detected.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Chronic, diffuse, lymphohistiocytic sialoadenitis with intranuclear and rare intracytoplasmic, eosinophilic inclusion bodies within enlarged acinar and occasional ductal epithelial cells (murine cytomegalovirus).

Ten 8 week old female BALB/c mice were injected I.P. with mouse salivary gland homogenate containing 8×10^5 $TCID_{50}$ or approximately 25 LD_{50} of murine cytomegalovirus (Cassazza strain). Nine of the 10 mice died within 3-8 days following the I.P. injection. All of these mice exhibited an acute systemic infection with hepatitis and splenitis. There were large eosinophilic intranuclear inclusion bodies in the hepatocytes and reticuloendothelial cells of the spleen. The lone survivor, clinically normal, was killed at 16 days post injection and alterations were found only in the salivary gland. Cytomegalovirus infection of the salivary glands of mice is usually latent with no evidence of clinical disease; only occasionally is there a mild inflammatory reaction in the gland. Generalized disease is essentially nonexistent in laboratory animals;

however, it may be experimentally induced if the virulence of the virus is enhanced by serial passage, if large doses of virus are injected, or if latently infected animals are immunosuppressed.

AFIP DIAGNOSIS Submaxillary salivary gland: Sialoadenitis, lymphocytic, diffuse, moderate, with acinar and ductal epithelial cytomegaly and intranuclear and intracytoplasmic inclusion bodies, BALB/c mouse, rodent.

CONFERENCE NOTE The differential diagnosis for sialoadenitis in mice also includes polyoma virus and reovirus type 3. Polyoma virus preferentially infects parotid salivary glands, while cytomegalovirus preferentially infects submaxillary glands. Polyoma virus produces intranuclear inclusions, but neither cytomegaly nor intracytoplasmic inclusions. Reovirus type 3 causes necrotizing sialoadenitis but does not produce inclusion bodies. Mouse thymic virus, another herpesvirus, infects salivary glands, but lesions have not been described. Mouse mammary tumor virus replicates in salivary glands but does not produce lesions. Mice are susceptible to experimental infection with sialodacryoadenitis virus.

Sections of lymph node with mild follicular lymphoid hyperplasia were present on some of the glass slides.

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REFERENCES

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MICROSLIDE 73

HISTORY An adult New Zealand white rabbit was inoculated intranasally with frozen, filtered liver homogenate taken from a dead rabbit. The animal was febrile at 24 hours post-infection and moribund at 48 hours post-infection.

GROSS PATHOLOGY The liver was pale tan, slightly mottled, and friable. The spleen was engorged to twice normal size. The lungs were congested and edematous, with blood-tinged fluid in the airways.

LABORATORY RESULTS By electron microscopy, intranuclear dense granular to globular inclusion bodies with closely associated linear arrays of 25-30 nm diameter viral particles were found in degenerate hepatocytes.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Hepatitis, diffuse, necrotizing, severe, acute. Etiology: hepatitis virus of rabbits.

Necrotic hepatitis of rabbits is an emerging disease in many parts of Europe and the Far East. It is known by various names - viral hemorrhagic disease of rabbits, X disease of rabbits, hemorrhagic pneumonia in rabbits, and infectious hemorrhagic disease of rabbits. In December of 1988, it appeared in the Mexico City area and within four months, was responsible for the deaths of over 40,000 rabbits in ten different Mexican states.

The signs reported include an acute disease with high fever, respiratory distress, bloody nasal discharge, and death a few hours after the onset of clinical signs. Morbidity is 30-80%, with mortality reaching 80-90%.

The etiologic agent is small (25-30 nm diameter) and morphologically consistent with a parvovirus, although some workers feel it is more like a picornavirus. Attempts to isolate the virus have so far been unsuccessful. No commercial vaccine is yet available for this new disease. Autologous killed vaccines have been shown to be effective under experimental conditions in the Peoples' Republic of China, but were not effective in the face of an outbreak. The most effective control measure is prevention. Once introduced, the only means to control the disease is depopulation and disinfection.

Immunostaining of infected tissues has shown viral antigen in the nuclei and cytoplasm of hepatocytes, intestinal epithelium, and cells in the splenic marginal zone. Cytoplasmic staining is also present in many cells of the mononuclear phagocytic system. Intravascular fibrin thrombi are common microscopic findings. A severe terminal coagulopathy is probably responsible for the diversity of lesions reported in this disease.

AFIP DIAGNOSIS Liver: Hepatitis, necrotizing, acute, diffuse, moderate, New Zealand white rabbit, lagomorph.

CONFERENCE NOTE The differential diagnosis was discussed. Although not shown in this case, hemorrhagic pneumonia is frequently a prominent occurrence in this disease. Cases have been diagnosed in Mexico and therefore may soon be seen in this country.

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REFERENCES

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MICROSLIDE 74

HISTORY These laboratory raised, adult, male and female, sheepshead minnows (Cyprinodon variegatus) were presented to the L.S.U. Aquatic Pathobiology Laboratory because the population from which they were taken was not spawning properly. Many of the fish within the population had swellings in the ventral throat area.

GROSS PATHOLOGY White to whitish-pink, smooth, moderately firm, submucosal masses were present in the ventral and lateral pharyngeal areas of all 8 fish submitted. The masses originated from the base of the gill arches, protruded into the opercular cavity and forced the ventromedial aspects of the operc[illegible]ula laterally. On cut surface, the masses were pinkish-red. In fish sectioned longitudinally, the masses extended rostrally to the level of the second gill arch and caudally past the fourth gill arch to a point immediately cranial to the heart. All other tissues were grossly normal.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Thyroid hyperplasia (goiter), diffuse, severe.

In most fish, except for cartilaginous species (sharks, skates and rays), thyroid follicles are not located within a discrete gland as in mammals but are scattered in the tissue surrounding the ventral aorta subjacent to the floor of the pharynx. This anatomic variation accounts for the distribution of observed hyperplastic follicles in this case.

We have diagnosed thyroid hyperplasia in several populations of sheepshead minnows submitted by aquatic toxicology laboratories. These fish are from breeding stocks and have not been exposed to any known toxicants. In the laboratory, these minnows are raised in artificial seawater formulated with one of the commercially available salt preparations. These preparations contain adequate iodine for most species of saltwater fish, however, the iodide requirement of sheepshead minnows is not known.

The cause of the goiter in these fish has not been determined. Diets have been supplemented with iodine but the effect of dietary supplementation for prevention of goiter in these fish is not yet known.

Thyroid hyperplasia in fish, initially diagnosed as thyroid carcinoma, was first reported in the early 1900's in salmonids caught from the Great Lakes. In recent years, incidence of the disease in Great Lakes fish has increased dramatically, reaching epizootic proportions in some feral fish species, especially populations of coho salmon. Although most cases of thyroid hyperplasia are recorded from wild populations, hyperplasia is not uncommon in captive fishes held in aquaria.

Low dietary iodide is the usual cause of thyroid hyperplasia in birds and mammals and was originally considered the primary cause of hyperplasia in fish. Iodide deficiency plays a role in the disease but other factors are involved since attempts to induce goiter by feeding diets low in iodine are often unsuccessful. This lack of success may be because fish obtain most of their iodine across the gill epithelium from water. Evidence to date suggests some goiters in fish are caused by various waterborne, goitrogenic pollutants (PCBs, etc.). The exact nature of most of these pollutants and their mode of action is not well understood.

AFIP DIAGNOSIS Thyroid tissue: Follicular hyperplasia (goiter), diffuse, marked, sheepshead minnow (Cyprinodon variegatus), piscine.

CONFERENCE NOTE A majority of conference participants diagnosed thyroid follicular hyperplasia, with thyroid follicular carcinoma as the primary differential. The dispersed distribution of thyroid tissue in the fish was discussed at length.

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REFERENCES

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MICROSLIDE 75

HISTORY This tissue is from one of several cockatoos of both sexes and of various ages from juvenile to adult (breeding age).

All birds have feather loss on the body and legs with some new feathers emerging in the affected areas.

GROSS PATHOLOGY New emerging feathers are often stubby, clubbed-shaped or constricted.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Psittacine beak and feather disease.

The important histologic features of beak and feather disease are found within the new emerging feathers and include: (1) edema and necrosis of the pulp with mixed inflammation, (2) hyperplasia and necrosis of epithelial cells of the feather follicle, (3) macrophages in the pulp and epithelium containing basophilic cytoplasmic inclusions and (4) occasional amphophilic intranuclear inclusions in epithelial cells. The inclusions consist of viral particles, 17 to 22 nm in size, and are the probable cause of the disease.

Generally, the disease affects young psittacine birds, involves the beak and feathers, is progressive over several months, and causes low mortality. The down feathers are affected first and the wing feathers usually last.

AFIP DIAGNOSIS Feather: Pulpitis, subacute, diffuse, mild, with multifocal follicular and epithelial necrosis, and epithelial intranuclear and histiocytic intracytoplasmic inclusion bodies, cockatoo, avian.

CONFERENCE NOTE Recently, investigators at the University of Georgia, College of Veterinary Medicine have isolated a dimunivirus from infected birds (unpublished work). Assuming their isolate corresponds to the viral particles observed by others, it is a non-membrane bound virus 20 nm in diameter that forms intracytoplasmic pleomorphic arrays in the epidermis of the feather and beak, feather follicle epidermis, macrophages, cells of the thymus and bursa, and rarely in the liver (RE cells).

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REFERENCES

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MICROSLIDE 76, 35 MM SLIDE 15

HISTORY This 25-month-old, castrated male domestic shorthair cat, and his brother, but not his two sisters, had a progressive, painless myopathy characterized by diffuse skeletal muscle hypertrophy and increased muscle rigidity.

GROSS PATHOLOGY Hypertrophy of skeletal muscles.

Foci of necrosis of interventricular septal and left ventricular cardiac muscle.

LABORATORY RESULTS Creatinine kinase 16920 to 32850; normal, less than 75 U/L.

Aspartate transaminase 330 to 558; normal, 11-39 U/L.

Lactic dehydrogenase 367 to 819; normal, 63-273 U/L.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Chronic, multifocal, myopathy with necrosis, regeneration, hypertrophy and splitting of myocytes and mild interstitial fibrosis, characteristic of a muscular dystrophy. Some sections have necrotic and mineralized myocytes in addition to the other listed findings.

An immunoblot analysis for dystrophin in muscle from both affected brother cats revealed the absence of dystrophin. Dystrophin genomic DNA analysis did not disclose any detectable alteration of the dystrophin gene that is located on the X-chromosome.

AFIP DIAGNOSIS Skeletal muscle: Myopathy characterized by hypertrophy, necrosis, regeneration, myocyte splitting and fibrosis; diffuse, severe, domestic shorthair, feline.

CONFERENCE NOTE This is the first report of a dystrophin-deficient muscular dystrophy of felids. Dystrophin deficiency has been shown to be responsible for the inherited X-linked muscular dystrophy of MDX mice, golden retriever dogs and man (Duchenne/Becker muscular dystrophy).

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REFERENCES

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MICROSLIDE 77

HISTORY This 10-year-old female rhesus monkey (Macaca mulatta) was inoculated intramuscularly with simian immunodeficiency virus (SIV mac 251, uncloned virus).

GROSS PATHOLOGY The animal was cachectic with fecal staining around the anus. Multifocal to coalescing cutaneous erosions with tan, granular crusting were evident on several body surfaces. The hair coat was thin. The lungs were multifocally tan-red and mildly wet on cut section. Lymph nodes and thymic tissues were difficult to find. The stomach contained a small amount of mucus, the small intestine contained a large amount of tan, fluid material, and the colon contained abundant green, watery feces.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Moderate to severe, diffuse, giant cell pneumonia associated with SIV infection.

The major abnormality in this lung, discernible at low magnification, is the diffuse distribution of large numbers of multinucleate giant cells. These cells have abundant, granular to finely vacuolated, eosinophilic cytoplasm with up to 50 or 60 round to oval nuclei which tend to be clumped around the periphery of the cell and which contain granular to clumped chromatin and occasional indistinct nucleoli. These cells are generally found in alveoli and are sometimes in the company of alveolar macrophages and neutrophils. There is, in addition, a mild, usually peribronchiolar, predominantly lymphocytic inflammatory infiltrate.

While the low magnification appearance of this disease closely resembles that of the giant cell pneumonia occasionally seen in children with measles virus infection, upon closer inspection the multinucleate cells in the simian disease, unlike the Warthin-Finkeldey giant cells seen with measles, contain neither intranuclear nor intracytoplasmic inclusions. Another possible differential diagnosis in animals infected with SIV is tuberculosis, but acid fast stains are negative for mycobacteria. In addition to lung, these syncytial cells are occasionally seen in lymph node, spleen, kidney and gastrointestinal tract and may also be seen in the granulomatous encephalitis associated with SIV infection. This is in contradistinction to the case of humans infected with HIV where syncytial cells are seen only in the encephalitic lesions. Although the animal from which these sections were taken was experimentally inoculated with SIV, these cells are also seen in the naturally occurring disease although not as frequently.

AFIP DIAGNOSIS Lung: Pneumonia, interstitial, subacute, diffuse, mild, with intra-alveolar syncytial giant cells, rhesus monkey, primate.

Lung, alveoli: Eosinophilic flocculent material, multifocal, mild.

CONFERENCE NOTE With immunohistochemical procedures, SIV antigen was demonstrated within the multinucleated syncytial cells. GMS impregnation revealed numerous phagocytized and extracellular organisms, consistent with Pneumocystis carinii, admixed with small amounts of the typical eosinophilic flocculent material.

CONTRIBUTOR New England Regional Primate Research Center
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REFERENCE

Baskin, G.B., Murphey-Corb, M., Watson, E.A., and Martin, L.N.: Necropsy findings in rhesus monkeys experimentally infected with cultured simian immunodeficiency virus (SIV)/delta. Veterinary Pathology, 25:456-467. 1986.

MICROSLIDE 78, 35 MM SLIDE 16

HISTORY This 14 to 16-month-old Wistar-Firth rat was a control animal on a two year carcinogenicity study.

GROSS PATHOLOGY At necropsy, a tan, 0.5 cm mass was observed in the pelvis of one kidney.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Renal oncocytoma.

Renal oncocytomas are benign neoplasms that appear, as in this case, as small, solid or cystic, well differentiated cortical masses. They are usually not visible on gross inspection. They are composed of a monomorphic population of oncocytic cells, which have finely granular, intensely eosinophilic cytoplasm. The cytoplasmic appearance is caused by a dense population of atypical mitochondria. Mitotic figures are rarely observed.

