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Department of Veterinary Pathology
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CONFERENCE 18
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Philadelphia, PA

CASE I – 98-443 (AFIP 2681375)

Signalment: 10 year-old female spayed American Eskimo dog.

History: 3-4 month history of inflamed, ulcerated, and hyperkeratotic foot pads +/- oral mucocutaneous lesions. The dog was treated with glucocorticoids, which exacerbated lesions; then with antibiotics, dietary change, and antihistamines

- 3-4 week history of inappetance; treated with Baytril, Dicural, and Reglan
- One week history of polyuria/polydypsia, vomiting, icterus, and hyperglycemia with glucosuria and ketoacidosis; treated with insulin, Reglan, and centrine with no improvement
- Upon referral, a tentative diagnosis of hepatocutaneous syndrome was made and the owners elected euthanasia

Gross Pathology: The footpads, elbows, vulva, and lips were variably ulcerated and hyperkeratotic. The animal was severely icteric. The liver was firm, yellow-brown with multifocal to coalescing nodules which replaced the entire normal parenchyma. (See gross photo)

Laboratory Results:

Test Result	7-2-98	9-17-98	9-23-98	
ALP	1724	NA	> 2400	NA = not available
ALT	158	142	45	
HCT	30	38.3	24.7	
WBC	16,500	22,900	28,500	
PMN	14,500	18,300	27,300	
GLU	NA	> 700	221 (on insulin)	
ALB	NA	3.1	2.4	

Contributor's Morphologic Diagnoses: 1. Skin, footpad: Superficial dermatitis with basal epidermal hyperplasia, epidermal pallor, and parakeratotic hyperkeratosis.
2. Liver: Severe lobular collapse with nodular regeneration, vacuolar hepatopathy, mild portal fibrosis and bile duct proliferation, and marked canicular bile stasis.

Contributor's Comment: Hepatocutaneous syndrome, also known as superficial necrolytic dermatitis, necrolytic migratory erythema, or diabetic dermatosis is the association of a specific skin lesion (hyperplastic basal epithelium, a zone of epidermal pallor, and parakeratotic hyperkeratosis) with severe liver disease. The liver lesion is typically a severe vacuolar hepatopathy with parenchymal collapse and nodular regeneration which mimics cirrhosis.

In humans the disease is most often associated with glucagon secreting tumors of the endocrine pancreas; however, some human cases have cirrhosis or chronic pancreatic disease with normal glucagon levels. In dogs, most cases are associated with liver disease with normal glucagon levels, but rare cases of glucagon secreting tumors have been reported. The pathogenesis is unknown.

AFIP Diagnoses: 1. Skin, footpad: Hyperkeratosis, parakeratotic, diffuse, severe, with marked basal epidermal hyperplasia, stratum spinosum edema and degeneration, mild subacute dermatitis, and focal ulcer with a serocellular crust, American Eskimo, canine.
2. Liver: Hepatocellular loss with stromal collapse, diffuse, severe, with nodular regeneration, multifocal vacuolar degeneration, mild bridging fibrosis, and biliary hyperplasia.

Conference Comment: Superficial necrolytic dermatitis (SND) is considered a paraneoplastic syndrome that more commonly occurs in association with hepatopathy than with glucagon-secreting neoplasia, giving rise to the familiar name of hepatocutaneous syndrome.⁵

Often the main presenting complaints of glucanoma-associated SND are progressive skin lesions of three weeks to many months, with concurrent lethargy and inappetance. The main dermatologic findings include erosions and ulcerations, with alopecia, exudation and adherent crusts on the feet, pressure points such as the elbows and hocks, flank, perineal area, muzzle, facial mucocutaneous junctions and/or oral cavity. Hyperkeratosis and fissuring of foot pads occur in all animals. Many dogs will also have hyperglucagonemia, hyperglycemia, and marked hypoanimoacidemia involving many amino acids.⁵

