The Armed Forces Institute of Pathology
Department of Veterinary Pathology
WEDNESDAY SLIDE CONFERENCE
2005-2006

CONFERENCE 19
15 March 2006

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Washington D.C.

CASE I – RP-11124 (AFIP 2994575)

Signalment: Adult male mallard duck (*Anas platyrhynchos*).

History: Wild duck found on zoo grounds unable to fly. Clinical findings included a fracture of the left humerus, emaciation and dyspnea. Because of a poor prognosis euthanasia was elected.

Gross Pathology: Multifocal to coalescing yellow nodules (granulomas) were found in multiple sites including the lung, liver, coelomic cavity and left humerus (with pathologic fracture). Impression smears demonstrated myriad beaded acid-fast bacilli within these lesions.

Laboratory Results: *Mycobacterium avium* was isolated from the lung, liver and humerus.

Histopathologic Description: Provided is a section of trachea and adjacent soft tissue. There is transmural inflammation that partially fills the tracheal lumen and results in focal loss of the osseous tracheal ring and tracheal mucosa. Lesions are composed of granulomas with abundant central cellular debris (predominantly degenerate heterophils) bounded by thin layers of macrophages that are frequently multinucleated. Macrophages, in turn, are bounded by small to moderate amounts of dense fibrous connective tissue containing variable numbers of macrophages, lymphocytes, plasma cells and non-degenerate heterophils. Within the tracheal lumen, debris is admixed with small to moderate numbers of septate, acute angle branching fungal hyphae. In some sections, hyphae are associated with light yellow pigmented conidiophores suggestive of *Aspergillus* sp. A Ziehl-Neelsen acid-fast stain shows myriad acid-fast bacilli within macrophages and admixed

within central debris in granulomas outside the trachea. Multifocally in some sections there are colonies of Gram-positive cocci admixed with debris both inside and outside the tracheal lumen. There is squamous metaplasia of remaining tracheal mucosa.

Contributor's Morphologic Diagnosis: Trachea: tracheitis, necrotizing, heterophilic and granulomatous, chronic and active, transmural, marked with intralesional fungal hyphae, intralesional acid-fast bacilli, and intralesional cocci bacteria

Contributor's Comment: This case represents a combined occurrence of mycobacteriosis (avian tuberculosis) and probable tracheal aspergillosis based on the morphology of fungal hyphae and the observation of distinctive conidiophores. Mixed infections with these common infectious agents can sometimes present a diagnostic challenge as it is easy to focus on the prominent fungal hyphae, while overlooking the acid-fast bacilli. The presence of multicentric granulomas with intralesional acid-fast bacilli (subsequent isolation of *M. avium*) was useful in avoiding this error in this case. The presumptive aspergillosis is interpreted as secondary lesion to debilitation associated with the disseminated mycobacteriosis. Speculatively, the tracheal mycobacterial lesion may also have disrupted airflow or local mucociliary clearance with subsequent settling of fungal spores within the tracheal lumen. The possibility that the tracheal mycobacterial lesion began within a cervical air sac with extension into the tracheal wall cannot be excluded.

Mycobacteriosis ("avian tuberculosis") is a relatively common disease in a wide variety of avian species (1). Although usually associated with members of the *M. avium-intracellulare complex* (MAI complex), infections with organisms such as *M. genavense* are increasingly recognized, and rarely other mycobacteria, including *M. tuberculosis* are identified as avian pathogens (1, 2). Many of the mycobacteria implicated in avian mycobacteriosis are environmentally ubiquitous and infections are frequently thought to be opportunistic. Infected birds may serve as sources of heavy environmental contamination (1). Mycobacteriosis can cause widespread mortalities in collections of zoo birds (3) as well as sporadic morbidity and mortality. Lesions can vary considerably from classic multicentric granulomas in a variety of tissues to diffuse histiocytic infiltrates without discrete granuloma formation (1, 4). Antemortem clinical diagnosis of avian mycobacteriosis is challenging and is a focus of current research (5).

