

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

CONFERENCE 11  
4 January 2006

**Conference Moderator:** Dr. Brian Wilcock, DVM, PhD  
Histovet Surgical Pathology  
Guelph, Ontario

**CASE I – 03 RD1618 (AFIP 2942334)**

**Signalment:** Bailey: 4-year-old neutered male domestic longhaired cat; *Felis catus*

**History:** The problem began with a rapid onset in early November 2003. Bailey was examined 11/12/03 and 12/10/03. An intraocular mass, probably vascularized, was noted medially displacing the iris forward and lens caudally. There was dyscoria and a low intraocular pressure. On the first exam, the fundus appeared normal. On second exam, the lesion was not visible due to a small pupil opening. The owners declined an attempt to excise the mass only. Enucleation of the left eye was performed.

**Gross Pathology:** There is a solid white mass distorting the ciliary body and cradling the lens.

**Contributor's Morphologic Diagnosis:** Feline iridociliary adenoma

**Contributor's Comment:** Feline iridociliary adenomas are the second most common primary neoplasm of the eye, following melanoma. This tumor is far less common than the iridociliary adenoma of dogs; however, it is more common than iridociliary epithelial tumors in humans. The morphology of the feline tumor is considerably different than the canine tumor. As in this case, the tumors in cats are usually made up of solid sheets of polygonal bland epithelial cells with a regular delicate vascular supply. Although the tumors may be large and extended to all corners of the globe, they always originate from the epithelial tissues which line the posterior chamber, iris and ciliary body. Feline iridociliary adenoma is a tumor which is commonly submitted for a second opinion because the feline tumor has a different morphology than the more common and familiar canine variant. A PAS stain is useful because a delicate basement membrane delineates individual cells or small clusters of cells highlighting the epithelial nature of this tumor.

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**AFIP Diagnosis:** Eye: Iridociliary adenoma, Domestic Longhair, feline.

**Conference Comment:** Conference attendees debated whether the tumor is present in the anterior chamber or the posterior chamber of the eye. Iridociliary adenomas originate from the iridociliary epithelium, which is of neuroectodermal origin. In this case, the neoplasm pushes the iris forward which partially fills the anterior chamber. Attendees speculated that the neoplasm may have effectively closed the filtration angle resulting in glaucoma. However, there is no loss of retinal ganglion cells suggestive of glaucoma.

Attendees included melanoma and extramedullary plasmacytoma in their differential diagnosis because of their frequencies and the distinct nesting and packeting arrangement of the cells. Keys to diagnosing iridociliary adenoma are recognition of the delicate fibrovascular stroma and the presence of a basement membrane. A Periodic Acid Schiff (PAS) procedure identifies the basement membrane and highlights the nest and packet pattern. Epithelial tumors require a basement membrane for support and survival. The only primary intraocular epithelial tumors are iridociliary adenoma and iridociliary adenocarcinoma.

In dogs, iridociliary adenomas range from papillary to solid, but almost all retain at least some regions with cuboidal epithelial cells. Pigment may be present, which can cause confusion, both clinically and histologically, with uveal melanocytoma. Additionally, the fibrovascular stroma is generally much more prominent in dogs than in cats (1). Anaplastic features and scleral invasion differentiate iridociliary adenocarcinomas from adenomas (3).

Although generally not required for diagnosis, immunohistochemistry may be useful in some cases. Iridociliary adenomas are almost always positive for vimentin, S-100 protein and neuron specific enolase (NSE) (1). Iridociliary adenocarcinomas are often positive for cytokeratin, but the normal iridociliary epithelium and cells of iridociliary adenomas are usually negative (3).

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## **CASE II – 05-488 (AFIP 2988013)**

**Signalment:** 10-year-old, female Miniature horse

**History:** Several days of not doing well. Off feed one morning and colic by evening.

**Gross Pathology:** Four well-delineated areas of jejunum, 6-10 cm in length, are thick and ulcerated and contain white to yellow plaques. Transmural necrosis and suppurative inflammation is present.

**Laboratory Results:** Leukopenia, acidosis, thickened small intestine on ultrasound

**Histopathologic Description:** Extensive necrosis, edema and suppurative inflammation of the submucosa with thrombosis of veins and lymphatics. Numerous gram-positive cocci are present throughout the submucosa forming dense colonies. The inflammation extends through the muscularis but is less severe than in the submucosa. The inner muscular layer has multifocal necrosis. The mucosa has focal necrosis and hemorrhage consistent with infarction.

**Contributor's Morphologic Diagnoses:** Transmural necrotizing, suppurative enteritis with thrombosis, infarction and gram positive cocci

**Contributor's Comment:** This horse had a bacterial infection of the intestinal wall caused by gram-positive coccoid bacteria. The organism was not cultured but is probably *Streptococcus equi* or *S. zooepidemicus*. The inflammation and probable organism suggest bastard strangles as the disease (1).