The histopathological findings of SND are distinctive and can be strongly suggestive of the disease. The epidermis has a “red, white, and blue” appearance on H&E. Parakeratotic hyperkeratosis and crusting create the upper eosinophilic layer. Edema and necrosis of keratinocytes within the stratum spinosum make up the middle “white” layer. Hyperplasia of the stratum basale gives rise to the deep basophilic layer. In addition, there may be secondary clefting in the devitalized middle layer, leading to ulceration and secondary inflammation. A mixed inflammatory infiltrate may be present in the superficial dermis.⁵

Although the exact pathogenesis is unknown, there are several current theories. The first is that glucagon results in sustained gluconeogenesis and is involved in the catabolism of amino acids, and chronic elevation of the hormone, as seen in glucagonomas, may result in hypoaminoacidemia, leading to epidermal protein depletion and subsequent keratinocyte necrosis. This is supported by cases of glucagonoma-associated SND that rapidly resolve following surgical resection of the tumor. However, there are also documented cases of SND in which glucagon concentrations are within normal limits.⁵ There may be other causes of hypoaminoacidemia. In one report, dogs with nonglucagonoma-associated SND, also had significantly lower amino acids concentrations than the control dogs or dogs with acute or chronic hepatitis.⁶ Another theory involves deficiencies in zinc or fatty acids. However, there has been little response to replacement therapy. Finally, hepatic impairment has also been implicated as a possible mechanism. Many, although not all canine patients with nonglucagonoma-associated SND have elevated serum glucagon. It may be that impaired hepatic function leads to decreased metabolism of glucagon, resulting in increased serum levels.⁵

Although SND is an uncommon skin disorder, it is an important diagnosis because it is one of the relatively few skin conditions from which one can diagnose a life-threatening disease, with confidence, from a skin biopsy.

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CASE II – 03-56361 (AFIP 2937321)

Signalment: 1 year-old male Standardbred horse.

History: This colt was purchased at an auction sale 6 weeks prior to presentation. Nasal discharge, lethargy, and limb edema had been evident for the past 4 weeks and the colt had been treated unsuccessfully with 2 courses of antibiotics. Physical examination revealed peripheral lymphadenopathy, severe pneumonia with suspected microabscesses in lung, and ventral edema.

Gross Pathology: Severe generalized lymphadenopathy involved peripheral and many internal lymph nodes, including submandibular, internal iliac, and mesenteric nodes. The enlarged nodes were diffusely white and nodular on sectioning, with occasional areas of necrosis. Left lung contained 2 discrete, 2 cm white foci similar in texture and appearance to the affected lymph nodes. Scrotal skin contained numerous 3-4 mm pale, poorly pigmented, non-haired papules that were deemed an incidental finding.

Contributor's Morphologic Diagnoses: 1. Multicentric lymphosarcoma
2. Equine molluscum contagiosum

Contributor's Comment: Slides demonstrate lesions of equine molluscum contagiosum only. Although lymphosarcoma was the most significant disease process in relation to this horse's clinical demise, lesions of equine molluscum contagiosum presented an additional concurrent and interesting disease process.

Equine molluscum contagiosum (MC) is an uncommon, mildly contagious, cutaneous poxviral disease generally involving skin of the face, neck, chest, trunk, limbs, and genitalia.¹ The virus is transmitted by contact with either an infected individual or contaminated fomites. Multiple 2-8 mm papules develop in affected skin, and although these lesions are benign, they are refractory to therapy and persist for months to years. The lesions in this horse were typical of equine MC and consisted of focal, discrete lobulated areas of epidermal hyperplasia bulging into the underlying dermis. Keratinocytes of the stratum spinosum were swollen and contained large intracytoplasmic inclusions ('molluscum bodies'). In some

sections, there is an impression of a central pore in the hyperplastic lobules, through which degenerate keratinocytes are exfoliated. Electron micrographs of the cutaneous lesions demonstrated numerous poxviral inclusions within epithelial cells. Although MC has been diagnosed in clinically normal horses, several literature reports describe MC in horses with other concurrent immunocompromised conditions, such as in this case.^{1,2} In humans, lesions of MC are generally more severe and widespread in patients with compromised cell-mediated immune function.³