Aspergillosis is common in a wide range of avian species and like mycobacteriosis is caused by organisms that are common environmentally. The most common isolates from affected poultry are *Aspergillus fumigatus* and *Aspergillus flavus* (6). Risk factors for the development of aspergillosis include environmental conditions that increase exposure to infective spores as well as less easily defined factors such as "stress" and debilitation associated with other underlying conditions. In

the absence of fungal culture, presumptive diagnosis of aspergillosis can be made by identifying characteristic conidiophores in histologic section. Diagnostic challenges for the pathologist are cases of aspergillosis without widespread respiratory lesions. These might include isolated lesions in the trachea ("aspergillomas") that are easily overlooked if the trachea (especially the syrinx) is not opened or carefully examined during necropsy or pectoral muscle infarcts associated with lesions in the great vessels or brachiocephalic arch (7).

AFIP Diagnosis: Tracheitis, necrotizing, heterophilic and granulomatous, transmural, focally extensive, severe, with ulceration, osteolysis, squamous metaplasia, fungi, colonies of cocci and many acid fast bacilli, mallard duck, avian.

Conference Comment: The contributor provides a brief review of both avian mycobacteriosis and aspergillosis. Attendees identified the colonies of cocci, fungal hyphae and conidiophores within the section but did not have the benefit of special stains prior to the conference. Acid-fast bacilli are readily identifiable with both the Fite-Faraco and Ziehl-Neelsen stains and fungal hyphae are clearly visible in Gomori's methenamine silver-stained sections.

Attendees recognized conidiophores and felt their morphology was distinctive for *Aspergillus* sp.. Aspergillosis is a common opportunistic infection and should be the top differential when fungi are identified in lesions lining airways. Conidiophores are specialized structures from which asexual spores, or conidia, arise. The end of the conidiophore forms a terminal vesicle which develops sterigmata (phialides) from which chains of conidia are produced. (8) Only if the *Aspergillus* is growing in a cavity with airspace can it form a "fungus ball" in which conidia and conidiophores are frequently observed. (9)

The moderator stressed the need to run acid-fast stains on all granulomatous lesions in birds and reptiles in order to detect mycobacterial infections which might otherwise be attributed to an opportunistic infection.

Readers are encouraged to review Wednesday Slide Conference 4, case 1, 2005-2006, for a case of avian mycobacteriosis in an Ara.

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CASE II – 05L-1248 (AFIP 2988622)

Signalment: 14 year old, female, *Equus caballus*, equine

History: The mare was presented for investigation of a visible pale coloured ocular mass in the anterior chamber of the right eye. Additionally, an intraocular retrolental mass was identified clinically and ultrasonographically. The affected eye was blind. There were no direct or consensual light responses in the left eye. The globe was enucleated.

Gross Pathology: Bulbus 4.6 cm anterior posterior axis, 4.9 cm naso-temporal axis. The dorso-medial segment of the anterior chamber is partly filled with a spongy, pale pink-whitish, soft and fragile tissue mass (1.6 x 2.6 x 0.5cm), which is adherent to the anterior surface of the iris, the corneal endothelium and protruding into the chamber angle.

The posterior eye segment reveals a comparable retrolental, cup shaped mass (≈ 3cm in diameter) extending from the posterior iridial surface and ciliary body into the posterior eye chamber towards the retina, by posterior displacement of the yellowish, liquid vitreous. In the ventro-medial segment, there is focal moderate acute subretinal haemorrhage.

Laboratory Results:
Immunohistochemistry:

Antibodies	Ciliary body (npe)	Retinal cells	Tumour cells
GFAP	-	+ +	±
NSE	+	+	±
Neurofilament	-	±	+
Synaptophysin	-	+	+ +
Retinal S-protein	±	+ +	±
Vimentin	+ +	+ +	+ +
Pan-Cytokeratin	-	-	-

+ - + + +	positive immune reaction
±	focally positive immune reaction (single cells)
-	no immune reaction

Special stains: PAS/Alcian Blue: multifocally Alcian blue positive areas between sheets of tumour cells, focally centrally in rosettes; multifocally linear PAS-positive structures at the apical cell junction (resembling outer limiting membrane)
Van Kossa Stain: multifocal areas of calcification within necrotic tumour tissue

Histopathologic Description: The tissue masses in the anterior eye chamber and anterior uvea are composed of undifferentiated spindloid to spherical cells with scant homogeneous eosinophilic cytoplasm and large, pleomorphic, hyperchromatic nuclei with 1-4 nucleoli of variable size (anisocytosis, anisokaryosis). Mitotic figures are frequent (2-4 per high power field).