Strangles is caused by *Streptococcus equi* and produces suppuration of the lymph nodes of the head and neck. Some horses develop more extensive systemic disease, referred to as bastard strangles, with abscesses in lymph nodes of the mesentery and mediastinum, liver, kidney, intestine and elsewhere. This horse had no lesions in any other organs or lymph nodes, which would be unusual for a case of bastard strangles.

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**AFIP Diagnoses:** Small intestine: Vasculitis, necrotizing, multifocal, severe, with thrombi, infarcts, and colonies of cocci, Miniature horse, equine.

**Conference Comment:** Conference attendees agreed that the central pathologic event occurring in this case is vasculitis, which was most likely the result of bacteremia, with subsequent thromboembolism and infarction. Some felt the pale areas of infarction were more representative of arterial rather than venous infarction.

Strangles is caused by *Streptococcus equi* subsp. *equi* and is characterized by suppurative rhinitis and mandibular and retropharyngeal lymphadenitis. Transmission occurs through feed, aerosolized droplets or exudate containing the bacterium. *S. equi* penetrates the nasopharyngeal mucosa and spreads via lymphatic vessels to the mandibular and retropharyngeal lymph nodes. In some cases there is hematogenous dissemination of bacteria resulting in abscesses of multiple organs including the lungs, liver, kidney, spleen, brain and joints; such cases are commonly known as "bastard strangles". Gross findings include mucopurulent nasal discharge with hyperemic respiratory mucosa, conjunctivitis, and purulent lymphadenitis. Occasionally, retropharyngeal and mandibular lymph nodes rupture leading to suppurative cellulitis and draining cutaneous ulcers. Sequelae to strangles include: bronchopneumonia due to aspiration of exudate; laryngeal hemiplegia ("roaring") resulting from enlarged retropharyngeal lymph nodes compressing the recurrent laryngeal nerves; facial paralysis and Horner's syndrome due to compression of cranial nerves; purpura hemorrhagica, believed to be caused by the deposition of *S. equi* antigen-antibody complexes in arterioles, venules, and capillaries resulting in vasculitis; infection of the paranasal sinuses and guttural pouches and cellulitis due to ruptured abscesses (2).

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### **CASE III – 05RD0943 (AFIP 2985166)**

**Signalment:** Chili Dog: 8-year-old, neutered male Catahoula Leopard dog, *Canis familiaris*

**History:** In May 2005, Chili Dog presented to the ophthalmologist with a three-week history of right eye abnormality. The veterinarian categorized her findings as buphthalmos, conjunctival hyperemia, severe corneal edema, dyscoria, ventro-medial cyclodialysis, lens luxation, central cupping of the optic nerve head, heterochromia OS, and normal gonioscopy OS. The dog also had glaucoma, OD. The right globe was enucleated.

**Gross Pathology:** A white-tan mass partially fills the anterior chamber (Fig 1).

**Histopathologic Description:** The tissue submitted is the formalin fixed right globe from an 8 year old, male castrated, Catahoula Leopard dog. Grossly and histologically, the globe is enlarged, associated with a basophilic cellular infiltration involving the inferior iris leaflet, ciliary body, and peripheral choroid, which is adherent to the lens. Histologically, the mass is composed of fascicles of neoplastic spindle cells, which contain oval nuclei and wispy eosinophilic cytoplasm. Neoplastic cells involve the inferior iris leaflet, ciliary body, and peripheral choroid, which is adherent to the anterior lens capsule and forms a cavitated area (cyclodialysis), with a broad band of neoplastic cells spanning to the end of Descemet's membrane. Furthermore, neoplastic cells line Descemet's membrane peripherally. Neoplastic cells are noted within the superior iris leaflet. There is corneal stromal neovascularization and suppurative inflammation. There is a preiridal fibrovascular membrane, associated with peripheral anterior synechiae, in addition to angle recession. There is fibrous metaplasia of lens epithelium (anterior subcapsular cataract). There is full thickness retinal atrophy, with sparing, in addition to gliosis and atrophy of the optic nerve head. Choroidal pigmentation is minimal.