Ultrastructurally, the cytoplasm, of these cells is filled with structurally abnormal mitochondria. Mitochondria often have a marked increase in the number of cristae or are pleomorphic in size and shape.

Renal oncocytomas occur in man, but the histogenesis of human renal oncocytomas is unclear. Some authors report that they arise in the proximal tubules, while others favor the distal tubule as a site of origin. Recent information suggests that in the rat, these tumors arise from oncocytic tubules which belong to the collecting duct system.

The submitted electron photomicrographs illustrate numerous mitochondria with an increased number of cristae.

AFIP DIAGNOSIS Kidney: Renal cell carcinoma, Wistar-Firth rat, rodent.

CONFERENCE NOTE The moderator and a majority of conference participants felt this neoplasm is more accurately diagnosed as a renal cell carcinoma because of the following features: (1) the neoplasm was relatively large (0.5 cm); the largest oncocytoma described in the literature we reviewed was 0.7 mm in diameter (in paraffin section), (2) the cytoplasmic granules in the neoplastic cells were not as intensely eosinophilic as observed with oncocytoma, (3) the mitotic rate in this neoplasm was high (1-2/HPF); mitoses are rare in oncocytoma and (4) the nuclei of neoplastic cells varied considerably in size, more so than would be expected with oncocytoma. Although the electron photomicrograph provided by the contributor demonstrates

cells containing large numbers of mitochondria, the mitochondria (at least at the magnification provided) do not exhibit the atypical morphologic features described with oncocytoma.

The staff of the Department of Renal Pathology reviewed this case and commented that, if this tumor were from a human, it would be classified as a renal cell carcinoma, granular type.

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MICROSLIDE 79

HISTORY This aged male Hartley guinea pig, Cavia porcellus, was one of two animals from a breeding colony of approximately 100 Hartley strain guinea pigs. Both guinea pigs died without exhibiting any clinical signs of disease.

GROSS PATHOLOGY The lungs did not collapse. The hilar pulmonary parenchyma was sunken, dark red and firm. The azygous lobe was atelectatic while the apical lung regions were pink and crepitant. There was an abrupt transition between the sunken hilar regions and the more normal aerated lung.

LABORATORY RESULTS Bacteriologic cultures of lung were negative for pathogens.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Bronchopneumonia, necrotizing, acute, multifocal, moderate-to-severe with intranuclear viral inclusions.

Histologically, acute necrosis of epithelial cells of bronchi, bronchioles, terminal bronchioles and associated alveoli is evident. Scattered sloughed bronchiolar epithelial cells contain hyalinized amphophilic intranuclear inclusions which marginated chromatin. Electron microscopy revealed hexagonal virions (60-70 nm in diameter) within nuclei of bronchiolar epithelial cells. Virions were either electron lucent or contained a central circular core. Virions were found singly or were packed into paracrystalline arrays.

Sporadic outbreaks of this disease have been reported from Germany, the United States and Australia. In all cases, the morbidity has been low and mortality high. The virus has not yet

been isolated and there is no serologic test available to determine the true prevalence of this virus in guinea pig colonies.

AFIP DIAGNOSIS Lung: Bronchitis/bronchiolitis, necrotizing, subacute, diffuse, mild, with intranuclear inclusion bodies, Hartley guinea pig, rodent.

Lung: Pneumonia, interstitial, subacute, diffuse, mild, with vasculitis, fibrin exudation, and hyaline membrane formation.

CONFERENCE NOTE The viral inclusions noted in the bronchioles are consistent with an adenovirus. Some conference participants felt that the changes noted involving the interstitium and alveoli are primarily due to the virus; other participants felt that the changes are due to an associated bacterial infection. Adenovirus and cytomegalovirus are the only known viral respiratory pathogens in guinea pigs. Guinea pigs have been known to have serological titers to Sendai virus, although clinical disease has not seen reported. Bordetella bronchoseptica is the primary bacterial respiratory pathogen of guinea pigs.

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REFERENCES

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MICROSLIDE 80

HISTORY This 1-year-old, male guinea pig was presented with a recurrent mass on the right maxilla between the lateral incisor and the first premolar. The mass was first noticed when the animal was 9 months of age. The tumor recurred 3 months after surgery. Clinically, the animal showed wheezing and occasionally sneezing. Radiography revealed a radiodense, polycystic mass extending into the nasal and oral cavity. The animal was euthanatized because of poor prognosis and submitted for necropsy.

GROSS PATHOLOGY Necropsy revealed a hard white mass (2.3 x 1.8 x 1.6 cm) on the right maxilla and multifocal ulcerative stomatitis. No other macroscopic lesions

were observed.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Odontoameloblastoma, maxilla, guinea pig.

Odontoameloblastoma (ameloblastic odontoma) is a rare odontogenic tumor in animals and has been described in the dog, cat, horse, cow, sheep, rat and monkey. They are described as low grade malignancies which rarely metastasize. Classification of odontogenic tumors can be difficult because of the complexity of the organ involved and the variety of cell types; histological appearance may vary between sections.

Odontoameloblastoma have distinct histomorphologic features including islands or cords of anastomosing ameloblastic cells with a layer of enamel, predentin and dentin. Odontoblasts appear as moderately differentiated polygonal cells. Other areas of the tumor consist of fibrovascular stroma reminiscent of the dental pulp. The differential diagnosis included ameloblastoma, ameloblastic fibroma, complex and compound odontoma.

AFIP DIAGNOSIS Maxillary mass (per contributor): Ameloblastic odontoma, guinea pig, rodent.

CONFERENCE NOTE The nomenclature of odontogenic tumors is confusing and histologic evaluation is often difficult. Most conference participants believe this neoplasm demonstrates features consistent with ameloblastic odontoma. The Department of Oral Pathology preferred the diagnosis of ameloblastic fibro-odontoma because of the prominence of the stromal spindle cells (considered a component of the neoplasm) that surrounds the islands of odontogenic epithelium.

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REFERENCES

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animals. Journal of Comparative Pathology, 97: 503-521. 1987.

MICROSLIDE 81

HISTORY This 8-week-old female Syrian golden hamster was treated with 50 micromoles of cadmium chloride subcutaneously 20 hours prior to necropsy.

GROSS PATHOLOGY Kidneys appeared normal.

CONTRIBUTOR'S DIAGNOSIS AND COMMENTS Necrosis, proximal tubules, focal.

There is moderate to severe necrosis of the proximal convoluted tubular epithelium. Degenerating and necrotic cells have eosinophilic, vacuolated cytoplasm, and nuclei show chromatin margination before undergoing lysis or pyknosis; re-epithelialization occurs within one week. Necrotic debris is cleared via the urine or is phagocytosed by macrophages and multinucleated giant cells. Mineralization may occur and may be visible grossly. Ultrastructurally, there is diffuse vacuolization of the cytoplasm involving the vesicular apparatus and endoplasmic reticulum. Initially small vesicles coalesce forming larger vacuoles until eventually cell rupture occurs.

The exact pathogenesis of $CdC1_2$-induced acute kidney lesions is not known, and the lesions are unique to the Syrian golden hamster. In other species, acute and chronic renal lesions have only been reported with cadmium-metallothionein. In the kidney there is a correlation between the sites of lesions and renal metabolism of compounds. Most lesions occur in proximal tubules due to resorptive processes or metabolic activation. Cadmium-metallothionein is resorbed in the proximal tubule by endocytosis and interacts with the phagolysomomal system before cell lysis occurs.

AFIP DIAGNOSIS Kidney, proximal tubules: Vacuolar degeneration and necrosis, acute, multifocal, moderate, Syrian golden hamster, rodent.

CONFERENCE NOTE Cytoplasmic vacuolation followed by necrosis of proximal tubular epithelial cells should suggest toxic renal damage. In contrast, ischemic damage is characterized by patchy coagulation necrosis of short segments of proximal tubules and the ascending loop of Henle. We staged the necrosis as acute because of the absence of regenerative changes and inflammation. Another effect of cadmium is injection site sarcoma when administered intramuscularly or subcutaneously.

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REFERENCES

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MICROSLIDE 82

HISTORY This adult male Zucker rat was one of three adults submitted for routine health screening.

GROSS PATHOLOGY No abnormalities were found on gross necropsy.

LABORATORY RESULTS Mycoplasma pulmonis was cultured from the nasopharynx of all three animals examined. All three animals had positive serological titers (ELISA) to Mycoplasma pulmonis.

CONTRIBUTOR'S DIAGNOSIS AND COMMENTS Lymphoid hyperplasia with follicle formation, submucosa of small bronchioles.

There was lymphocyte infiltration of the muscularis, lamina propria, and respiratory epithelium. Cilia-associated respiratory (CAR) bacillus was identified by electron microscopy. Mycoplasma pulmonis was identified by culture and serology.

Experimental investigations have proven that the cilia-associated respiratory bacillus of rats is a primary pathogen. The CAR bacillus causes peribronchial lymphoid hyperplasia, bronchiectasis and bronchiectasis. We have proven in our laboratory, as have other investigators, that the CAR bacillus infection occurs concurrently with Mycoplasma pulmonis, another agent that reportedly can cause lymphoid hyperplasia, bronchiectasis and bronchiolectasis. There is some concern for what actual role Mycoplasma pulmonis plays in causation of murine chronic respiratory disease. The early studies with Mycoplasma pulmonis did not specifically look for (neither with serology nor electron microscopy), or attempt to culture the CAR bacillus.

AFIP DIAGNOSIS Lung, bronchus associate lymphoid tissue: Hyperplasia, focally extensive, moderate, with cilia-associated bacteria, Zucker rat, rodent.

CONFERENCE NOTE Spontaneous infection with cilia-associated respiratory (CAR) bacillus has been reported in rats, mice, and rabbits. Although, as the contributor comments, several studies indicate CAR bacillus may be a primary pathogen, the organism's role in murine chronic respiratory disease has yet to be fully elucidated.

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REFERENCES

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MICROSLIDE 83

HISTORY This male Fischer 344 rat was a control in a two-year carcinogenesis bioassay. The animal was euthanatized at 112 weeks of age at the termination of the study.

GROSS PATHOLOGY The kidneys exhibited a diffusely "granular" external surface.

CONTRIBUTOR'S DIAGNOSIS AND COMMENTS Nephropathy, chronic, marked.

This case is a representative example of a spontaneously occurring aging lesion seen in many strains of laboratory rats. This condition is known under many synonyms including chronic progressive nephrosis, old rat nephropathy, glomerulosclerosis, chronic nephritis, glomerulonephritis and others. The exact pathogenesis is unknown.

Generally, lesions are more pronounced in male rats than in females. In advanced cases, the kidneys are grossly enlarged with irregularly pitted and granular external surfaces.

Typical histologic features demonstrated by this case include: thickened hyalinized glomerular and cortical tubular basement membranes, dilatation of cortical tubules by homogeneous eosinophilic proteinaceous casts, regenerative tubules with cuboidal basophilic epithelium, glomerular adhesions, increased mesangial density and interstitial accumulations of primarily mononuclear inflammatory cells.

AFIP DIAGNOSIS Kidney: Nephropathy, chronic, diffuse, moderate, Fischer 344 rat, rodent.

CONFERENCE NOTE The diagnosis of nephropathy was felt to be an adequate morphological diagnosis since this condition is a well recognized entity in the rat. In addition to the histologic features listed by the contributor, we also believe there is multifocal hyperplasia of proximal

tubular epithelial cells. Although there are many current and often conflicting reports on the affects of caloric and/or protein restricted diets, the role of factors such as age, sex, strain and diet in the pathogenesis of this syndrome remains undetermined.

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REFERENCES

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MICROSLIDE 84

HISTORY A group of 15-18 month old retired breeder female C3H/HeNCr MTV mice developed respiratory distress of varying degrees.

GROSS PATHOLOGY Lungs were congested and swollen.

LABORATORY RESULTS None.

CONTRIBUTOR'S DIAGNOSIS AND COMMENTS Foreign body inhalation pneumonia, food or stomach contents.

The 3 mice exhibit varying degrees of inhalation pneumonia. In a study of more than 100 female mice of this strain, approximately 10% have varying degrees of inhalation pneumonia. No other mice in this facility display such a high incidence of this condition. The cause is unknown. Muscle lesions in the larynx or other tissues indicative of vitamin deficiency have not been found. Standard diets are used.

AFIP DIAGNOSIS Lung: Bronchopneumonia, pyogranulomatous, multifocal to coalescing, severe, with plant material, C_3H mouse, rodent.

CONFERENCE NOTE None.

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REFERENCE

Dixon, D., and Jure, M. N.: Diagnostic exercise: Pneumonia in a rat. Laboratory Animal Science 38:727-728. 1988.

MICROSLIDE 85, 35 MM SLIDE 17

HISTORY This 5-year-old male domestic shorthair cat was anoretic and depressed. An abdominal mass was evident upon palpation and radiographically. A laparotomy was performed and a mass from the duodenum was removed along with an associated mesenteric lymph node and a biopsy of liver.

GROSS PATHOLOGY The duodenal mass was round and measured 3.5 cm in diameter. The lymph node was enlarged to 2x3 cm. The liver was yellow and friable. The pancreas appeared within normal limits.

LABORATORY RESULTS WBC of 28,000.

ALP = 555 U/L (normal less than 90 U/L).

Total bilirubin = 2.5 mg/dl.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Globule leukocyte tumor, duodenum, mesenteric lymph node, and liver, domestic shorthair, cat.

Cytologic, histologic, and ultrastructural features were consistent with the diagnosis of globule leukocyte tumor and were similar to a case recently described in the literature (Honor, D.J. and De Nicola, D.B., et. al. 1986). Impression smears of the duodenal mass revealed round cells with moderate numbers of round and purple intracytoplasmic granules. These granules were pale and eosinophilic with an H&E stain and were not metachromatic with a Giemsa stain. Ultrastructurally the granules measured approximately 1 um in diameter and were bound by a single unit membrane. As noted in one of the submitted electron micrographs the morphology of the granules is distinct from the appearance of the feline eosinophil granule and is not consistent with the description for those from neoplastic large granular lymphocytes (Franks, P.T. and Harvey, J.W., et. al. 1986).