Equine MC bears clinical and histologic resemblance to the human skin disease of the same name, and recent *in situ* hybridization experiments have shown that the viruses causing lesions in these two species have significant nucleic acid homology, leading to the hypothesis that equine MC is an anthroozoonosis.³ Equine MC has been identified in horses worldwide and clinical lesions are similar to those of Uasin Gishu disease, which has been identified in horses only in Kenya.^{2,3} Although the Uasin Gishu virus can be cultivated, the virus associated with equine MC has yet to be isolated. The poxvirus of equine MC is currently classified in the subfamily Chordopoxvirinae, genus Molluscipoxvirus.

AFIP Diagnosis: Haired skin: Hyperplasia, epidermal, focal, marked, with large, eosinophilic intracytoplasmic inclusion bodies (molluscum bodies), Standardbred, equine.

Conference Comment: Equine molluscum contagiosum (MC) is caused by a molluscipoxvirus that is either identical with, or very closely related to, its human equivalent, molluscum contagiosum virus (MCV). MCV is a poorly characterized pox virus which causes a human skin disease characterized by benign but persistent papular lesions with a central opening or umbilicus. Lesions are commonly found on the face, trunk, lower limbs, and anogenital regions. Humans with immunodeficiency have a more severe, disseminated form of the disease. Molluscum contagiosum has been reported in horses, chimpanzees, and kangaroos. In all of the cases, lesions clinically and histologically were very similar to those seen in humans. MC may represent an anthroozoonosis, wherein disease is transmitted from humans to animals.³

In horses, affected animals commonly have hundreds of lesions, especially on the chest, shoulders, neck, limbs, and external genitalia. Lesions on haired skin consist of papules that are initially tufted, but usually become alopecic and covered with a powdery crust or scale. Papules in glabrous skin may be smooth, shiny, hypopigmented, and umbilicated, or hyperkeratotic and hyperpigmented. Lesions are usually nonpruritic and nonpainful. Histologically, lesions are well-demarcated

and characterized by epidermal hyperplasia and papillomatosis. Keratinocytes above the stratum basale become swollen and contain ovoid, eosinophilic, floccular intracytoplasmic inclusion bodies (molluscum bodies). These inclusions increase in size and density as the keratinocytes move toward the skin surface. Molluscum bodies exfoliate through a central pore that forms in the stratum corneum and enlarges into a central crater. Usually there is no dermal inflammatory reaction. Ultrastructural examination reveals mature virions that are brick-shaped and approximately 150 x 300 nm, with a biconcave nucleoid and two lateral bodies, typical of poxviral inclusions.¹

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CASE III – 04-2262 (AFIP 2941563)

Signalment: 10 year-old, female, mixed breed dog (*Canis familiaris*).

History: The dog was being treated for lymphoma and immune-mediated thrombocytopenia with a standard chemotherapy protocol (cyclophosphamide, doxorubicin, vincristine, prednisone). Small nodular dermal lesions developed after 11 treatments with progression to severe exudative dermatitis following the last treatment.

Gross Pathology: Variably sized irregular erythematous exudative skin lesions were randomly distributed on the trunk.

Contributor's Morphologic Diagnosis: Severe generalized necrotizing dermatitis and vasculitis with intralesional protozoal organisms.

Contributor's Comment: The slides vary somewhat in the degree of necrosis and ulceration. The lesion is characterized by extensive epidermal ulceration and necrosis that includes the follicular epithelium and adnexal structures. Necrosis extends from the epithelium to the superficial hypodermis with edema, hemorrhage and an inflammatory infiltrate of neutrophils, histiocytes, small lymphocytes and plasma cells that is diffuse to perivascular. The perivascular inflammatory infiltrates extend into the hypodermis. Small 1-2 μm oval organisms were found free in the necrotic tissue debris, within macrophages and, occasionally, within endothelial cells and epithelial cells of the epidermis, follicles and adnexal glands. Immunohistochemistry was performed and the organisms reacted strongly with a Polyclonal rabbit anti-*Toxoplasma gondii* antibody and did not react with a Polyclonal rabbit anti-*Neospora caninum* antibody.