**Contributor's Morphologic Diagnoses:**

1. Spindle cell tumor of blue-eyed dogs
2. Glaucoma
3. Preiridal fibrovascular membrane, associated with peripheral anterior synechiae and posterior synechiae
4. Anterior subcapsular cataract
5. Cyclodialysis
6. Iridocorneal angle recession

**Contributor's Comment:** Spindle cell tumors of the iris occur in dogs that have blue irides or partially blue irides. These tumors usually occur as flat masses with

diffuse but irregular involvement of the anterior uvea. More than one half of the cases diagnosed in the pathology lab are recognized as being neoplastic by the clinical ophthalmologist. Morphologically, the spindle cells range from bland appearing, elongate cells with small, oval nuclei to highly pleomorphic and anaplastic cells with multinucleate cells and karyomegaly. Mitotic figures are highly variable. Neoplastic cell organization suggestive of peripheral nerve sheath tumor is commonly seen. Antoni A and Antoni B cellular organization is often prominent. However, less than half of the tumors tested stained positive with S100. More work is needed before a cell of origin can be identified for these spindle cell tumors of blue-eyed dogs. Extension into the posterior uvea, vitreous base, or through the sclera into the episcleral connective tissue is fairly common; however, metastasis has not been recognized. We have seen two cases that have recurred within the scleral shell after an evisceration procedure. Almost all affected eyes have glaucoma secondary to tumor infiltration in the iridocorneal angle or interruption of aqueous flow for other reasons. Siberian Husky is the most common breed affected. However, that is almost certainly because it is the dog breed most commonly seen with blue eyes (1, 2).

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**AFIP Diagnosis:** Eye, anterior uvea: Spindle cell neoplasm of blue-eyed dogs, Catahoula Leopard Dog, canine.

**Conference Comment:** Conference attendees debated whether the lesion is proliferative or neoplastic. All agreed the lesion represents a nearly pure population of one cell type that overlays the corneal endothelium and obliterates the filtration angle, which are convincing characteristics of a neoplasm. The loosely arranged streams and bundles of spindle cells suggest nerve sheath origin.

Some attendees favored melanoma but did not have the benefit of immunohistochemistry prior to the conference. In this case, neoplastic cells are immunopositive for glial fibrillary acidic protein (GFAP) and negative for S-100 protein. GFAP immunoreactivity eliminates melanoma as a differential and supports the diagnosis of spindle cell tumor of blue-eyed dogs.

The cell of origin comprising spindle cell tumor of blue eyed dogs has not been determined; however, immunohistochemistry and cellular morphology suggest peripheral nerve sheath origin. Metastatic spread has not been described (3,4).

Spindle cell tumor of blue-eyed dogs occurs in dogs with poorly pigmented uveal tissue. In this case, careful examination reveals an exceedingly thin choroid with very little pigment. Within the retina there is loss of the neurons from the ganglion cell layer as well as blending of the inner and outer nuclear layers. Blending of the

nuclear layers occurs as a result of the loss of dendrites and axons which make up the inner and outer plexiform layers. Retinal degeneration, a thin choroid, and obliteration of the drainage angle by neoplastic cells indicate glaucoma.

Readers are encouraged to review WSC Conference 1/ Case 1 from the 2003-2004 academic year for another example of spindle cell tumor of blue eyed dogs.

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**CASE IV – N05-145 (AFIP 2984015)**

**Signalment:** 2.5 year old, spayed female Golden Retriever (*Canis familiaris*)

**History:** The patient presented for a 3 week history of vomiting and intermittent diarrhea, with development of anorexia and lethargy. On ultrasound of the abdomen, small intestinal loops were fluid-filled and dilated, with no apparent motility, and mesenteric lymph nodes were moderately enlarged. The ileus was unresponsive to prokinetic therapy or enteral feeding. Thoracic radiographs revealed alveolar infiltrates in the left and right cranial, and right middle lung lobes, suggestive of aspiration pneumonia. Due to the patient's lack of response to therapy, the dog was euthanized.

**Gross Pathology:** The patient is in poor body condition, with markedly depleted adipose stores and prominence of bony protuberances. There are multifocal loose

fibrous adhesions affecting several loops of mid-jejunum. Diffusely, the small intestinal serosa is discolored gray to red. The wall of the jejunum is flaccid and thin, and the contents are thick, pasty, and bright green. Mesenteric lymph nodes are moderately prominent.

Diffusely, the right middle lung lobe is dark red, firm, and consolidated, with fibrinous adhesions to the mediastinal adipose tissue. The remaining lung lobes are congested and moderately edematous. The heart appears slightly rounded. The mitral valve is mildly expanded by raised, 1-2 mm, glistening tan nodules (valvular endocardiosis).

**Laboratory Results:** Microbiology: Bacterial culture of the small intestine yielded light growth of *E. coli* and moderate growth of group D *Streptococcus*, which were judged to not be of clinical significance. Cultures of small intestine for *Nocardia* species, *Mycobacterium*, and *Bartonella* were negative. Fungal cultures of the small intestine were negative.

Virology: No viral etiology was isolated in specimen-infected MDCK cells from sections of small intestine, and no viral particles were found on electron microscopy of cell culture.