The cells resemble primitive neuroectodermal tissue, are arranged in sheets, nodules or cords, form rosette like structures with or without a central cavity (Flexner-Wintersteiner / Homer-Wright rosettes) and surround well differentiated, thin walled (venous) blood vessels of variable caliber.

Within the endophytic neoplastic mass, apoptotic cells are numerous and there are extensive areas of cellular debris (necrosis) and calcification. Tumour cells focally invade the anterior iridial stroma (infiltrative growth) and expand subretinally (exophytic growth) causing focal retinal detachment. The non-pigmented

epithelium of the ciliary body reveals mild to moderate cytoplasmic vacuolation and variable cellular size (atrophy). In the vitreous, small numbers of erythrocytes and macrophages containing intracytoplasmic granular brown pigment (melano-macrophages) and focally macrophages with intracytoplasmic erythrocytes (erythrophagocytosis) are seen. Epiretinally, appearing bandlike (outer limiting membrane) and vitreally/ subretinally appearing globular, there are large amounts of basophilic material (condensed chromatin). The retina shows mild to moderate atrophy, predominantly of the neuronal and outer granular layers. Focally there are moderate amounts of subretinal amorphous eosinophilic material (subretinal oedema) and a focal mild hypertrophy of the retinal pigment epithelium (rpe). The conjunctiva shows a mild subepithelial infiltration with lymphocytes, plasma cells and neutrophils.

Contributor's Morphologic Diagnosis: Eye: Tumour, primary intraocular, endo- and exophytic, primitive neuroectodermal, (non-teratoid medulloepithelioma-/retinoblastoma-like), with transplantation metastasis into the anterior eye chamber, atrophy of non-pigmented epithelium of the ciliary body, retinal atrophy and detachment, vitreal/subretinal haemorrhages and oedema, chronic neutrophilic conjunctivitis, equine, female, 14 years

Contributor's Comment: Primary intraocular tumours of neuroectodermal origin are divided into two groups: tumours deriving from mature neuroepithelium, i.e. adenomas, adenocarcinomas of the (unpigmented/pigmented) ciliary epithelium and neoplasms derived from primitive medullary epithelium (during differentiation of the optic vesicle or optic cup) including medulloepitheliomas and retinoblastomas[1].
In animals, medulloepitheliomas in general are rare, congenital tumours, deriving from primitive neuroectoderm of the optic cup. They have multipotentiality and a retained ability to differentiate into retina, ciliary epithelium, vitreous or neuroglia[2]. The majority of medulloepitheliomas are reported in young animals, classified as benign or malignant, nonteratoid or teratoid. Nonteratoid medulloepitheliomas are solely composed of neuroepithelial cells, whereas teratoid medulloepitheliomas include neuroectodermal tissue and heteroplastic elements, e.g. hyaline cartilage, spindle cells and a myxomatous matrix[3,4]. Medulloepitheliomas are described in goldfish, cockatiels, llamas, feline, canine, and equine species[4].

The entity of retinoblastomas in animals is still under debate[3]. One confirmed case of a retinoblastoma in a dog has been described[5]. The morphological and immunohistological features described in this case are consistent with a primitive neuroectodermal tumour, exhibiting the characteristics of both, medulloepithelioma and retinoblastoma.

Due to the fact, that the distinction between medulloepithelioma and retinoblastoma can be very difficult, primitive primary neuroectodermal intraocular tumours may exhibit features of both entities and the diagnosis requires a series of criteria[6,7], a final decision in this case could not be made solely on a morphological and immunohistological basis.