Globule leukocyte neoplasms have only been reported twice previously and have only been described in cats. As in these previous reports this tumor involved the small intestine and liver. Unfortunately, additional tissues including thymus, bone marrow and blood were unavailable for analysis. Although first described in 1919, the origin and function of globule leukocytes remains controversial. Derivation of these cells from lymphocytes, plasma cells, eosinophils, mast cells, and a unique mesenchymal cell population has been suggested. The latter is considered most likely at present. The function of globule leukocytes is poorly understood. The association between these cells and parasitic infections has been well-documented and intracellular IgE has been demonstrated in intestinal globule leukocytes of rats with enteric parasites. Increased numbers of globule leukocytes have been shown to be associated with gastrointestinal parasitism in sheep, cats, dogs, rats and mice.

AFIP DIAGNOSIS Duodenum (per contributor): Malignant round cell tumor with eosinophilic granules, domestic shorthair, feline.

CONFERENCE NOTE Based on the materials available for our review, we are unable to confirm or refute the contributor's diagnosis. Compared to the easily distinguishable globule leukocytes in the mucosa (interpreted as normal), the eosinophilic granules in neoplastic cells are smaller and less numerous than we would expect in a globule leukocyte neoplasm.

The Department of Hematologic and Lymphatic Pathology favored a leukemic process, most likely of granulocytic origin; however, the Leder's stain is negative.

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REFERENCES

Finn, J. P., and Schwartz, L. W.: A neoplasm of globule leukocytes in the intestine of a cat. Journal of Comparative Pathology, 82: 323. 1972.

Franks, P. T., and Harvey, J. W., et al.: Feline large granular lymphoma. Veterinary Pathology, 23: 200-202. 1986.

Honor, D. J., and De Nicola, D. B., et al.: A neoplasm of globule leukocytes in a cat. Veterinary Pathology, 23: 287-292. 1986.

MICROSLIDE 86

HISTORY This adult male rhesus macaque was one of several hundred macaques dying from a hemorrhagic diathesis. This animal first demonstrated signs of anorexia, lethargy and ruffled hair coat on a Saturday. By the following Monday he was extremely depressed, could not stand up and had epistaxis. The monkey was euthanatized.

GROSS PATHOLOGY There was subcutaneous hemorrhage noted in the temporal region of the head. Lymph nodes and spleen were twice normal weight and white pulp was not evident grossly on the cut surface of the spleen. A thin line of hemorrhage was present at the gastoduodenal junction. The urinary bladder did not contain urine and the kidneys were friable and had a granular appearance. The synovium of the stifle joint appeared hyperemic.

LABORATORY RESULTS Numerous schistocytes were seen on peripheral blood smears. Both PT and APTT were prolonged. Platelet numbers were low-normal but failed to aggregate in response to either ADP or collagen.

Clinical chemistries revealed:

AST = 1980 U/L.

ALT = 142 U/L.

LDH = 16,050 U/L (normal = 426).

FDP titer= 128.

CONTRIBUTOR DIAGNOSIS AND COMMENT Necrosis, lymphoid cells, diffuse, moderate to severe, white pulp, spleen, rhesus macaque. Etiology: Simian hemorrhagic fever.

Necrosis with fibrin deposition, diffuse, severe, cords of Bilroth, red pulp, spleen. Etiology: Simian hemorrhagic fever.

Other histologic lesions included: disseminated lymphoid necrosis including complete necrosis of the thymic cortex; necrosis of the hematopoietic cords in the bone marrow; multifocal fibrin thrombi in the medulla of the kidney; lymphocytic epididymitis; hemorrhagic gastritis; necrosis of villus tip macrophages and intraepithelial lymphocytes in the small intestine; and diffuse, nonsuppurative meningoencephalitis and myelitis.

In a period of approximately 2 months during 1989 more than 400 rhesus and cynomolgus monkeys died at a primate research facility in New Mexico due to infection with Simian Hemorrhagic Fever (SHF). The outbreak coincided with the importation of 50 cynomolgus monkeys from an importer in Miami, Florida. Within 2 weeks of arrival these animals all died or were euthanatized and macaques in other rooms and in outside cages had begun to die. Animals of all ages were affected. Clinical signs included epistaxis, periocular hemorrhage, melena, petechiae, ecchymoses, lethargy, anorexia and incoordination. The cause of death was not immediately apparent in the cynomolgus monkeys which died acutely. Rhesus monkeys had proximal duodenal hemorrhage allowing a presumptive diagnosis of SHF although other hemorrhagic diseases were considered in the differential diagnosis including Ebola virus, Marburg virus, Lassa fever, and Rift valley fever. These were subsequently ruled out by virus isolation and immunohistochemistry. SHF was confirmed by isolation, EM, and FA. This animal had signs and lesions typical of the final stage of disease. Monkeys are generally viremic with elevated FDP's for several days prior to the onset of clinical signs or abnormal serum chemistries. Screening monkeys for antibody against SHF is not useful. Infected animals typically do not develop detectable antibody against SHF. Furthermore, African primates, which serve as a reservoir for the virus, may or may not be antibody positive while being viremic. To date, due to the lack of an antigen-capture ELISA, the only effective way to scree primates is by viral isolation. The gross lesions described and associated histopathology are not pathognomonic for SHF and can be seen in any of the diseases described above. The splenic lesion in particular frequently appears in association with DIC. Discrete foci of hepatic and adrenal necrosis, however, are not typical of SHF infection and if present should be considered presumptive evidence of Ebola or Marburg virus infection. USAMRIID is requesting information from pathologists who currently or previously have noted lesions consistent with SHF or one of its differential diagnoses, particularly in recently imported macaques from the Philippines.

AFIP DIAGNOSIS Spleen, red pulp and cords of Bilroth: Necrosis and fibrin deposition, diffuse, severe, rhesus monkey, primate.

Spleen, white pulp: Necrosis, diffuse, mild, with marginal zone hemorrhage.

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REFERENCES

Allen, A. M., and Palmer, A. E., et al.: Simian hemorrhagic fever. Studies in pathology. American Journal of Tropical Medicine and Hygiene, pages 413-421. 1968.

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MICROSLIDE 87

HISTORY This 2-year-old female Nubian goat was a chronic poor-doer which came from a herd of purebred Nubian goats known to be positive for caprine arthritis and encephalitis virus. She developed an increased respiratory rate with harsh lung sounds, was not responsive to antibiotics and was afebrile for five weeks. She was euthanatized after developing increased respiratory difficulty with open mouthed breathing and was nonresponsive to treatment.

GROSS PATHOLOGY The carcass was thin with a rough hair coat and there was scant subcutaneous or visceral fat. The lungs were firm. There was a sharp demarcation between the firmer deeper red cranioventral regions and the firm red dorsal and diaphragmatic regions. This was most pronounced in the right cranioventral region. The pulmonary parenchyma was diffusely mottled red and white in a "snowflake" pattern. The tracheobronchial lymph nodes were enlarged. The mammary glands, joints and brain were grossly normal.

LABORATORY RESULTS Aerobic (bacterial) culture of the lungs was negative. Serology was positive for CAEV.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Interstitial pneumonia, severe, chronic, diffuse, with type II pneumocyte hyperplasia and multifocal lymphoid nodules, lung.

Suppurative bronchopneumonia, acute, multifocal, moderate to severe, lung. Etiologic agent: Caprine arthritis-encephalitis virus.

Caprine arthritis-encephalitis (CAE) is a multiple system disease with worldwide distribution in goats. It is caused by a Lentivirus which is antigenically related to maedi-visna virus of sheep but which is distinguishable from the ovine-specific virus on the basis of large differences in the nucleic acid sequences of the envelope gene. The macroscopic and histopathologic appearance of the lesions in this goat are fairly typical of those described previously in CAE virus pneumonia. This case is of interest given the relatively young age of the goat, her known CAE positive status and the absence of lesions in brain, joints and mammary gland.

AFIP DIAGNOSIS Lung: Pneumonia, interstitial, subacute, diffuse, severe, with marked type II pneumocyte and BALT hyperplasia, and intraalveolar eosinophilic exudate, Nubian, caprine.

Lung: Bronchopneumonia, suppurative, multifocal, moderate.

CONFERENCE NOTE Extrapulmonary lesions associated with CAEV infection are demyelinating nonsuppurative leukoencephalomyelitis, chronic proliferative arthritis/synovitis with carpal hygromas, and lymphocytic mastitis. The above lesions are similar to those seen in sheep with OPP. The main differences in pulmonary lesions between these two diseases are the extensive alveolar filling by proteinaceous material and the prominent type II pneumocyte hyperplasia seen in CAE. The lentivirus causing each disease appears to be cell associated with viral replication and survival within cytoplasmic vacuoles of macrophages.

We believe the bronchopneumonia may represent a secondary bacterial infection.

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REFERENCES

Cork, L. C., and Narayan, O.: The pathogenesis of viral leukoencephalomyelitis-arthritis in goats. Laboratory Investigation, 42: 596-602. 1980.

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Robinson, W. F. and Ellis, T. M.: Caprine arthritisencephalitis virus infection: From recognition to eradication. Australia Veterinary Journal, 63: 237-241. 1986.

MICROSLIDES 88 and 89

HISTORY This 11 ½-year-old female dog died in the Inhalation Toxicology Research Institute Hospital after a 4 month history of severe, progressive, idiopathic anemia.

GROSS PATHOLOGY All mucous membranes were severely blanched. The spleen was moderately enlarged and had rounded edges. On section, the spleen was firm. The femoral marrow was slightly pale, reduced in quantity and easily removed as a firm cylindrical mass.

LABORATORY RESULT A CBC performed 3 weeks prior to death yielded the following results: Total plasma protein: 6.6 gm/dl, 2.13x10^6 RBC/mm^3, HCT 15.1%, HGB 4.0 gm/dl, high platelet numbers, 3 nucleated red cells per 100 white blood cells, MCV 70 cubic microns, MCH 19 pgms, MCHC 25%, 3+ anisocytosis, 2+ poikilocytosis, 2+ hyperchromasia, 2+ giant platelets, total white count of 7,700/mm^3. The differential showed 71% segmented neutrophils, 4% band neutrophils, 1% eosinophils, and 24% lymphocytes.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Myelofibrosis, bone marrow, dog. Extramedullary hematopoiesis, severe, diffuse, dog.

The bone marrow is characterized by an absence of adipose tissue, large amounts of fibrous connective tissue, a marked myeloid predominance, and severe hypoplasia of the erythroid series. A left shift is also present in the myeloid series. Megakaryocytic hyperplasia is marked in some sections but this change tends to be locally extensive and varies between sections. Iron stores are adequate. Trabecular bone is thickened in this section of femur.

The splenic changes are considered secondary to the alterations in the bone marrow. These changes consist of marked extramedullary hematopoiesis, principally of myeloid and megakaryocytic cells. Hemosiderosis is marked.

The myeloid predominance, megakaryocytic hyperplasia, and marked erythroid hypoplasia are typical of myelofibrosis. This case of myelofibrosis was idiopathic in origin.

AFIP DIAGNOSIS Bone marrow: Myeloproliferative disease with myelofibrosis and osteomyelosclerosis, Beagle, canine.

Spleen: Myeloproliferative disease.

CONFERENCE NOTE Histologic changes in both the bone marrow and spleen are identical to those described for myeloid metaplasia in man which is a form of chronic myeloproliferative disease (specifically chronic myelogenous leukemia). This diagnosis was based upon the presence of abnormal megakaryocytes and granulocytes in both the spleen and bone marrow. Megakaryocytes occurred in clusters, were highly variable in size, often contained numerous nuclei indicating excessive ploidy, and frequently displayed bizarre mitotic figures. They were extremely numerous in the spleen exceeding expected numbers for extramedullary hematopoiesis. The prominent population of mononuclear cells in the spleen stained positive for chloroacetate esterase (Leder's) but not for factor VIII related antigen indicating they were of granulocytic lineage. Clusters of these cells were also observed in the marrow. Since there was no block in the maturation of the leukemic cells, the myeloproliferative disease (MPD) was

AFIP DIAGNOSIS Rib: Osteodystrophy characterized by metaphyseal infraction, osteoblast atrophy and osteonecrosis, with medullary fibrosis, periosteal hemorrhage and cambial hyperplasia, rhesus monkey (Macaca mulatta), primate.

CONFERENCE NOTE Vitamin C deficiency occurs only in species unable to synthesize ascorbic acid endogenously. Ascorbic acid is synthesized in the liver of most mammals and in the kidney of reptiles and amphibians. Most birds produce the vitamin in the liver, but in a few the kidney serves this function. Man, nonhuman primates, guinea pigs and bats lack the enzyme L-gulonolactone oxidase which is required for the synthesis of ascorbic acid. Insects, invertebrates, and fish generally do not synthesize vitamin C. Vitamin C is rapidly and readily absorbed from the small intestine. Prolonged periods of negative balance fail to produce scurvy since the vitamin is stored in many tissues and organs throughout the body, principally in the adrenal and pituitary glands. Other sites also have substantial reserves, including the brain, liver, spleen, pancreas, kidney and heart muscle.

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REFERENCES

Jones, T. C., and Hunt, R. D.: Veterinary Pathology, Fifth Edition, pages 1039-1042.

Cotran, R. S., Kumar, V., and Robbins, S. L.: Pathologic Basis of Disease, Fourth Edition, pages 456-459. 1989.

MICROSLIDE 92, 35 MM SLIDES 21 and 22

HISTORY This 4-year-old spayed female cat had a history of firm nodules present for weeks. A biopsy of the biggest protuberance near the scapula was sent in and the diagnosis "osteochondrosarcoma" given. The cat was destroyed some weeks later because of weakness.

GROSS PATHOLOGY There were numerous calcified nodules in all parts of the skeleton, but not in the nasal cavity or meninges.

LABORATORY RESULTS Positive for FeLV.

CONTRIBUTORS DIAGNOSIS AND COMMENT Feline osteochondromatosis (viral-induced), connected with FeLV.

AFIP DIAGNOSIS Bone, site unspecified: Multiple cartilaginous exostosis, breed unspecified, feline.

CONFERENCE NOTE Multiple cartilaginous exostosis is a condition described in dogs, horses, cats and man. It involves partially ossified protuberances of cartilage and bone usually arising from bone of endochondral origin. The condition in cats, which has been associated with feline leukemia virus, usually differs from that of the dog and horse in small ways. In the dog and horse the lesions usually develop early, arise in association with metaphyseal growth plates and are not juxtacortical since the marrow cavity of the bone is continuous with the marrow

spaces of the tumor. In cats, the tumors occur later (in young adults), often involve the skull, seldom involve the long bones and typically do not develop near osteochondral junctions.