Necrotizing dermatitis in the dog due to a *Toxoplasma gondii* – like apicomplexan organism and *Neospora caninum* has been previously described.^{1,2} In addition to these two organisms, the differential diagnosis for protozoal dermatitis in the dog includes *Sarcocystis canis*, *Leishmania sp.*, and *Caryospora sp.*^{2,3} *Caryospora sp.*, *Sarcocystis canis* and *Leishmania sp* have sufficiently distinctive morphologic characteristics to assist in their diagnosis (i.e. caryocysts, schizont formation and the presence of a distinct kinetoplast, respectively).

Toxoplasma gondii and *Neospora caninum* are sufficiently similar in routine histological sections that immunohistochemistry and electron microscopy (EM) are usually employed to distinguish between these two apicomplexan organisms. The *Toxoplasma gondii*-like organism described by Dr. Dubey, et al¹ reacts with a polyclonal rabbit antibody to *T. gondii*, but its ultrastructural characteristics differ significantly. The *T. gondii*-like organism formed schizont-like organisms with a residual body that was best appreciated on EM but could be seen on close examination of the routine histological sections. In addition, the rhoptries in this organism were several in number and electron dense as compared to the few electron lucent rhoptries in *T. gondii*. Close examination of the sections in this case did not reveal any definitive residual bodies. However, electron microscopic examination would be necessary to determine if the protozoa in this case are *T. gondii* or the organisms described by Dr. Dubey.

Predisposing factors such as chronic ehrlichiosis, cardiomyopathy and immune-suppressive therapies for immune-mediated disease and neoplasia were reported in many cases of protozoal dermatitis in the dog.^{1,2}

AFIP Diagnosis: Haired skin and subcutis: Dermatitis and vasculitis, necrotizing, acute, diffuse, severe, with multifocal erosion, and myriad intra- and extracellular protozoal tachyzoites, mixed-breed, canine.

Conference Comment: As mentioned by the contributor, organisms are noted both intra- and extracellularly. The intracellular organisms are present in many different cell types including fibroblasts, follicular and epidermal keratinocytes, sebocytes, apocrine ductular epithelium, endothelium, macrophages, adipocytes, and myocytes of the erector pili muscles.

This case was reviewed in consultation with Dr. J.P. Dubey, United States Department of Agriculture, Animal Parasitic Diseases Laboratory, who performed immunohistochemistry using polyclonal antibodies to *Neospora caninum* and *Toxoplasma gondii*. In his laboratory, the organisms exhibit weak positive immunoreactivity for *Neospora caninum* and strong positive immunoreactivity for *Toxoplasma gondii*. Electron microscopy is necessary to positively identify the organism.

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CASE IV – 433846B (AFIP 2948686)

Signalment: Three month-old lamb, belonging to a sheep flock of 380 lambs of which 40% died.

History: The animal presented with a fever, swollen eyelids, excessive lacrimation, and mucopurulent nasal discharge.

Gross Pathology: Skin lesions were found on the areas free from wool and were characterized by raised, circular plaques that occasionally had congested borders.

Laboratory Results: Sheep pox antigen was identified in cryostat sections by immunohistochemistry using monoclonal antibodies.

Contributor's Morphologic Diagnosis: Skin: Hydropic degeneration of stratum spinosum keratinocytes, epidermal hyperplasia, and intracytoplasmic eosinophilic inclusion bodies, predominantly in squamous epithelium of hair follicles, consistent with sheep pox.

Contributor's Comment: The epidermis is hyperplastic with hyperkeratosis, acanthosis and significant ballooning degeneration. Occasionally, within keratinocytes, there are pale eosinophilic intracytoplasmic inclusion bodies. In the dermis, there is multifocal infiltration of neutrophils, lymphocytes, and histiocytes.