AFIP Diagnosis: Eye: Primitive neuroectodermal tumor with features of medulloepithelioma and retinoblastoma, *Equus caballus*, equine.

Conference Comment: The contributor provides an example of an ocular primitive neuroectodermal tumor (PNET) with areas that have medulloepithelioma and retinoblastoma-like features. The majority of the neoplasm is composed of sheets of primitive neuroectodermal tissue with a high mitotic index. Multifocally, neoplastic cells form large rosettes or tubular structures that have a pseudostratified, columnar arrangement, rest along segments of PAS positive basement membrane and have a vague inner limiting membrane, features consistent with medulloepithelioma. Also present are many, small, Flexner-Wintersteiner rosettes. Rosettes are considered a characteristic feature of both medulloepitheliomas and retinoblastomas making interpretation, beyond neuroectodermal origin, difficult. Within medulloepitheliomas, rosettes are generally interpreted as the neoplasm's attempt to recapitulate portions of the optic vesicle or cup. In retinoblastomas, rosettes are interpreted as neuronal cells forming photoreceptors in an attempt to recapitulate portions of the retina. In this case, the contributor also provided TEM findings supportive of retinoblastoma. Within the luminal protrusions of a small rosette, cilia and basal bodies are present.

Medulloepitheliomas are uncommon; however, they are the most common primary intraocular neoplasm in horses. As mentioned by the contributor, medulloepitheliomas are derived from the embryonic neuroectoderm lining the inner layer of the optic cup and have the potential to differentiate into iridociliary epithelium, retina, vitreous and neuroglia. (7)

Retinoblastomas are neuroepithelial tumors that usually arise in the posterior retina and are the most common primary intraocular malignancy of children. In approximately 40% of human cases, retinoblastoma occurs in those who inherit a germ-line mutation of one retinoblastoma (RB) allele. Germ line associated retinoblastomas can be bilateral and may also be associated with pinealoblastoma, referred to as "trilateral" retinoblastoma. Since there has been only one confirmed case of retinoblastoma in a dog; with the remainder of animal cases occurring in fish, transgenic mice, and primates, the biologic behavior of these tumors in animals is unknown. In humans, retinoblastomas may invade along the optic nerve

and choroid and those cases which involve the pineal gland are associated with a dismal outcome. (1,8)
The diagnosis of retinoblastoma in human medicine is based on histomorphology, immunohistochemical demonstration of retinal S-antigen, GFAP and carbonic anhydrase (CA), and identification of mutations in the RB1 gene. Histologically, this horse tumor has features of both medulloepithelioma and retinoblastoma. Based on immunohistochemistry performed at the Armed Forces Institute of Pathology, the tumor is negative for GFAP, S-100 protein, NSE, and NFP. It diffusely stained lightly to moderately strong for CD99.

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CASE III – 360/05 (AFIP 2983553)

Signalment: Six-year-old male Maine Coon cat (*Felis catus*).

History: The animal was found dead without any previous clinical signs.

Gross Pathology: Except for acute congestion of liver, spleen, lung and kidneys as well as an acute alveolar edema of the lung, the animal was grossly unremarkable.

Histopathologic Description: Unfortunately the tissues contain artefacts due to the frozen condition in which the animal was sent to the institute. Remarkable histopathologic changes were found in the brain of the cat (cerebellum submitted). There are lightly basophilic inclusions within the cytoplasm of nerve cell bodies, neuronal processes, and scattered within the neuropil. Most of the inclusions can be found in Purkinje cells. The inclusions are of variable size ranging from 2 to 10 μm in diameter. They are non membrane-bound spherical structures containing a deeply hematoxyphilic central core with a spiculated outline and a surrounding zone of less staining material. The inclusions stain positively with PAS and Alcian blue (slides not submitted).

Contributor's Morphologic Diagnosis: Cerebellum: Multifocal polyglucosan inclusions (Lafora bodies Type II) within neuronal perikarya, neuronal processes and scattered within the neuropil, most numerous in Purkinje cells.