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REFERENCES

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Doige, C. E.: Multiple cartilaginous exostosis in dogs. Veterinary Pathology, 24: 276-278. 1987.

Pool, R. R., and Carrig, C. B.: Multiple cartilaginous exostosis in a cat. Veterinary Pathology, 9: 350-359. 1972.

MICROSLIDE 93

HISTORY This tissue was taken from one of three clinically normal 1-year-old male Hartley albino guinea pigs. These were stock animals with no history of prior treatment or manipulation.

GROSS PATHOLOGY Joints were not disarticulated prior to fixation and sectioning, hence the articular surfaces were not examined grossly.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Chronic osteoarthritis.

Sections are from femorotibial joints of three different 1-year-old animals with various stages of osteoarthrosis. All have moderate to marked degenerative changes on the medial tibial plateau characterized by chondrocyte and proteoglycan loss in the superficial and middle zones of the articular cartilage and proliferation of subjacent chondrocytes to form clones. Varying degrees of fibrillation of the hypocellular matrix are present. In some animals, similar degenerative changes are evident on the medial femoral condyle and meniscus. Two animals have tibial and femoral osteophytes which have distorted the normal joint contour. Synovial membrane changes, when present, consist of slightly increased mature fibrous connective tissue and mild synoviocyte hyperplasia with occasional papillary projections. Inflammatory cell infiltration is generally not present despite fairly marked degenerative changes in the articular surfaces on the medial side. Medial femoral and tibial subchondral marrow in two animals have areas in which the hematopoietic marrow has been replaced by fibroblasts or adipocytes, and in one animal there are foci of cartilaginous metaplasia.

AFIP DIAGNOSIS Stifle: Osteoarthrosis, moderate, characterized by articular cartilage fibrillation, clefts, erosion and chondrones, and by periarticular osteophytes, Hartley guinea pig, rodent.

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REFERENCES

Bendele, A. M.: Progressive chronic osteoarthritis in femorotibial joints of partial medial meniscectomized guinea pigs. Veterinary Pathology, 24: 444-448. 1987.

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Silverstein, E., and Sokoloff, L..: Natural history of degenerative joint disease in small laboratory animals. Osteoarthritis in guinea pigs. Arthritis Rheumatism, 1: 82-86. 1958.

MICROSLIDE 94

HISTORY This 7-month-old female domestic shorthair cat was adopted as a stray at about 6 weeks of age. The cat had tremors which progressed to ataxia and eventually paralysis. She was euthanatized.

GROSS PATHOLOGY The head was flattened and dysmorphic with small pinnae. There were two hemivertebrae and marked kyphosis of the lumbar spinal column.

LABORATORY RESULTS MPS spot test-positive.

Dermatan sulfate and chondroitin sulfate in urine.

Arylsulfatase A - 0% activity.

Arylsulfatase B - 50% activity.

Vacuolation of mononuclear blood cells on smear.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Diffuse neuronal cytoplasmic granular inclusions with multifocal loss of myelin and axonal swelling - brain and spinal cord - feline.

Intracytoplasmic vacuolation of hepatocytes, renal epithelial cells, subsynovial cells, chondrocytes, follicular dendritic cells of spleen, and interstitial cells of heart. Gm_2 Gangliosidosis Type 2.

Enzyme assays of liver samples from this cat confirmed that there was absence of both hexosaminidase A and B activity. Assays of brain tissue revealed excess accumulation of Gm_2 and Gm_3 gangliosides. These changes are diagnostic of Sandhoff disease, a variant of Gm_2 gangliosidosis in humans. Tay-Sachs disease is the other more common variant, and is characterized by a deficiency of hexosaminidase A only. Gm_2 gangliosidosis, Type 2 has been described in domestic short haired cats and Korat cats. The clinical signs and lesions in this cat are typical of the reported disease.

AFIP DIAGNOSIS Cerebrum, glia and neurons: Swelling, vacuolation and degeneration, diffuse, moderate, with axonal degeneration, domestic shorthair, feline.

CONFERENCE NOTE In addition to neuronal involvement, most attendees felt that glial cells, particularly astrocytes, were also affected. Several other lysosomal storage diseases of cats and their related enzymes [GM_1 gangliosidosis (beta-galactosidase), sphingomyelin lipidosis (sphingomyelinase), alpha mannosidosis (alpha mannosidase), mucopolysaccharidosis VI (arylsulfatase B) and MPS I (alpha-L iduronidase)] were briefly discussed.

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MICROSLIDE 95, 35 MM SLIDES 23 and 24

HISTORY This wild-caught female northern leopard frog (Rana pipiens) had been in captivity with approximately 15 other wild caught leopard frogs for two years. Recently it and several other frogs in its enclosure became lethargic and developed marked ascites.

GROSS PATHOLOGY This frog's kidneys were both multinodular, white-tan, and enlarged 3-4 times normal. A fibrinous exudate covered the anterior aspects of both ovaries and extended over approximately one-half the liver's capsular surface. Another frog from the same enclosure (gross Kodachrome provided) had a multinodular, white-tan, left kidney which was enlarged approximately 10 times normal, but a normal right kidney.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Bilateral papillary renal adenocarcinoma. Etiology: Lucke' herpesvirus.

Granulomatous and hypertrophic protozoal oophoritis with secondary fibrinous and granulomatous coelomitis. Possible etiology: Microsporidium scheuetzi.

The Lucke herpesvirus, which causes renal adenocarcinoma in northern leopard frogs, has two distinct temperature dependent phases. At 4-9°C, the virus is in the algid, or winter phase, and can replicate in renal convoluted tubular epithelial cells with host cell lysis and shedding of virus in the urine. During this phase, the virus can be isolated from infected tissue and intranuclear viral inclusions are readily demonstrated. At 22-25°C, the virus is in the calid, or summer phase, and cannot be isolated from the kidney nor can viral inclusions be demonstrated. It is during this phase that viral DNA is integrated into renal tubular epithelial cell DNA, and neoplastic transformation occurs.

The frog's ovaries exhibit marked oocyte hypertrophy associated with numerous intraocytic microsporidian spores. Host cell hypertrophy induced by intracytoplasmic parasitic proliferation is referred to as a xenoma, and has been described for several genera of microsporidians (Gluea spp, Pleistophora spp). The genus and species of the microsporidian parasite in this frog's ovaries and coelomic cavity could not be determined due to our inability to identify all of its life stages, or to ascertain the number of coils in its polar filament and nuclei in its spores. Microsporidium schuetzi, described by Schuetz, Selman, and Samson, is a microsporidian found in oocytes of leopard frogs. Since M. schuetzi has morphologic features similar to those of the microsporidian in this case and has also been associated with oocyte hypertrophy, it is our tentative etiologic agent pending further studies.

AFIP DIAGNOSIS Kidney (per contributor): Adenocarcinoma, papillary, northern leopard frog, amphibian.

Ovary: Oophoritis, granulomatous, multifocal, moderate, with follicular rupture and intrahistiocytic microsporidia.

Ovarian follicle: Hypertrophy, cystic, with myriads of microsporidian parasites.

CONFERENCE NOTE An unstained section submitted by the contributor was subjected to a periodic acid-Schiff (PAS) reaction. The protozoans each contained a PAS-positive polar granule diagnostic for Microsporidium sp.

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REFERENCES

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MICROSLIDE 96

HISTORY This 1-year-old mixed breed male dog developed severe facial swelling, lethargy, nausea and methemoglobinemia following administration of an organic compound. The dog was euthanized approximately 24 hours after dosing.

GROSS PATHOLOGY There was facial (muzzle, palpebral conjunctiva) and paw edema. The liver was markedly congested and had an accentuated lobular pattern. There was considerable hilar edema of the gallbladder and marked mesenteric/pancreatic edema.

LABORATORY RESULTS

CLINICAL CHEMISTRIES

ALT	1092 IU/l
AST	546 IU/l
T. Bili	1.2 mg/dl
D. Bili	0.5 mg/dl
SAP	439 IU/l
GGT	45
LDH	290
Albumin	3.0 g/dl
BUN	10 mg/dl
Glucose	99 mg/dl

HEMATOLOGY

WBC	($x10^3$)	8.4
RBC	(xlO^6)	4.86
HCT	(%)	30.4

Differential

Segs	86	(7224)
Bands	1	(84)
Lymphs	9	(756)
Mono	4	(336)

Plasma protein 4.6 gm%
Slight polychromasia

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Severe acute centrilobular hepatic necrosis (coagulative). Etiology: Acute acetaminophen toxicity.

This dog was given a single oral dose (600 mg/kg) of acetaminophen in capsular form. Within 6 hours facial and paw edema accompanied by methemoglobinemia were observed. Elevations in ALT and AST (140 IU/l and 110 IU/l, respectively) were observed at 12 hours. The animal was subjected to euthanasia and necropsy 24 hours after dosage.

AFIP DIAGNOSIS Liver: Necrosis, centrilobular, diffuse, severe, mixed-breed, canine.

CONFERENCE NOTE The reactive metabolite of acetaminophen is formed by the endoplasmic cytochrome P-450 enzyme system. This metabolite is rapidly conjugated with

glutathione (via hepatic glutathione transferase) and converted to a nontoxic mercapturic acid. Hepatic necrosis occurs after sufficient acetaminophen is administered to deplete more than 70% of hepatic glutathione. Cats lack the specific glucuronyl transferases required to conjugate the aromatic rings of many xenobiotics. This explains their increased sensitivity to acetaminophen and other aromatic toxins.

Considered in the differential diagnosis are hepatotoxicities or idiosyncratic reactions with mebendazole, blue-green algae, anticonvulsant therapy, or inhalation anesthetics.

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REFERENCES

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MICROSLIDES 97 and 98

HISTORY This 6 ½-year-old male Australian terrier had a five week history of pneumonia with a high respiratory rate and fever. This coincided with corticosteroid treatment for lameness of the left elbow and shoulder. There was a history of pulmonary tuberculosis in family. Radiographs showed diffuse severe alveolar infiltrates; trachea washings were positive with Ziehl-Neelsen stain.

GROSS PATHOLOGY Throughout all lung lobes on both sides there are multiple, coalescing areas of firm, pale, grey tissue measuring up to a few millimeters in greatest dimension. There is umbilication of the pleural surfaces of these abnormally firm areas. The lungs are fixed whole in formalin in view of the bacteriological suspicion of infection. The visceral and pleural surfaces are noticeably smooth and glistening, with the exception of occasional pale, miliary foci on the parietal pleura of the thorax and the cranial diaphragm.

LABORATORY RESULTS Tracheal washings reveal moderate numbers of acid and alcohol fast bacilli. Post mortem samples of lung yielded a growth of Mycobacterium tuberculosis. Since this is of presumed human origin, further specific typing is in progress.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Lung: Pneumonia, chronic-active, multifocal.

Spleen, liver, lymph nodes, pleura and pericardium: Granulomatosis, multifocal, coalescing. Consistent with tuberculosis.

This case is of interest because of the present relative infrequency of TB in dogs. This dog is from a household in which two family members have been successfully treated for pulmonary tuberculosis. The recent history of clinical illness in this previously well dog coincided with the use of corticosteroid for treatment of an elbow lameness. It is a matter for speculation that the immunosuppressive effects of this treatment caused exacerbation of a clinically unrecognized tuberculous infection.

AFIP DIAGNOSIS Lung: Pneumonia, Pyogranulomatous, multifocal to coalescing, moderate, with congestion, edema, acid fast bacilli, and granulomatous pleuritis, Australian terrier, canine.

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REFERENCES

Carpenter, J. L., et al.: Tuberculosis in five basset hounds. Journal of the American Veterinary Medical Association, 192: 1563-1568. 1988.

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MICROSLIDE 99

HISTORY This adult female Patas monkey (Erythrocebus patas) had been normal until the evening of her death, when she exhibited anorexia and lethargy. The monkey had been purchased from a sale one month previously. She was housed in an outdoor cage until one week prior to her death when she was moved indoors in a cage adjacent to rhesus monkeys. Her diet consisted of various fruits.

GROSS PATHOLOGY The liver contained disseminated 1-2 mm diameter gray foci. The spleen was enlarged.

LABORATORY RESULTS A herpesvirus was isolated from the liver. Gel electrophoresis of major structural proteins and restriction endonuclease analysis of the isolate were consistent with Herpesvirus simiae (B virus).

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Liver: Necrosis, multifocal, coalescing, acute, severe with intranuclear inclusions and syncytial cell formation, due to Herpesvirus simiae (B virus), Patas monkey (Erythrocebus patas).

The B virus is indigenous to macaques with 100% of adults in some colonies being infected. Clinical infection is characterized by lingual and/or labial vesicles which may ulcerate. Histologically, lesions are characterized by ballooning of cells, necrosis of epithelial cells, and intranuclear inclusion bodies. Multinucleate epithelial cells can be observed. Persistence of the agent in a latent form often occurs in the kidney and trigeminal ganglia of apparently healthy animals; thus seropositive animals should be regarded as carriers. Fatal herpesvirus infection has been previously recorded in patas monkeys which were housed near rhesus monkeys.

Herpesvirus simiae, while ubiquitous among certain non-human primates, is of considerable importance regarding its zoonotic potential. Fortunately, symptomatic infection in humans is rare with less than 35 cases having been recorded. However, most of the infected patients died from an encephalitis. Fatal cases were recently reported in Florida and Michigan. Human to human transmission of B virus was reported in one of the Florida cases.

AFIP DIAGNOSIS Liver: Necrosis, multifocal to coalescing, moderate, with hepatocellular syncytia and eosinophilic to amphophilic intranuclear inclusion bodies, patas monkey (Erythrocebus patas), primate.

CONFERENCE NOTE Discussion centered on infections by herpes viruses in a variety of animals.

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REFERENCES

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MICROSLIDE 100

HISTORY This 14 to 16-month-old Wistar-Firth rat was a control animal on a two year carcinogenicity study.

GROSS PATHOLOGY At necropsy the endocardium was diffusely thickened and white.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Endocardial schwannoma (neurilemoma).

Endocardial schwannomas (ES) are often an incidental finding at necropsy and consist of a white, opaque firm coating over the endocardial surface. The frequency of spontaneous ES varies among strains of rats, but they are much more common in aged rats. Rare cases of cardiac neurogenic tumors have been reported in man, but they usually do not originate from the endocardium.