Sheep pox is a malignant pox disease of sheep characterized by fever, multiple non-vesicular swellings on the skin and mucous membranes, rhinitis, conjunctivitis, respiratory distress (due to the pressure on the upper respiratory tract from the swollen retropharyngeal lymph nodes and developing lung lesions) and death. Sheep pox occurs in all ages of sheep but the disease is most severe in lambs, with mortality reaching 80-100%.

Skin lesions are often less obvious on post mortem examination of acutely infected animals than in live animals with disease. Gross findings include necrotic mucous membranes, enlarged and edematous lymph nodes, and typical pox lesions characterized by papules, which may be ulcerated, on the abomasal mucosa, tongue, hard and soft palates, trachea and esophagus, and sometimes on the wall of the rumen and the large intestinal mucosa. Pale areas of approximately 2 cm in diameter may occasionally be seen on the surface of the kidney and liver, and have been reported on the testicles. Throughout the lung, but particularly in the diaphragmatic lobes, there are numerous white-gray firm lesions up to 5 cm in diameter. The pathological lesions and clinical signs mentioned above are pathognomonic for sheep pox.

Generalized contagious ecthyma (Orf) or parapox viral infection is rare. Confusion could occur between mild sheep pox and orf or even insect bites. Sheep pox causes extensive economic loss through high mortality, reduced meat, milk or wool yields, quarantine requirements, and the cost of disease prevention programs.

Capripox is endemic in Africa, north of the equator, the Middle East, including Israel, Turkey and Iran, and in Afghanistan, India, Nepal, parts of the Peoples

Republic of China, and since 1984, Bangladesh. Recently, it has made frequent incursions into southern Europe.

Transmission of infection is by direct contact with diseased sheep or indirect contact via a contaminated environment. Transmission is usually via aerosolization, but virus can also be spread mechanically by insect bites or experimentally by intradermal or subcutaneous injection.

The incubation period is 4-7 days and is followed by a leukocyte-associated viremia. The virus localizes in many organs including the skin, where virus concentration peaks at 10-14 days post infection. The skin lesions develop 1-2 days later, especially in the sparsely woolled areas and typically involve the eyelids, cheeks, nostrils, vulva, udder, scrotum, prepuce, ventral surface of the tail, and the medial thigh.

Sheep pox lesions have a prominent vesicular stage. The vesicles are umbilicated, multiloculated, and yield only a small amount of fluid if punctured. Occasionally, a large vesicle forms as a result of separation of the necrotic epidermis from the underlying dermis. The pustule stage is characterized by the formation of a thin crust. There may be marked gelatinous dermal edema and in severely affected animals the lesions coalesce. Healing of the skin lesions is slow, taking up to 6 weeks and a scar may remain. Highly susceptible animals often develop hemorrhagic papules early in the course of the disease and later ulcerative lesions in the gastrointestinal and respiratory tracts. Approximately one third of animals develop multiple pulmonary lesions composed of foci of pulmonary consolidation. The kidneys have multifocal, circular, fleshy nodules throughout the cortices.

Histologically, sheep pox lesions have the typical epithelial changes seen with poxviruses, including marked hydropic degeneration of keratinocytes in the stratum spinosum, microvesiculation, epidermal hyperplasia and eosinophilic intracytoplasmic inclusion bodies. The lesions affect both surface epithelium and follicular epithelium. Marked dermal lesions reflect the systemic route of cutaneous involvement and may be due to immune mediated disease as well as direct viral damage. During the papular stage, large numbers of mononuclear cell accumulate in the increasingly edematous dermis. These cells, first described by Borrel, are called "cellules claveleuses" or sheep-pox cells, and are characteristic of the disease. The nuclei of sheep-pox cells are vacuolated and have margined chromatin. The vacuolated cytoplasm contains single, occasionally multiple, eosinophilic intracytoplasmic inclusion bodies. Sheep-pox cells are virus-infected monocytes, macrophages and fibroblasts, but not endothelial cells.