Contributor's Comment: Lafora bodies are composed of polyglucosans (polymers of sulphated polysaccharides) and regarding composition and staining characteristics they are similar to corpora amylacea.[2] The inclusions stain positively with PAS, Alcian blue, and methenamine silver.[5] However within the Lafora bodies there are differences in internal structure and staining characteristics and based on these varieties some authors recognize three types of Lafora bodies[4]:

1. Type I - Small (3 to 10 μm in diameter), fine, evenly stained granules. This is the most common type and is usually found in middle and deep layers of the cerebral cortex and in glial cells of the cerebellum. Ultrastructurally, these bodies consist of branching fibrillar structures without a limiting membrane. The branching filaments measure about 8 - 10-nm in diameter[4].

2. Type II - These larger bodies (13 to 30 μm in diameter) have a strongly PAS-positive homogeneous core with a more faintly staining radiating periphery. This form is commonly found in Purkinje cells of the cerebellum and in the midbrain. Electron microscopy reveals osmiophilic granules in a central core surrounded by fibrillar material. Rough endoplasmic reticulum in affected neurons may be dilated with increased numbers of coarse ribosomes free in the cytoplasm. Such changes suggest abnormalities in protein synthesis[4].

3. Type III - These bodies range from 5 to 20 μm in diameter and are occasionally found in the midbrain. These structures exhibit a dense peripheral ring of PAS-positive material[4].

In humans Lafora's disease is a severe progressive myoclonic epilepsy caused by inherited recessive mutations in the EPMA2A (chromosome 6q24) or EPMA2B (chromosome 6p22.3) genes[1].

In domestic animals Lafora bodies have been found as incidental changes within the central nervous system in dogs, cats and various other species[5]. Commonly they were found in aging animals (> 8 years old)[6] but occasionally they occurred in young aminals[5]. In some cases Lafora bodies were associated with neurological signs including depression, somnolence and seizures in advanced stages of the disease[4].

When the inclusions are associated with clinical signs the Lafora bodies are often located within the neuronal perikarya, whereas the incidentally found polyglucosan bodies were more often located in the neuropil than in the cytoplasm of the nerve cell bodies[5]. In this cat, inclusions can be found within the neuronal perikarya, although the owner reported no clinical signs.

AFIP Diagnosis: Cerebellum, Purkinje cells, molecular and granule cell layers, and white matter: Polyglucosan (Lafora) bodies, numerous, Maine Coon, feline.

Conference Comment: The contributor provides a brief review of Lafora's disease (LD) and more specific ultrastructural details of the three types of Lafora bodies.

Lafora bodies are carbohydrate-rich intracytoplasmic neuronal inclusions that are widespread in the CNS of humans and other animal species with Lafora's disease. Lafora bodies are most commonly found in thalamic neurons and cerebellar Purkinje cells and less commonly in glial cells. They may be located in the perikaryon, dendrites and axons and/or occasionally free within the neuropil. Lafora bodies may also be found in the heart, liver, voluntary muscles and skin, especially the sweat glands, which are often biopsied in humans to diagnose LD in its early stages.

Lafora bodies are basophilic to amphophilic and range from 5-20 um in diameter. They generally have a dense core, are occasionally concentrically laminated, and may have a radiating or sunburst pattern in their outer zone.

Lafora's disease in humans is a rare, fatal, neurometabolic disorder with an autosomal recessive mode of inheritance. Patients with LD present in late childhood or adolescence, after a period of normal development, with myoclonus

and seizures which become intractable within two years of onset. Sadly, patients undergo progressive cognitive decline resulting in a vegetative state and continuous myoclonus prior to death. Death typically occurs within 10 years of onset of clinical signs.

Several mutations in the EPM2A or EPM2B gene have been identified as the cause of LD in humans. The EPM2A gene encodes for the protein laforin which may detect accumulation of polyglucosans and initiate elimination mechanisms to prevent further formation. The EPM2B gene encodes for the protein malin, an ubiquitin ligase that binds laforin and together they may destroy or prevent the production of polyglucosans; however, the relationship between laforin and malin remains uncertain.