ES are malignant neoplasms which are often locally invasive, but invasion of other mediastinal structures has been reported. Lung metastases are uncommon. It is hypothesized that ES arise from intracardiac branches of the vagus nerve. ES consist of a uniform, dense population of elongate, fusiform cells which are arranged in an parallel fashion. Often the tumor cells are arranged in an undulating pattern, reminiscent of the appearance of normal peripheral nerve cut in longitudinal section. Tumor cells stain immunocytochemically positive for S-100 protein.

AFIP DIAGNOSIS Heart: Endocardial schwannoma, Wistar-Firth rat, rodent.

CONFERENCE NOTE Anitschkow's nuclear pattern is a prominent feature of tumor cells in some areas of the neoplasm. Anitschkow's nuclei are ellipsoidal, have distinct nuclear membranes, and are pale except for an intensely stained central bar of chromatin along the long axis. One of the cited references (Rice, et al.) comments that this pattern has been observed in most normal cell types in the heart and in carcinoma cells metastatic to the heart.

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REFERENCES

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MICROSLIDE 101

HISTORY This was an incidental finding in a 4-month-old male Fischer 344 rat.

GROSS PATHOLOGY A firm, pale nodule, 1.5 cm diameter, involved the left kidney.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Nephroblastoma.

Slight focal cystic tubular dilatation.

Diagnosis of nephroblastoma was made based on the presence of blastemal, epithelial and stromal elements. Mitotic figures were not common. Although uncommon, these neoplasms have been described in a variety of rat strains. Occurrence is greatest in rats 5 to 6 months old. This lesion was interpreted to be unrelated to treatment.

AFIP DIAGNOSIS Kidney: Nephroblastoma, Fischer 344 rat, rodent.

CONFERENCE NOTE Primary renal neoplasms are uncommon in domestic animals. Spontaneous nephroblastoma has been reported in a number of species, including pigs, chickens, rabbits, dogs, cats, sheep, horses, foxes, rats and snakes. Nephroblastoma is the most common primary renal tumor of pigs and chickens; most are observed in young animals. Most authors suggest that nephroblastoma arises from the metanephrogenic renal blastema which normally develops into both the nephrons and the renal interstitial tissue. Metanephric blastema is known to persist into the post natal period and probably represents the source of this tumor. Nephroblastoma has been induced with various chemical compounds.

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REFERENCE

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MICROSLIDE 102

HISTORY This 6-month-old female Cashmere goat was one of a group of 400 on pasture. Several died and this one was found recumbent with fecal staining of the perineum. The goat was drenched with a broad spectrum anthelmintic one week earlier.

GROSS PATHOLOGY The carcass is not grossly anaemic. There is marked edema of the mesentery, mesenteric lymph nodes and wall of the jejunum. The luminal contents of the small intestine are watery and dark brown. The feces in the rectum are pelleted.

LABORATORY RESULTS Total serum protein: 22 g/L (normal range 60-70 g/L)

Microbiology: Liver: A single colony of Yersinia pseudotuberculosis.

Jejunum, ileum, colon: Heavy mixed growth dominated by Y. pseudotuberculosis.

Parasitology: Total worm count: Abomasum: 200 Haemonchus contortus

Small intestine: 600 Trichostrongylus colubriformis

Fecal egg count: 1720 Strongyle eggs per gram. (Differential.: 2% Haemonchus sp., 91% Trichostrongylus sp., 7% Ostertagia sp.)

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Necrotizing enteritis with microabscessation; intestinal yersiniosis. Marked hypoproteinemia from intestinal trichostrongylosis and yersiniosis.

Yersinia pseudotuberculosis is an opportunistic pathogen which in this case was superimposed on a heavy gastrointestinal infection of mainly Trichostrongylus sp. The high fecal egg count is from the heavy nematode burden killed by anthelmintic treatment one week before necropsy.

At all levels of the small intestine there are multiple, often confluent, necrotic foci at the villous tips with bacterial microcolonization and a peripheral zone of neutrophilic infiltration. Earliest lesions are within the lamina propria below intact epithelium at the tip of the villi and at the base of the crypts. These segmental lesions of microabscessation are characteristic of infection by Y. pseudotuberculosis. There are many globule leukocytes in the epithelium and lamina propria. Small numbers of coccidial forms were found in some sections.

AFIP DIAGNOSIS Small intestine (per contributor): Enteritis, necrotizing, acute, multifocal, mild, with multiple bacterial colonies composed of myriads of coccobacilli, Cashmere, caprine.

Small intestine: Enteritis, subacute, proliferative, diffuse, moderate, with microherniation of crypts.

CONFERENCE NOTE In addition to the presence of coccidia in some sections, several participants also described a focal submucosal granuloma containing foreign material (perhaps plant material or remnants of a metazoan parasite).

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REFERENCES

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Slee, K. J., Brightling, P., and Seiler, R. J.: Enteritis in cattle due to Yersinia pseudotuberculosis infection. Australia Veterinary Journal, 65: 271-275. 1988.

MICROSLIDE 103

HISTORY This tissue is from a 10-year-old male Doberman Pinscher. It had a 5 month history of ascites and diuretics were administered during that time. The dog had bilirubinuria but no other urinalyses or blood chemistries were done. One week before death the dog became stuporous and nonambulant.

GROSS PATHOLOGY There were 4 liters of yellow, clear, watery fluid in the peritoneal cavity. The liver was approximately 2/3 normal size, firm and had several 1-4 cm diameter nodules projecting from all lobes. All other organs were grossly unremarkable.

LABORATORY RESULTS Atomic absorption analysis of liver revealed a copper content of 600 ppm on a wet weight basis (normal = 50-70 ppm).

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Cirrhosis with diffuse, periportal, intrahepatocellular cytoplasmic pigment granules.

Hepatocellular degradation, diffuse, mild.

Intracanalicular bile stasis, multifocal, mild.

Tissue sections stained with rubeanic acid revealed copper-positive granules in the cytoplasm of hepatocytes predominantly in periportal regions. This is the typical distribution of copper in Doberman Pinschers with copper-associated hepatitis/cirrhosis. In contrast, Bedlington terriers and West Highland white terriers with copper toxicosis initially accumulate copper-positive granules in centrilobular hepatocytes. Other stains useful for detecting copper or copper-associated protein in histologic sections are rhodamine and orcein, respectively.

This case was interesting because it involved a male Doberman Pinscher. Approximately 95% of the reported cases have been in females. The reasons for this sex predilection are unknown.

The etiology of this disease is also unknown. Due to the breed predilection, some investigators have suggested a genetic abnormality may be involved. The role of copper in the pathogenesis

of hepatitis in Doberman Pinschers is unresolved.

AFIP DIAGNOSIS Liver: Cirrhosis, moderate, with multifocal portal to random subacute hepatitis, hepatocellular necrosis, and hepatocellular cytoplasmic pigment granules, Doberman Pinscher, canine.

Intrahepatic portal veins: Thrombosis, organizing, multifocal, moderate to severe.

CONFERENCE NOTE As in past conferences, the liver lesions as seen in this case elicited substantial controversy over the proper histomorphologic diagnosis. The one chosen above, though not perfect and unlikely to please everyone, was considered to best represent the histopathologic changes present in the sections we reviewed; it is basically the same as used by the contributor. We also observed organizing thrombi in several medium to large portal veins. The hepatic parenchymal changes seen in this case may be a result of both chronic-active hepatitis due to copper overload and a portal venopathy. Special stains demonstrated both iron and copper within Kupffer cells throughout the liver.

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REFERENCES

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MICROSLIDE 104

HISTORY This 3-year-old spayed female domestic shorthair cat had abnormal footpads. The central pads of one front and one hind foot were soft and swollen. The central pad of the opposite front foot was ulcerated and swollen; tissue from this pad was submitted.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Ulcerative plasmacytic pododermatitis.

The subepithelial tissue is heavily infiltrated by a predominantly plasmacytic inflammatory cell population. Many of the plasma cells contain globular eosinophilic Russell bodies which often appear as a single large round eosinophilic structure in an artefactual clear vacuole. Specimens are often submitted from ulcerated foot pads with granulation tissue, bacterial infection, suppurative inflammation and plant material present beneath the ulcerated surface. Often, as in this case, the deep tissue is infiltrated by numerous plasma cells and lymphocytes. The syndrome is similar to lymphoplasmacytic stomatitis/gingivitis/pharyngitis and both footpad and oral lesions are sometimes seen in the same animal.

AFIP DIAGNOSIS Central pad from forepaw (per contributor): Pododermatitis, ulcerative, lymphoplasmacytic, chronic, diffuse, severe, domestic shorthair, feline.

CONFERENCE NOTE Tissue Gram stains and fungal stains (GMS, PAS) failed to reveal an etiologic agent. A recanalized thrombosed vein is present in some sections. The cause of this condition is unknown. It sometimes occurs as an outbreak in colonies of cats. Lymphocytosis, neutrophilia and hypergammaglobulinemia are common. Viral, bacterial and fungal cultures are usually negative. In all reported cases, the cats were negative for feline leukemia virus infection. The disease is suspected to have an immunologic basis.

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REFERENCES

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MICROSLIDE 105, 35 MM SLIDE 25

HISTORY A mass on the top of the head of this 9-year-old spayed female yellow Labrador retriever dog had been surgically removed three times within one year. The mass had been growing more rapidly after the last surgery, approximately one month prior to the latest presentation. Radiographs revealed pulmonary metastases, and the dog was euthanized. The dog was receiving cortisone therapy at the time of the most recent presentation.

GROSS PATHOLOGY There was a 7x12x12 cm firm swelling over the left parietal bone. On cross section, this mass was pink/tan with a dark red center and gritty texture throughout. Red/brown gelatinous material filled a 1x2x3.5 cm central cavitation. Adjacent parietal bone was soft and adherent to the meninges where the mass had locally invaded. The lungs contained numerous tan, slightly gritty, firm, 0.25-1.0 cm diameter nodules randomly distributed throughout all lobes.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Multilobular osteosarcoma/chondrosarcoma.

The gross and histologic features of the parietal mass were typical of a multilobular osteoma/chondroma (chondroma rodens), with characteristically aggressive local growth. Many pulmonary metastases were less well differentiated, although special stains conclusively demonstrated the presence of both cartilage and osteoid in the metastatic foci. Thus this is a rare example of a malignant multilobular osteochondroma.

AFIP DIAGNOSIS Mass from calvarium (per contributor) and lung: Multilobular osteochondrosarcoma, Labrador retriever, canine.

CONFERENCE NOTE Multilobular osteochondrosarcomas (also known as multilobular osteosarcoma, multilobular osteoma/chondroma, or chondroma rodens) have a trilaminar morphology characterized by a peripheral dense fibrous connective tissue stroma (corresponding to the fibrous layer of the periosteum) surrounding an inner myxomatous layer containing more polyhedral mesenchymal cells (corresponding to the cambium layer), and a central chondro-osseous tissue. This arrangement recapitulates intramembranous ossification.

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REFERENCES

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MICROSLIDE 106, 35 MM SLIDE 26

HISTORY This 8-month-old male golden retriever-cross dog had bilateral exophthalmos and impaired vision for a 5-day period. Intraocular examination findings were normal except for a mild increase in intraocular pressure.

GROSS PATHOLOGY All extraocular muscles, except the retractor bulbi, were swollen and had extensive pale regions that were most severe in their mid-bellies.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Bilateral, acute, severe, diffuse, lymphocytic and histiocytic, extraocular polymyositis. Etiology: suspect immune-mediated response to molecular antigens of 6 of the 7 extraocular muscles (the retractor bulbi was not involved).

This previously unreported syndrome has been recognized in dogs of several breeds and has been successfully treated with corticosteroids.

AFIP DIAGNOSIS Extraocular skeletal muscle: Myositis, lymphohistiocytic, multifocal to coalescing, severe, golden retriever, canine.

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REFERENCES

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MICROSLIDE 107

HISTORY The tissue is from an aborted near-term Dorset lamb. There have been other recent abortions in the herd and one year prior there was a similar problem with abortions and stillbirths. Ewes continue to eat. They are fed hay and mixed grain, are housed inside and are vaccinated against Campylobacter.

GROSS PATHOLOGY The placenta shows severe, leathery yellow thickening in intercotyledonary areas throughout. Some cotyledons, survive but others are small and yellow. The horns of the placenta are completely affected. There are no lesions in the lamb.

LABORATORY RESULTS Large numbers of mixed flora (beta-hemolytic Streptococcus sp., Bacterioides sp., E. coli) were recovered from the placenta. Examination of multiple placental smears with a modified acid-fast stain (modified Koster's) did not reveal Coxiella organisms; autolysis may have been too advanced. The organisms were confirmed to be Coxiella burnetti by transmission electron microscopy.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Severe necrotizing placentitis; Coxiella burnetti.

The finding in trophoblasts of massive numbers of small basophilic organisms with a vacuolated or granular appearance is typical of Coxiella infection. Organisms are usually abundant in smears, are easily recognizable (0.2 x 0.5 um coccoid form, 0.2 x 2 um filamentous form) and are more pleomorphic than chlamydia, which are 0.3 - 0.5 um cocci. Failure to identify organisms in such a case is unusual (stainability of the organisms may have been lost due to autolysis). Immunity to Coxiella is poor, and repeat abortions are to be expected.

AFIP DIAGNOSIS Placenta, cotyledon: Placentitis, necrohemorrhagic, acute, diffuse, moderate, with intratrophoblastic and extracellular bacterial colonies, and acute vasculitis, Dorset, ovine.

CONFERENCE NOTE Infection with Coxiella burnetti typically involves the intercotyledonary placenta without vasculitis; therefore the presentation of this case is somewhat unusual. Involvement of the cotyledonary placenta with vasculitis is more commonly observed in brucellosis and chlamydiosis. Coxiella burnetti is known to infect most wild and domestic mammals, birds and man. It is found in many species of ticks and can be passed transovarially or transtadially. Ruminants appear to be the major reservoir for this organism. Transmission to susceptible animals is by tick bites, ingestion of tick feces, contaminated material, ingestion of contaminated body tissues and fluids and by inhalation (most common means of contamination for man).

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REFERENCES

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MICROSLIDES 108 and 109

HISTORY This approximately 9-month-old aborted equine fetus was submitted along with the placenta to the Louisiana State Veterinary Diagnostic Laboratory. The fetus and placenta were normal in size and development and were not autolytic. The fetus weighed 14 kilograms. The dam, a 9-year-old Quarterhorse, appeared normal before the abortion.