**Histopathologic Description:** Jejunum: Diffusely, throughout the small intestine, there is a highly cellular infiltrate within the wall of the small intestine which completely effaces the tunica muscularis. The infiltrate is often most intense in the circular muscular layer, and slightly less intense in the outer longitudinal muscular layer. The cellular infiltrate is composed of a mixture of lymphocytes and foamy macrophages in a loose, fibrillar stroma. There are scattered individual apoptotic cells and scattered cellular debris within the infiltrate. Scattered lymphocytes are present in the serosa. The muscularis mucosa remains unaffected in all sections examined. The mucosal lamina propria contains a mild to moderate cellular infiltrate composed predominantly of lymphocytes and plasma cells, with scattered eosinophils. Multifocally there are regions of mild edema in the lamina propria. The intestinal lumen contains degenerate cellular debris and numerous mixed bacteria.

**Contributor's Morphologic Diagnosis:** Small intestine, jejunum and ileum: Severe, diffuse lymphohistiocytic mural myositis; moderate, diffuse lymphoplasmacytic and eosinophilic enteritis.

**Contributor's Comment:** Various special and immunohistochemical stains were applied to sections of the intestinal lesion. No etiologic agents are identified on Gomori's methenamine silver (GMS), Ziehl-Neelsen acid fast (AFB), or Periodic acid Schiff (PAS); scattered mast cells are demonstrated by AFB.

Immunohistochemistry for lysozyme demonstrates strong intracytoplasmic immunoreactivity within the large histiocytic population within the mural infiltrate, as well as within scattered mature plasma cells and neutrophils. Approximately 30% of the small lymphocyte population exhibits positive immunoreactivity for CD79a, and moderate numbers of the small lymphocytes exhibit immunoreactivity for CD3, indicating a mixed lymphocytic population.

The cells comprising the infiltrate are well differentiated, with minimal anisocytosis and anisokaryosis, and it is a mixed cell population of histiocytes and lymphocytes, more characteristic of an inflammatory rather than a neoplastic process. Additional diagnostics failed to reveal an underlying etiologic agent, leading to consideration of an immune-mediated myositis.

Chronic intestinal pseudo-obstruction (CIPO) has been described as a phenomenon in humans in which enteric neuromuscular disease or visceral myopathy lead to a functional obstruction of the intestine with ileus, with no apparent underlying mechanical cause (1). A small number of case reports describe CIPO in dogs, with infiltration of the tunica muscularis by mononuclear infiltrates, with variable amounts of fibrosis and atrophy. There is replacement of much of the muscular tunic by connective tissue containing lymphocytes, macrophages, and occasional plasma cells and neutrophils (2, 3). Clinical signs include progressive weight loss, vomiting, ileus, and abdominal distention. The myositis centered on the muscular tunic in this dog is consistent with chronic idiopathic intestinal pseudo-obstruction syndrome. Lesions in this patient were widespread through the gastrointestinal tract, with less severe, presumably earlier lesions extending to the stomach and to the cecum and colon; the jejunum and ileum were most severely affected. There is not a remarkable amount of fibrosis in the intestinal sections in this patient, possibly related to duration of illness.

The underlying cause of CIPO in dogs has not been definitively established, although immune-mediated mechanisms remain under investigation. Full-thickness biopsies are critical in diagnosis of the disease. The therapeutic benefit of immunosuppression is uncertain, but may be beneficial, particularly in early stages of the disease.

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**AFIP Diagnosis:** Small intestine, tunica muscularis: Myositis, lymphohistiocytic, diffuse, severe, Golden Retriever, canine.

**Conference Comment:** The contributor provides an excellent review of an obscure but interesting condition. As noted by the contributor, the inflammation is centered on and limited to the inner and outer layers of the tunica muscularis. Attendees

agreed that such target organ specificity suggests an immune mediated disease. Likewise, the inflammatory cells are predominantly a mixture of lymphocytes and histiocytes which does not support an infectious or neoplastic process. Like the contributor, attendees did not recognize fibrosis and atrophy yet previous journal articles list them as prominent features of this disease.

Interestingly, a recent journal article describes autoimmune enteric-leiomyositis as a rare cause of CIPO in human infants. The article describes the histological hallmark as a dense T-lymphocyte infiltrate and degeneration of smooth muscle fibers within the muscularis propria of the intestinal wall. The article also points out the need for full thickness biopsies of the small intestine for definitive diagnosis (4).

Attendees debated whether or not enteritis is present. Most considered the mucosa, muscularis mucosa and submucosa as within normal limits. The moderator specifically noted that there is not a universally accepted reference range for numbers of lymphocytes, plasma cells and eosinophils in the intestinal mucosa and, that these cell types are normal inhabitants of the intestinal mucosa and submucosa. Without architectural damage to the intestine, one should not diagnose enteritis based upon these cell types alone; however, the presence of neutrophils should be recognized as an important indicator of inflammation.

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