Lafora's Disease in animals has been referred to as neuronal glycoproteinosis and has infrequently been reported in dogs, a fennec fox (7), a Maine Coon cat (5), and a Hereford/Angus crossbred cow (9). Animals may present with seizures or myoclonic contractions. Recently a research group at the Hospital for Sick Children in Toronto, Canada identified a genetic mutation in the EPM2B gene as responsible for LD in a group of Miniature Wirehaired Dachshunds. More than 5% of this breed in the UK have LD (8).

The differential diagnosis for Lafora bodies includes:

- Lafora-like bodies have similar histomorphologic characteristics and are found primarily in the neuropil but rarely within perikaryon. Lafora-like bodies are associated with aging but not with neurological signs.
- Corpora amylacea have similar morphology and staining patterns but are typically found within axons and astrocytic processes and not within the perikaryon of neurons. Corpora amylacea have been identified in multiple tissues and are also associated with aging.
- Amylopectin bodies are deposits of abnormal polysaccharides found in tissues of patients with glycogen storage disease IV or glycogen branching enzyme deficiency. Clinical signs may be present at birth or within the first few months of life. Humans with this disease develop liver dysfunction with hepatosplenomegaly, progressive cirrhosis, and chronic hepatic failure followed by early death. Glycogen storage disease type IV has been described in Norwegian Forest cats and amylopectinosis has been described in neonatal Quarter horses. Polysaccharide deposits can be found in the liver, heart, tongue and CNS. Inclusions are PAS (+), diastase resistant and black when stained with iodine.

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CASE IV – 15652-03 (AFIP 2935879)

Signalment: 16-yr-old, male castrate, mixed breed dog, *Canis familiaris*

History: The dog had an inflamed left eye that became enlarged and bulging over a 2-3 month period with corneal hyperemia. The left eye pressure was 30 and the right eye 20. The eye with a pale retrobulbar mass was submitted from the enucleation surgery. The dog is doing fine 5-months after surgery.

Gross Pathology: Pale, firm, conical mass surrounding the eye and extending posteriorly. Gross photo submitted of the formalin-fixed tissue.

Histopathologic Description: The tumor cells are somewhat pleomorphic with large, vesicular and sometimes nearly clear, nuclei with a prominent nucleolus and are polygonal and round to elongated and plump with a moderate amount of eosinophilic and pale amphophilic cytoplasm and rare to absent mitoses. The cells

are cohesive with mostly indistinct cell borders and are in nests and sheets with wide infiltration throughout the muscle, fibrous fascia, and fat with local dense fibroplasia or scirrhous reaction. The compact nests of the large cohesive, polygonal cells do have distinct cell membranes. There are occasional small nests of compact swirling tumor cells (meningothelial pattern) but no psammoma bodies (laminated tumor cell whorls with central mineralization).[8] The retina is atrophic with loss of the ganglion cell layer and partial loss of the inner nuclear layer cells and nests of tumor cells are also infiltrating the outer sclera. Some blocks in the present case show occasional foci of osseous metaplasia within the tumor

Contributor's Morphologic Diagnosis: Orbital (retrobulbar) meningioma

Contributor's Comment: This unusual tumor, although rare, is the most common primary orbital tumor in dogs[1] with other tumors being nasal carcinoma, mast cell tumor, and osteosarcoma.[1] They are usually benign to locally infiltrative, but at least one in a dog metastasized to the lungs.[1] The tumor cells are reportedly vimentin positive and are usually negative for cytokeratin except for some epithelioid types.[2,3] The diagnosis is largely based on the morphology and location. Primary optic nerve meningiomas arise from the arachnoid cap cells inside the dura of the optic nerve.[1,4,5] Secondary tumors infiltrate within the dura from the brain and have been occasionally seen in dogs.[4,6,7] Differentiation requires demonstration of normal tissue behind the tumor in the orbit, or CT scans for the presence or absence of tumor in the brain. The tumor is usually tightly wrapped around the optic nerve and back of the eye and tapers down as it approaches the optic nerve foramen and brain.[3] The tumor cells are variably sized and invade the local fatty fascia and muscle, usually with some large cells with plentiful eosinophilic, glassy, cytoplasm.[3] There is often a myxoid component with cartilage and bone metaplasia.[1, 2,3] The larger tumor cells have an eosinophilic cytoplasm and their tight clusters can be mistaken for invasive, anaplastic carcinoma.[3]

We believe that this is probably a case of primary orbital meningioma since there are no CNS signs and the dog is clinically normal 5-months later.