GROSS PATHOLOGY The chorion was diffusely purple-red and had scattered large, red-brown areas of necrosis. There was a moderate amount of thick, yellow-brown exudate adhered to the chorion surrounding the cervical star. Numerous, variably-sized epithelial

plaques were scattered on the surface of the amnion. A moderate amount of white, flaky material resembling keratin was scattered over the hair coat of the fetus. No gross lesions were present in the internal organs of the fetus.

LABORATORY RESULTS Placenta - greater than 300 CFU Nocardia spp. The isolate was submitted to NADL for specific identification. Although the isolate did not match the identifying keys exactly, it had characteristics most similar to Nocardia aerocolonigenes.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Placentitis, necropurulent, subacute, diffuse, severe, with intralesional gram-positive bacteria. Etiology: Nocardia spp., probable Nocardia aerocolonigenes.

Nocardial infections in horses are rare. In a review of Nocardia asteroides infections in horses, only 16 cases were diagnosed over a 18 year period. Fourteen of the horses had severe systemic Nocardial infections; in 13 of these animals there was strong evidence of immunosuppression (combined immunodeficiency disease of Arabian foals, hyperadrenocorticism, neoplasia). Two of the 16 cases had localized Nocardia infections associated with traumatically induced wounds.

Recently, two cases of equine abortion caused by Nocardia were reported. In both cases, there were lesions in the placenta and in fetal lung and liver. Diagnosis of Nocardia sp. infection in both these abortions was based on morphologic (beaded, branching, filamentous rods) and staining features (gram-positive, partially acid-fast) of the organism.

In this case of nocardial abortion, lesions were limited to the placenta; the internal organs of the fetus were normal. Large numbers of gram-positive, branching, filamentous bacteria were seen in tissue sections from the placenta. However, repeated attempts to stain the organism in tissue section with the Ziehl-Neelsen and Fites stains for acid-fast bacteria were unsuccessful. Nocardia asteroides is often acid-fast in tissue section but other species of the Nocardia usually do not stain acid-fast. The National Animal Disease Laboratory identified the organism isolated from this case as a species of Nocardia with characteristics most similar to Nocardia aerocolonigenes.

AFIP DIAGNOSIS Placenta, chorion: Chorionitis, necrosuppurative, subacute, diffuse, severe, with gram positive filamentous bacteria, breed unspecified, equine.

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REFERENCES

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MICROSLIDE 110

HISTORY This 30-year-old female giant panda was ill for more than 5 months. The signs included loss of appetite, lethargy, convulsions, tetany and reduced body weight. Some days prior to death there was epistaxis.

GROSS PATHOLOGY Three enlarged lymph nodes in the mesentery root and a medium sized tumor in mesentery were seen. The intestinal wall was swollen and there were more than 20 small tumorous masses on the serous surface. The liver, spleen and portal lymph nodes were all enlarged.

LABORATORY RESULTS

	A	B
Hemoglobin	9g	6g
Number of RBC	488 x $10^4/mm^3$	600 X $10^4/mm^3$
Total number of WBC	12,400/mm	10,000/mm
Neutrophil	80%	76%
Lymphocyte	18%	22%
Monocyte	2%	1%

A -- results three months after disease
B -- results one week before death

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Lymphosarcoma of giant panda.

The structure of cortical and medullary regions of the lymph node were destroyed. There were diffuse infiltration of lymphocytes in the trabeculae, capsule and medullary cords. Sinuses were filled with tumorous lymphocytes. The tumor cells were different in size and mitosis was obvious.

AFIP DIAGNOSIS Lymph node: Lymphosarcoma, giant panda (Ailuropeda melanoleuca).

CONFERENCE NOTE Effacement of nodal architecture and invasion of the capsule and adjacent tissues by sheets of uniform, morphologically abnormal lymphoid cells is diagnostic of lymphosarcoma. Lymphosarcoma was diagnosed in another giant panda that recently died at the Mexico City Zoo.

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MICROSLIDE 111

HISTORY This 1-year-old female mixed breed dog developed a dark red to black vaginal discharge 5 weeks post-partum. The condition failed to respond to antibiotic therapy (Amoxicillin). Exploratory laparotomy and an ovariohysterectomy were done at 7 weeks post partum.

GROSS PATHOLOGY The uterus was enlarged with many oval swellings. Intraluminal tissue at these sites was nondescript, red-brown and necrotic, but attached to the endometrium. Ovaries were not submitted for evaluation.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Subinvolution of placental sites (SIPS).

The history of this case is different than most SIP cases we have seen. A persistent blood tinged vaginal discharge is usually observed. Recovery from surgery was uneventful.

Most sections show cytoplasmic vacuolation of endometrial surface epithelium. The contents of the uterine lumen consists of a mixture of amorphous eosinophilic material, tissue debris and endometrium attempting regeneration.

AFIP DIAGNOSIS Uterus: Involution, incomplete, with hemorrhage, breed unspecified, canine.

CONFERENCE NOTE Without knowledge of the time elapsed since parturition, the above diagnosis is appropriate. The cause of this condition is unknown. Differences in degree of involution between placental sites in the same uterus suggest that hormonal influence is unlikely to be a major factor.

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Al-Bassam, M. A., et al.: Normal postpartum involution of the uterus in the dog. Canadian Journal of Comparative Medicine, 45: 217-232. 1981.

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MICROSLIDE 112

HISTORY This tissue is from a 6-year-old female Dutch horse which had been sent to the School of Veterinary Medicine for signs of central nervous system disturbance and lethargy. It did not respond to treatment with cortisone and antibiotics, became recumbent and showed tonic clonic convulsions. The horse came from an area where Borna disease had not been reported, but it had spent a few days in a village where the disease is endemic.

LABORATORY RESULTS Fluorescent antibody test for Borna Virus antigen on frozen brain sections: positive reaction.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Severe nonsuppurative encephalitis, predominantly polioencephalitis with numerous intranuclear eosinophilic inclusion bodies in neurons and moderate nonsuppurative meningitis; due to Borna disease virus, an unclassified RNA virus.

The lesions seen on this slide are typical for Borna disease and consist of a nonsuppurative meningoencephalitis with eosinophilic intranuclear inclusion bodies (Joest-Degen-bodies). The meningitis may be mild or absent even in areas with severe encephalitis. The lesions were found throughout cerebrum, hippocampus, brainstem and cerebellum and there was no significant variation in the severity of the lesions between these areas. Borna disease is strictly endemic and occurs spontaneously only in parts of Germany and Switzerland. It is caused by an unclassified RNA virus and horses, sheep and rabbits may contract the disease spontaneously. The natural reservoir for the virus is assumed to be infected domestic animals.

AFIP DIAGNOSIS Cerebrum: Encephalomeningitis, nonsuppurative, diffuse, mild, with neuronal eosinophilic intranuclear inclusion bodies, breed unspecified, equine.

CONFERENCE NOTE Conference attendees did not believe that the inflammation was as severe as recorded by the contributor. Sections varied but most revealed only mild encephalitis. The amount of meningeal involvement also varied between sections as did the presence of the inclusions.

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REFERENCES

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MICROSLIDE 113

HISTORY This 10-year-old thoroughbred mare had been transported to Kentucky to be sold. She died 10 days following transport. She had nasal discharge and labored respiration.

GROSS PATHOLOGY The thorax was filled with yellow discolored fluid. Large amounts of fibrin were deposited on the pleural surfaces of the lung, pericardial sac, rib cage and diaphragm.

LABORATORY RESULTS Streptococcus zooepidemicus was isolated from the lung and pleural fluid.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Pleuritis, fibrinopurulent, diffuse, severe, lung, thoroughbred, equine.

Pleuritis and/or pleuropneumonia associated with extensive pleural effusion constitutes a severe respiratory disease of working horses. It is seen most commonly in horses on the racetrack, horses which are in training, or horses which have been recently transported. It may be secondary to pre-existing pneumonia or lung abscessation. Streptococcus sp. is the most common isolate from affected horses; however, other aerobic bacteria, as well as, anaerobes may be isolated.

AFIP DIAGNOSIS Lung, pleura: Pleuritis, fibrinosuppurative, chronic, diffuse, severe, with mixed bacterial colonies, thoroughbred, equine.

Lung: Congestion and edema, diffuse, moderate.

CONFERENCE NOTE Small cocci, often forming chains and occasionally forming large colonies, and bacilli of varying sizes and shapes were observed in the pleural exudate; the latter were considered to represent postmortem contamination. Gram stains revealed phagocytized gram-positive cocci within neutrophils in the exudate.

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187: 721-724. 1985.

MICROSLIDE 114

HISTORY This colt foal was born alive but died an hour later. Its dam was from a dealer's yard of 30-40 horses; two other abortions occurred within the prior month.

GROSS PATHOLOGY Throughout the lungs on both sides of the chest there are irregular, alternating areas of pale aeration and firmer, solidified, pink red parenchyma. There is conspicuous peribronchial and perivascular edema. The pleura is smooth and glistening and the pleural cavity contains a slight excess of clear yellow unclotted fluid.

LABORATORY RESULTS No bacterial growth from lung or liver. Inoculation of cell-free extract of lung outer tissue culture produced a typical herpetic CPE.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Lung: Bronchiolitis, necrotizing.

Histologically the lung is congested with pulmonary and interlobular septal edema. There is multifocal necrosis of bronchiolar epithelium and patchy alveolar collapse. Within surviving bronchiolar epithelial cells eosinophilic intranuclear inclusion bodies are numerous. The histological appearance is of necrotizing bronchiolitis and lung collapse; it is compatible with herpes virus infection (EHVI).

AFIP DIAGNOSIS Lung: Bronchitis/bronchiolitis, necrotizing, acute, multifocal, moderate, with multifocal interstitial pneumonia and eosinophilic intranuclear inclusion bodies, breed unspecified, equine.

Lung: Edema, diffuse, moderate.

CONFERENCE NOTE Equine herpesvirus type 1 (EHV-1) is believed to cause three distinct clinical syndromes: abortions, respiratory disease, and neurological disease. Currently it is felt that two distinct subtypes of this herpesvirus cause these lesions. Subtype 1 causes the abortions and subtype 2 causes respiratory disease. Since these two subtypes are distinctly different, some investigators have designated these viruses as EHV-1 (equine abortion virus) and EHV-4 (equine rhinopneumonitis virus).

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MICROSLIDE 115

HISTORY This 15-year-old pony mare was submitted to an equine clinic because of recurrent colic and staff gait. On rectal examination a mass was palpated in the area of the right kidney. The horse was euthanatized per its owner's request.

GROSS PATHOLOGY The right kidney was greatly enlarged (4125 g). The renal cortex and medulla were interspersed with numerous solid white nodules. These nodules ranged from 0.5 cm to 1.0 cm in diameter.

LABORATORY RESULTS

Hematology

RBC (x 1012/1)		4.94
PCV (1/1)		0.25
Hemoglobin (g/1)		75.3
WBC (x 109/1)		4.40
Band neutrophils	(%)	7
Neutrophils	(%)	25
Lymphocytes (%)		62
Eosinophils	(%)	6

Total serum protein: 105 g/1

Electrophoretogram: elevated alpha- and beta-globulin peaks

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Severe granulomatous nephritis due to Micronema deletrix.

Renal lesions are characterized by coalescing granulomas. Parasites which are frequently associated with blood vessels are located in the center of the granulomas. Adult nematodes, larvae and eggs are detectable. Adults and larvae are characterized by a rhabditiform esophagus. They are surrounded by neutrophils. In the periphery macrophages, lymphocytes, plasma cells, eosinophils and multinucleated giant cells are present. In a suspension of renal tissue, adults had an average size of
244 um x 16.5 um and larvae had an average size of 146 um x 13.5 um.

Parasites resemble the earth nematode Micronema deletrix in size and morphology. Sporadic cases of infection by Micronema deletrix have been reported in horses and humans. Infections most likely occur through dermal or mucosal lesions. In several cases granulomas the oral and nasal cavity were described as initial alterations. In some cases the site of entrance remains unknown. Parasites spread via blood and lymph to different organs. Preferential locations are kidneys and central nervous system. No successful therapy is known and all cases described in the literature ended fatally.

AFIP DIAGNOSIS Kidney: Nephritis, granulomatous and eosinophilic, chronic, focally extensive, severe, with larval and adult rhabditid nematodes, breed unspecified pony, equine.

CONFERENCE NOTE Halicephalobus deletrix is synonymous with Micronema deletrix. Infection of two half-sibling foals born to the same mare one year apart was recently reported.

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REFERENCES

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MICROSLIDE 116, 35 MM SLIDE 27

HISTORY This tissue is from a 10-week-old cross-bred pig from a 400 pig feeder operation. The animal was euthanized for necropsy by the referring veterinarian because of chronic diarrhea and emaciation.

GROSS PATHOLOGY Significant changes were limited to the alimentary system. A severe diffuse necrotizing typhlocolitis was present.

LABORATORY RESULTS Treponema hyodysenteriae was isolated in moderate numbers from the colon. No other bacterial pathogens including Campylobacter sp. and Salmonella sp. were isolated from this animal. Fluorescent antibody tests for TGE and rotavirus were negative on frozen sections of intestine. Electron microscopic examination of intestinal contents yielded negative results for the presence of viral particles.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Colon: Colitis, necrosuppurative, subacute, diffuse, severe with large numbers of protozoan parasites consistent with Balantidium coli. Etiology: Treponema hyodysenteriae.

There is severe segmental erosion of the superficial colonic mucosa with extensive suppurative exudation. Marked goblet cell depletion of colonic crypts with occasional dilation of crypt lumens by streams of mucus are also noticed. Numerous protozoan parasites with morphologic

features consistent with Balantidium coli are present within the superficial fibrinonecrotic pseudomembrane. Warthin-Starry silver stain revealed the presence of large numbers of spirochetes within the crypt lumens and in the superficial exudate. Marked degeneration of the colonic epithelium with intracellular invasion by small groups of spirochetes are present in thin sections of the colonic mucosa examined by transmission electron microscopy. Few intraluminal inflammatory leukocytes, mostly neutrophils, are present.