Approximately 10 days post-infection, and corresponding with the peak of dermal infectivity, a severe necrotizing vasculitis develops. Virus particles have not been

identified in endothelial cells and it is thought that the vasculitis may be due to immune complex deposition. Ischemic necrosis of the dermis and overlying epidermis follows. The pulmonary lesions are proliferative alveolitis and bronchiolitis with focal areas of caseous necrosis. Alveolar septal cells contain intracytoplasmic inclusion bodies. Histologic lesions, characterized by the accumulation of sheep pox cells, may involve the heart, kidney, liver, adrenal glands, thyroid gland, and the pancreas.

AFIP Diagnosis: Haired skin: Dermatitis, hyperplastic, subacute, multifocal, moderate, with epidermal and follicular keratinocyte ballooning degeneration, eosinophilic intracytoplasmic inclusion bodies, and sheep pox cells, breed not specified, ovine.

Conference Comment: As mentioned by the contributor, intracytoplasmic inclusion bodies may be present in several cell types. Conference attendees noted intracytoplasmic inclusion bodies in surface and follicular epithelial cells, and in dermal fibroblasts and macrophages.

Many strains of poxviruses are species specific and are given the name of the species that they infect (i.e turkeypox virus, canarypox virus, cowpox virus, etc.), while others may infect a wide range of hosts. Some pox diseases of vertebrates (family Poxviridae, subfamily Chordopoxvirinae) include the following:^{5,6,7,8}

Genus	Virus	Major Hosts	Geographic Distribution
Orthopoxvirus	Variola virus (smallpox)	Humans	Eradicated globally
	Vaccinia virus	Numerous: humans, cattle, buffalo, swine, rabbits	Worldwide
	Cowpox virus	Numerous: cattle, humans, rats, cats, gerbils, lg. felids, elephants, rhinoceros, okapi	Europe, Asia
	Camelpox virus	Camels	Asia, Africa
	Ectromelia virus (mousepox)	Mice, voles	Europe
	Monkeypox virus	Numerous: squirrels, monkey, anteaters, great apes, humans	Western and central Africa
	Uasin Gishu disease virus	Horses	Eastern Africa
	Tatera poxvirus	Gerbils (<i>Tatera kempfi</i>)	Western Africa
	Raccoon poxvirus	Raccoons	North America
	Vole poxvirus	Voles (<i>Microtus californicus</i>)	California
	Seal poxvirus	Grey seals	North Sea
Capripoxvirus	Sheeppox virus	Sheep, goats	Africa, Asia
	Goatpox virus	Goats, sheep	Africa, Asia
	Lumpy skin disease virus	Cattle, Cape buffalo	Africa

Genus	Virus	Major Hosts	Geographic Distribution
Suipoxvirus	Swinepox virus	Swine	Worldwide
Leporipoxvirus	Myxoma virus	Rabbits (<i>Oryctolagus</i> and <i>Sylvilagus</i> spp.)	Americas, Europe, Australia
	Rabbit (Shope) fibroma virus	Rabbits (<i>Oryctolagus</i> and <i>Sylvilagus</i> spp.)	Americas, Europe, Australia
	Squirrel fibroma	Gray squirrels and woodchucks	Eastern United States
	Hare fibroma	European hares	Europe
Molluscipoxvirus	Molluscum contagiosum virus	Humans, horses, chimpanzees, kangaroos	Worldwide
Yatapoxvirus	Yabapox virus	Monkey, humans	West Africa
	Tanapox virus	Monkey, humans	West Africa
Avipoxvirus	Fowlpox virus	Chickens, turkeys, other birds	Worldwide
Parapoxvirus	Orf virus	Sheep, goats, humans	Worldwide
	Pseudocowpox virus	Cattle, humans	Worldwide
	Bovine papular stomatitis virus	Cattle, humans	Worldwide
	Auzdyk virus	Camels	Africa, Asia
	Seal parapoxvirus	Seals, humans	Worldwide

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