AFIP Diagnosis: Retrobulbar tissue: Meningioma, mixed breed dog, canine.

Conference Comment: Meningiomas of the optic nerve have been reported in people and dogs. These tumors grow within the space between the optic nerve and the surrounding muscle cone, causing slowly progressive compression of the optic nerve which can result in amaurosis - blindness without an apparent lesion to the eye. Some tumors infiltrate through the sclera into the choroid, along the optic nerve into the subretinal space, or through the optic foramen into the skull.

Extensive growth within the orbit may cause substantial orbital remodeling and even expansion. (2)

Of the seven convincing cases of canine optic nerve meningiomas reported in the literature; three were euthanized upon diagnosis, one was euthanized 3 months after removal of the retrobulbar tumor because of blindness caused by meningioma involving the optic chiasm and three were removed but recurred. Of the three that recurred, two had pulmonary metastasis. (1,6,7,9,10,11,13)

In this case, the contributor provided an update on the dog; it was euthanized nine months after the left eye and retrobulbar meningioma were removed. At the time of euthanasia, the right eye was inflamed and the dog was going blind. Chest radiographs a few months earlier did not identify metastases. A necropsy was not performed.

In general, meningiomas are usually discrete tumors with smooth surfaces and broad dural attachments which can arise anywhere along the meninges. They can be soft or firm, and may be gritty when cut. There are nine histologic variants of meningiomas seen in domestic animals. While all of them, except the anaplastic (malignant) variant, have similar biologic behavior i.e. they grow slowly and cause clinical signs by compressing the underlying nervous tissue, the subtypes are used to help identify all of these histologically different tumors as meningiomas and not another primary CNS or metastatic tumor. (8) The nine histologic variants of meningiomas are listed below:

- Meningothelial meningioma: composed of solid, moderately cellular lobules of polygonal cells
- Fibrous (fibroblastic) meningioma: composed of spindle cells arranged in long interlacing fascicles
- Transitional (mixed) meningioma: features of both meningothelial and fibrous meningiomas
- Psammomatous meningioma: a meningioma with psammoma bodies
- Angiomatous meningioma: a meningioma characterized by numerous large or small blood vessels
- Papillary meningioma: composed of meningothelial cells arranged in papillary structures on vascular cores
- Granular cell meningioma: composed of oval-to-polygonal cells with abundant granular eosinophilic cytoplasm, granules are PAS (+) and diastase resistant
- Myxoid meningioma: vacuolated neoplastic cells are separated by moderate-to-abundant myxomatous matrix which stains well with PAS, Alcian blue, and mucicarmine
- Anaplastic (malignant) meningioma: exhibits several features of malignancy e.g. frequent mitoses, high cellularity, uninterrupted patternless growth, extensive necrosis, brain invasion and metastasis

In cats, meningiomas often arise from the tela choroidea of the third ventricle and are easily separated from the brain parenchyma; whereas, in dogs meningiomas frequently interdigitate with the superficial brain parenchyma and are more difficult to remove. (8)

Contributor: Diagnostic Laboratory, Arkansas Livestock and Poultry Commission http://www.arlpc.org

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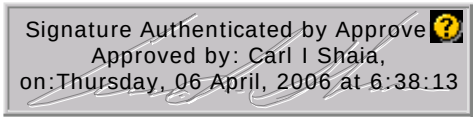

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*Sponsored by the American Veterinary Medical Association, the American College of Veterinary Pathologists and the C. L. Davis Foundation.