Clinically, swine dysentery is characterized by severe mucohemorrhagic diarrhea, reduced weight gain, and frequently, death of susceptible pigs mostly during the weaning and growing-finishing periods. The role of B. coli in this disease has not been determined. Although swine dysentery can be controlled by drug therapy, progressive failure because of drug resistance and reinfection of medicated pigs from the environment remains a problem. Moreover, pigs which have recovered from swine dysentery may remain asymptomatic shedders of pathogenic organisms in the environment. A recent report of natural infection in swine characterized by extensive colonization of the colonic epithelium by spirochetes intimately attached to the apical cell membrane suggest a new mechanism of bacterial infection of the pig colon.

AFIP DIAGNOSIS Colon: Colitis, necrosuppurative, diffuse, moderate, with mild epithelial hyperplasia, breed unspecified, porcine.

Colon: Intraluminal protozoa. Etiology: consistent with Balantidium coli.

CONFERENCE NOTE The conferees agreed with the contributor's comments except for the statement regarding marked goblet cell depletion. It was generally agreed that goblet cells may be mildly reduced in number.

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REFERENCES

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1979.

MICROSLIDE 117

HISTORY Four, 2-week-old mixed breed puppies with acute neurologic signs, were presented to the submitting veterinarian. The primary clinical sign was "seizuring". The veterinarian euthanized three of the puppies and, sent the fourth, less affected, puppy home.

GROSS PATHOLOGY The carcasses were grossly unremarkable except for the brains, which exhibited severe generalized malacia.

LABORATORY RESULTS Virus isolation yielded a Bunyavirus belonging to the California encephalitis group (most likely La Crosse virus).

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Severe necrotizing panencephalitis, La Crosse virus.

The family Bunyaviridae consists of arthropod-borne animal viruses, or arboviruses, which can infect both vertebrate and invertebrate hosts. La Crosse (LAC) virus is a Bunyavirus (genus) of the California serogroup and was first isolated from the brain of a fatal case of encephalitis in a 4 year-old girl in La Crosse, Wisconsin in 1960. LAC causes more childhood illness than any other arbovirus in the United States. Most reported cases of LAC encephalitis are from the Midwest, with sporadic reports from other areas of the country. In the Midwestern United States, the virus is maintained in nature primarily in chipmunks, tree squirrels and the mosquito, Aedes triseriatus, which also serves as the primary vector of the virus. Clinically, LAC causes fever and headache followed by vomiting in 24 to 48 hours. Fifty percent of cases experience seizures and recovery is usually within 5 to 7 days. Mortality is less than 0.5%.

In 1984, LAC was reported to be endemic in southeastern Georgia. This observation was based on the occurrence of eight serologically confirmed cases within the southeastern quadrant of the state. These cases involved children ranging in age from 3 months to 10 years old and had onset dates from May 1 to August 30.

The tissue sections submitted to AFIP are from the brain of one of three puppies from which a Bunyavirus in the California serogroup was isolated. This virus is believed to be LAC. These puppies were from southeast Georgia, and similar to previously reported cases in Georgia children, experienced onset of CNS signs in the month of May. There is serologic evidence that infection with California serogroup viruses is common in domestic animals. Contrary to previous information stating the virus to be nonpathogenic in animals, there have been recent reports of clinical disease in domestic species in which the animals exhibited significant serologic responses to California serogroup viruses. LAC encephalitis has been produced experimentally in puppies; but, the virus has never before been isolated from a case of naturally occurring disease in an animal other than man.

AFIP DIAGNOSIS Cerebrum: Meningoencephalitis, necrotizing, nonsuppurative, diffuse, severe, breed unspecified, canine.

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MICROSLIDE 118

HISTORY The death of this 4-year-old cow was caused by acute heart failure.

GROSS PATHOLOGY There were multiple papillomatous growths on the skin of the four teats. The udder was not involved.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Multifocal hyperplastic dermatitis with hydropic degeneration of the epidermis and the presence of intracytoplasmic inclusion bodies. Cause: Poxvirus.

The lesions are characterized by a marked hyperplasia, hypergranulosis and orthokeratotic hyperkeratosis of the epidermis. In the hyperplastic epidermis there are focal areas of hydropic degeneration which contain large intracytoplasmic eosinophilic inclusion bodies. There are mild mononuclear cell infiltrates in the superficial dermis. Grossly, these lesions were diagnosed as papillomas of the teats. Histologically, the lesions are strongly suggestive of a poxvirus infection and very similar to those of papular stomatitis.

AFIP DIAGNOSIS Teat, epidermis: Hyperplasia, multifocal, moderate with ballooning degeneration, orthokeratotic hyperkeratosis and large intracytoplasmic eosinophilic inclusion bodies, breed unspecified, bovine.

CONFERENCE NOTE Conference participants felt that it was difficult to identify the specific poxvirus responsible for this lesion. The large intracytoplasmic inclusions were unusual for common ortho- and parapoxviruses that affect cattle. Some participants remarked that these lesions were reminiscent of molluscum contagiosum; however, this condition has not been reported in cattle. Although the proliferative nature of this lesion is suggestive of bovine papular stomatitis, the large size of the intracytoplasmic inclusions are unusual for this disease. Bovine pustular stomatitis causes lesions primarily around the muzzle and mouth. Occasionally lesions are found on the teats, esophagus, posterior oral cavity and tail (rat tail syndrome).

When evaluating pox lesions in cattle one must be aware of the other poxviruses that affect the udder and teat of cattle. These viruses have a predilection for the udder and teat and cause primarily vesicles and pustules. These poxviruses are cowpox, vaccinia, and pseudocowpox.

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MICROSLIDES 119 and 120

HISTORY This tissue is from an adult male German shorthair pointer. The dog had been fed salmon infected with Nanophyetus salmincola and Neorickettsia helmintheca as part of an experimental protocol. Albon was administered beginning with a dose of 1.63 grams on the day the animal was infected and then daily in decreasing doses over an 8 day period. Three weeks after the last dose of Albon, the dog developed an acute onset of severe bloody diarrhea, dehydration and depression. IV fluid therapy was administered and chloramphenicol given. The dog's condition deteriorated and 2 days after the onset of clinical signs, he was euthanized.

GROSS PATHOLOGY There was marked generalized lymphadenopathy including the tonsils. The lymph nodes were edematous and many of the mesenteric nodes contained focal areas of hemorrhage. The lymphoid aggregates in the small intestine were prominent. The mucosa of the proximal colon was hyperemic. The intestinal contents were green and watery.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Small intestine: Enteritis, histiocytic and plasmacytic, necrotizing, diffuse, severe, subacute. Intestinal trematodiasis (morphologically compatible with Nanophyetus salmincola.

Colon: Colitis, histiocytic, necrotizing, diffuse, severe, subacute.

Lymph nodes: RE hyperplasia, severe, diffuse. Lymphoid depletion, moderate to severe. Hemorrhage, multifocal, mild to severe depending on node examined.

Wolbach's Giemsa stain was used to attempt to identify Neorickettsia helmintheca organisms in macrophages in the gut, lymph nodes and spleen. No lesions were found in the brain. The incubation period for salmon disease in dogs averages 5-7 days but can be as long as 2-4 weeks. The manner in which the rickettsial organisms leave the trematode vector is not clear. The organisms are usually found in the reticuloendothelial cells of the lymph nodes, spleen,

tonsils and intestinal lymphoid follicles. The snail Oxytrema silicula, which is indigenous to the Northern California, Oregon and Washington coastal regions, is the first intermediate host for the trematode.

AFIP DIAGNOSIS Small intestine: Enteritis, necrotizing, diffuse, moderate, with villous atrophy, blunting and fusion, and crypt hyperplasia, German shorthair pointer, canine.

Lymph node: Lymphoid depletion, hemorrhage and edema, diffuse, severe, with mild sinus histiocytosis.

CONFERENCE NOTE Most conference participants commented on the paucity of lymphocytes and plasma cells in the lamina propria of the small intestine; several thought there was an overabundance of histiocytes. Based on the H&E section, canine parvovirus was considered the primary differential.

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MICROSLIDE 121

HISTORY These tissues are from a yearling Chinook salmon from a group of 190,000 fish that had a low grade mortality of 0.1% per week. The average weight of these fish was 430 grams; the rest of the group averaged 1.2 kilograms.

GROSS PATHOLOGY There was chronic low grade fibrinous peritonitis. The peritoneum was hyperemic with petechial hemorrhages. The kidneys were diffusely swollen and gray. The liver was mildly swollen and pale. The spleen was meaty, congested and enlarged. The heart had a slight fibrinous cast.

LABORATORY RESULTS Renibacterium imoninarum was isolated on culture.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Renibacterium salmoninarium.

The lesions vary between sections, depending on severity and the length of illness.

Kidney: On all sections there are multiple or single granulomas with caseonecrotic cores surrounded by a variable thick wall of macrophages, neutrophils and fibroblasts. There is mild diffuse necrosis of renal hematopoietic tissue. On some sections the nephron is relatively spared, whereas in others the nephrons are caught up in the expanding granulomas and are destroyed. The glomeruli focally reveal eosinophilic/hyaline thickening of the membranes. Gram positive diplobacilli are present within the necrotic debris and within macrophages.

Liver: There are multifocal to coalescing granulomas throughout the hepatic parenchyma with variable loss of hepatocytes characterized by pyknosis and karyorrhexis. Some central veins have a mixed inflammatory infiltrate within the media, or complete necrosis of the wall, and thrombosis with obliteration of the vessel.

Heart: The fish heart is composed of an outer compact layer and inner spongy layer. The epicardium contains a thin meshwork of fibrin impregnated with neutrophils and macrophages. Focal areas of necrosis are also apparent and some slides contain melanomacrophages within the inflammatory infiltrate. There are multifocal areas of myocardial necrosis within both the spongy and compact layers with a mixed inflammatory infiltrate of neutrophils and macrophages, gram-positive diplobacilli are present in macrophages.

Spleen: The lesions are quite variable and this mainly reflects the degree of necrosis of the parenchyma - most ellipsoids are intact and there are focal pyogranulomatous aggregates, often with a caseonecrotic core.

Gill: Several of the secondary lamellae contain thrombosed vessels; one section reveals gram-positive diplococci within pillar cells. Chloride cells at the base of the secondary lamellae are prominent. The blood present between lamellae and focal aneurysms are artifacts.

Mesentery: The majority of large vessels are partly or completely thrombosed. There are intramural large focal granulomas which contain macrophages and bacteria. Focal small loci of necrosis are present within the pancreas and also in mesenteric tissue.

This organism is the cause of the major bacterial disease of cultured and wild Pacific salmon. The disease is typically chronic with periodic epizootics associated with stress. The systemic disease pattern is associated with the portal system and phagocytes of the renal hematopoietic tissue, splenic hematopoietic tissue, and the phagocytic endothelial cells of the heart/liver, etc. Pillar cells are also phagocytic and can be involved with the disease. In the severe chronic forms of the disease any organ may be involved with granuloma formation even the skin (subepidermal) and retrobulbar tissue with exophthalmus. The vascular lesions are associated with chronicity.

AFIP DIAGNOSIS

Liver: Hepatitis, necrogranulomatous, multifocal, severe, with necrotizing phlebitis and phagocytized coccobacilli, chinook salmon, piscine.

Heart: Pancarditis, necrogranulomatous, multifocal and coalescing, mild, with phagocytized coccobacilli.

Spleen: Splenitis, necrogranulomatous, diffuse, severe, with phagocytized coccobacilli.

Kidney: Nephritis, necrogranulomatous, diffuse, severe with phagocytized coccobacilli.

Gill, pillar cells: Intracellular coccobacilli.

CONFERENCE NOTE Aneurysm (telangiectasia) of gill lamellar capillaries is an acute response to branchial injury that results from breakdown of vascular integrity and pooling of blood due to rupture of pillar cells. The thrombosis of the secondary lamellae of the gills and mesentery, as described by the contributor, was not evident in all sections examined. Conference participants felt that the lesions observed in this case are compatible with the disease condition called bacterial kidney disease caused by Renibacterium salmoninarium. Bacterial kidney disease is caused by a gram-positive nonmotile diplobacillus. This disease affects all salmonids. The disease follows a slow course with clinical signs not present until the fish is well grown. The fish may exhibit exophthalmos, skin darkening, hemorrhage at the base of the fins, and cutaneous vesicles and ulcers. As in this case, numerous granulomas develop in many organs. Contraction of muscles is occasionally observed. Transmission is believed to be via direct contact with contaminated fish. It is felt that the organism may enter through the epidermis or fecal-orally and then become a systemic disease.

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MICROSLIDE 122

HISTORY This 6 ½-year-old, male, Bernese mountain dog has been under treatment for multiple growths in the lungs and liver.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Malignant histiocytosis, lung, Bernese mountain dog.

The pulmonary tissue is completely effaced by sheets of morphologically atypical mononuclear histiocytes and multinucleate giant cells interspersed with delicate fibrovascular stroma and scattered areas of coagulation necrosis. The mononuclear histiocytes are pleomorphic with many bizarre mitotic figures. Cytophagocytosis of leukocytes and occasional erythrocytes by neoplastic histiocytes and multinucleate giant cells is not uncommon. Occasional neoplastic histiocytes are noted in dilated lymphatic vessels. A similar lesion was also seen in the liver. PAS and acid-fast stains of the lung did not demonstrate causative organisms. The diagnosis of malignant histiocytosis, in this case, is based on light microscopic examination (atypical histiocytes and multinucleate giant cells with bizarre mitotic figures), lesion distribution (lung and liver involvement), and clinical history (adult male Bernese mountain dog).

Malignant histiocytosis of dogs is a rapidly progressive fatal disease, characterized by systemic neoplastic proliferation of morphologically atypical histiocytes and their precursors. The disease is seen most often in the Bernese mountain dog. Malignant histiocytosis of Bernese mountain dogs usually affects middle-aged males and has a high incidence of pulmonary involvement. It

appears to be a sex-linked recessive trait.

The definitive diagnosis of malignant histiocytosis relies on confirmation of histiocytic origin of tumor cells by transmission electron microscopy and immunocytochemical staining for lysozyme (and/or alpha-1-antitrypsin) in the tumor cells.

Differential diagnosis for malignant histiocytosis in Bernese mountain dogs include systemic histiocytosis, disseminated granulomatous disease, histiocytic lymphoma, myeloproliferative disorder, large cell anaplastic pulmonary carcinoma, and lymphomatoid granulomatosis.

AFIP DIAGNOSIS Lung (per contributor): Malignant histiocytosis, Bernese mountain dog, canine.

CONFERENCE NOTE An immunohistochemical procedure for lysozyme was performed, about 40 percent of the neoplastic cells were decorated, including giant cells, atypical cells, and cells with mitoses. Most, if not all, conference participants found it impossible to identify the affected tissue in this case.

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MICROSLIDE 123

HISTORY This young adult, female, cynomolgus monkey (Macaca fascicularis) was wild caught in Southeast Asia or the Philippines. She was a control animal on a 14 week study and in apparently good health when euthanatized.

GROSS PATHOLOGY Liver: Tan areas in all lobes of both surfaces, multiple, pinpoint.

Cecum and colon: Raised areas in wall, gray, multiple, 3x3 mm to 5x5 mm.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Liver: Granulomatous inflammation, periportal, moderately severe, with numerous multinucleated giant cells, some containing parasitic ova consistent with Schistosoma probably hematobium.

Mesenteric lymph nodes: Granulomatous inflammation, moderately severe with multinucleated giant cells, some containing parasite ova consistent with Schistosoma sp.

Some sections may also contain sections of Oesophagostomum sp. with embryonated eggs.

AFIP DIAGNOSIS Liver: Hepatitis, portal, granulomatous and eosinophilic, multifocal, moderate, with schistosome eggs, acute eosinophilic vasculitis and thrombosis, cynomolgus monkey, primate.

Lymph node: Lymphadenitis, granulomatous and eosinophilic, multifocal, moderate, with schistosome eggs, diffuse lymphoid hyperplasia, and acute eosinophilic vasculitis.

Mesenteric arteries and veins: Vasculitis, eosinophilic, transmural, mild.

CONFERENCE NOTE Conference participants felt that the eggs were compatible with Schistosoma species. It was felt that this parasite probably was not S. hematobium because the eggs are less than 60 microns in length and do not contain terminal spines. In addition, the monkey is from the wrong geographical area (S. hematobium is found in Africa). S. japonicum is considered the most likely diagnosis.

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MICROSLIDE 124, 35 MM SLIDE 28

HISTORY This adult female frog (Rana pipiens) was one of 40 of 48 frogs that died over a one wee' period. The frogs were received one week prior and were housed in plastic tubs with non-circulating water.

GROSS PATHOLOGY The skin of the ventral abdomen and ventro-medial surface of the thighs had multifocal, nonulcerated, discrete, irregularly-shaped red loci up to 10 mm at the widest diameter. An occasional digit and the tip of the nares contained a small amount of bright-red firm tissue.

LABORATORY RESULTS Blood cultures yielded Aeromonas hydrophila.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT A definitive diagnosis of redleg septicemia is made by isolation of the organism from blood culture.

Aeromonas hydrophila is a common cause of morbidity and mortality in cold-blooded animals. It has been reported to cause ulcerative stomatitis and pneumonia in snakes, death in fishes,

turtles and alligators and "red leg" disease in frogs. Redleg is a commonly used term for septicemia in frogs and describes the erythematous discoloration on the legs and abdomen. Although Aeromonas hydrophila is the most frequently isolated organism from frogs with redleg, other gram-negative bacteria can produce the disease. Clinical signs of septicemia in frogs include anorexia, weight loss, lethargy and cutaneous erythema. Because of its common occurrence and high mortality, redleg is the first disease to be considered in clinically ill frogs. Aeromonas hydrophila can be isolated from enteric contents of healthy frogs and is an ubiquitous organism in fresh and brackish water. Redleg is a peracute to chronic disease usually associated with frogs housed under stressful conditions such as overcrowding, water temperature extremes and poor water quality. In humans, Aeromonas hydrophila is considered an opportunistic pathogen that causes septicemia in the immunocompromised patient; however, recent evidence indicates that Aeromonas hydrophila may be an enteropathogen in the normal host.

AFIP DIAGNOSIS

Kidney: Glomerular thrombosis and necrosis, multifocal, moderate to severe, with tubular ectasia and degeneration, frog, Rang pipiens, amphibian.

Kidney: Crystals, anisotropic, intratubular, multifocal.

Liver, veins: Fibrin thrombi, multifocal, moderate, with multifocal hepatocellular degeneration.

CONFERENCE NOTE The crystals observed in the renal tubules are consistent with oxalates. In the experience of the moderator, oxalate crystals are a common incidental finding in the renal tubules of frogs; on a few occasions he has seen nephrosis secondary to oxalate crystalluria in frogs feed diets high in oxalates while in their larval (tadpole) stage.

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MICROSLIDES 125 and 126

HISTORY This raccoon was found dead on the side of the road apparently killed by a car. The brain was submitted for rabies examination the following day.

GROSS PATHOLOGY Necropsy revealed a well nourished young raccoon with multiple traumatic injuries consisting of fractured ribs and hemothorax.

LABORATORY RESULTS The brain was negative for rabies on fluorescent antibody impression smears.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Severe multifocal acute ulcerative glossitis with intranuclear inclusion bodies (INI). Moderate capillariasis and minimal sarcocystosis.

Intestine (small & large) a few cross sections of helminths in lumen. Numerous banana-shaped protozoa present in tips of villi, not associated with inflammation, compatible with sarcocystis.

Other associated lesions not on distributed sections:

Moderate, multifocal acute necrotizing adrenalitis destroying approximately 20-30% of adrenal with many intranuclear inclusion (INI) bodies in adjacent adrenal cells of both large basophilic and small eosinophilic Cowdry type A and B, respectively.

Severe multifocal and diffuse suppurative bronchopneumonia and necrotizing bronchiolitis with INI and numerous bacteria.

Severe bilateral multifocal acute ulcerative pyelitis and papillary necrosis extending into lower excretory ducts in one kidney. Much necrotic debris in pelvic cavities, many INI.

Severe lymphoid depletion and multifocal parasitic granulomas in mesenteric nodes.

Bilateral minimal multifocal interstitial lymphocytic nephritis.

Etiologic diagnosis: Disseminated Herpes, bacterial pneumonia, lingual capillariasis and sarcocystosis, and intestinal sarcocystosis.

AFIP DIAGNOSIS

Tongue: Glossitis, ulcerative, acute, multifocal, moderate to severe, with eosinophilic intranuclear inclusions, raccoon, procyonid.

Tongue: Nematodes, adults and eggs, intraepithelial, multifocal. Etiology: Capillaria sp.

Tongue, skeletal muscle: Sarcocysts, multiple.

Small intestine, lamina propria: Sporulated coccidian oocysts, diffuse. Etiology: Sarcocystis sp.

Small intestine, lumen: Adult cestode.

CONFERENCE NOTE Several conference participants commented that they observed intracytoplasmic inclusions in addition to intranuclear inclusions in affected glossal epithelium; most participants, however, observed intranuclear inclusions only. (The intracytoplasmic structures may have been keratohyalin, degenerative organelles, or transposed erythrocytes.)

The life cycle of Sarcocystis sp. was discussed in depth. Sarcocystis is one of few coccidians which sporulate in the lamina propria of the intestine. The sarcocysts in the tongue are a different species than that seen in the intestine; Sarcocystis has an obligate two host life cycle.

Not all sections contained capillarids in the glossal epithelium or a cestode in the lumen of the small intestine.

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MICROSLIDE 127

HISTORY This tissue is from a 15-month-old beagle dog. At the age of 8 months this dog was excluded from drug safety studies due to a persistent eosinophilia ($3.2 - 4.2\ 10^3/mm^3$).

GROSS PATHOLOGY The accessory lobe of the right lung was enlarged (12xl3 cm), firm and pale yellow. Cut sections revealed coalescing multinodular pale tissue. The tracheobronchial lymph nodes were enlarged (5x3 cm), firm and white. Similar pale tissue was present focally in the sternal lymph node.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Lymphomatoid granulomatosis, lung, dog.

Microscopically the tumor is composed of sheets of atypical pleomorphic lymphoid cells infiltrated by eosinophils, plasmacytes and occasional Mott cells. A few binucleated cells are seen. Highly distorted blood vessels are infiltrated by tumor cells. There are occasional epithelial remnants of airways and necrotic loci up to 3 mm in diameter. Tumor cells do not stain for lysozyme, alpha-1-antitrypsin or S-100 protein. Ultrastructurally tumor cells lack intercellular junctional complexes and pericellular basement membrane material. A limited amount of cellular interdigitation is observed. The tumor cells contain a few lysosomes but show no evidence of phagocytosis.

Lymphomatoid granulomatosis is a rare pulmonary neoplasm with intrathoracic lymph node involvement that affects young dogs. It is hypothesized that it might represent a form of lymphoma. In humans the lungs are predominantly involved with skin, brain and kidneys also frequently affected. Recent evidence suggests that lymphomatoid granulomatosis is a peripheral or post-thymic T-cell lymphoma as are three other disorders - midline granuloma, angioimmunoblastic lymphadenopathy and histiocytic medullary reticulosis.

Canine lymphomatoid granulomatosis must be differentiated primarily from malignant histiocytosis that is reported in Bernese mountain dogs, pulmonary nodular eosinophilic granulomatosis that is related to heartworm disease, and the giant cell variant of large cell anaplastic carcinoma of the lung. Malignant histiocytosis is composed histologically of an infiltrate of bizarre, highly phagocytic mononuclear and multinucleated cells that have a positive immunoreactivity for lysozyme and alpha-1-antitrypsin. Pulmonary nodular eosinophilic granulomatosis is characterized by extensive eosinophilic granuloma formation that does not primarily involve blood vessels. The giant cell variant of large cell anaplastic carcinoma of the lung contains poorly differentiated cells with pleomorphic multinucleated giant cells that may form cords and some beginnings of glandular formation.

AFIP DIAGNOSIS Lung: Lymphomatoid granulomatosis, beagle, canine.

CONFERENCE NOTE A review of the literature indicates that this case is histomorphologically consistent with what has been diagnosed as lymphomatoid granulomatosis in dogs.

The staff of the Department of Hematolymphatic Pathology, reviewed the case and diagnosed malignant lymphoma, large cell type. In lymphomatoid granulomatosis in humans, the infiltrate is typically more pleocellular than as seen in this case where large lymphoid cells predominated. Regardless of the nomenclature (lymphoma vs lymphomatoid granulomatosis), they would have given a human patient the same prognosis and recommended the same treatment. In humans, 10-13% of the cases of LG progress to malignant lymphoma.

Neoplastic cells were negative for lysozyme using immunohistochemical techniques. An additional differential diagnosis discussed at the conference was mast cell tumor. (Giemsa stained sections did not demonstrate metachromatic granules in neoplastic cells.)

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MICROSLIDE 128

HISTORY This yearling steer was in a group of 120 animals wintered in direct contact with sheep. This animal was presented with bilateral panophthalmitis, coronitis, elevated body temperature (110°F), depression and mild lymphadenopathy. He had erosions of the oral mucosa, muzzle and interdigital areas. The steer developed terminal convulsions and was subsequently euthanatized. One other animal in this group had died previously with similar clinical signs.

GROSS PATHOLOGY Significant gross lesions include severe multifocal erosions and ecchymotic hemorrhages of the muzzle, oral and esophageal mucosa, bilateral keratoconjunctivitis, severe erosive interdigital dermatitis and coronitis.

LABORATORY RESULTS Virus isolation attempts in cell culture systems were negative. Indirect fluorescent antibody examination for serum antibody for malignant catarrhal fever (MCF) were considered positive at a titer of 1:100. Virus neutralization for MCF was negative at a titer of 1:4. This animal was negative for bluetongue virus (BT) by agar gel immunodiffusion and was immunoreactive to bovine virus diarrhea (BVD) at a titer of 1:320 by virus neutralization.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT Vasculitis and perivasculitis, necrotizing, lymphocytic, histiocytic, neutrophilic, extensive, kidney.

Nephritis, interstitial, multifocal, lymphocytic, mild.

Dermatitis, ulcerative, necrotizing and vesiculating, lymphocytic, histiocytic, neutrophilic, with folliculitis, adenitis, myositis, and vasculitis, severe. Etiology: African form of MCF: Cell associated gammaherpesvirus (bovine herpesvirus 3). European/American form of MCF: Virus not yet identified.

We feel that the clinical, gross and histopathological findings are compatible with a diagnosis of malignant catarrhal fever. The gastrointestinal tract and peripheral lymph nodes were minimally involved in this case. Other tissues with histologic evidence of vasculitis and perivasculitis included brain, myocardium, spleen, lymph node and pharynx.

AFIP DIAGNOSIS Haired skin: Dermatitis, necrotizing, lymphohistiocytic, diffuse, severe, with vesicles, epidermal hyperplasia, and lymphohistiocytic arteritis, folliculitis, and myositis, Hereford, bovine.

Kidney: Arteritis, lymphohistiocytic, segmental to circumferential and occlusive, multifocal, moderate to severe, with multifocal mild subacute interstitial nephritis.

CONFERENCE NOTE Predominantly lymphocytic/lymphoblastic cellular infiltrates coupled with vasculitis and perivasculitis is generally considered diagnostic of MCF.

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MICROSLIDE 129

HISTORY This 10-year-old Holstein cow was killed following a two day history of anorexia, fever, nasal discharge, and seizures.

GROSS PATHOLOGY There is bilateral scleral injection and corneal opacity. Marked reddening of the nasal mucosa is also present.

CONTRIBUTOR'S DIAGNOSIS AND COMMENT All vascularized ocular tissues: Lymphoplasmacytic vasculitis.

Cornea: Keratitis and corneal edema.

Lymphoplasmacytic uveitis.

Malignant catarrhal fever is a pansystemic disease of cloven-hooved animals characterized by vasculopathy and lymphoid proliferation. The etiologic agent of the African form of MCF is a herpes virus carried by the wildebeest. The cause of the sheep-associated form of MCF is unknown.

Ophthalmic lesions are often present in cases of malignant catarrhal fever. Active lymphoid proliferation, indicated by the presence of mitotic figures in lymphoblasts, differentiates the ocular lesions of MCF from other bovine ocular diseases. In contrast, ocular lymphoblastic responses are not a feature of bovine virus diarrhea-mucosal disease, thromboembolic meningoencephalitis, infectious bovine rhinotracheitis, or infectious keratoconjunctivitis.

AFIP DIAGNOSIS Eye: Panophthalmitis, lymphocytic, multifocal, mild to moderate, with lymphocytic vasculitis, Holstein-Friesian, bovine.

CONFERENCE NOTE Panophthalmitis was diagnosed because inflammation affected all layers of the eye as well as the anterior and posterior chambers.

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