

# **53<sup>rd</sup> AAVLD Diagnostic Pathology Slide Session**



**American Association of Veterinary Laboratory  
Diagnosticians  
Minneapolis, Minnesota  
Saturday, November 13, 2010  
3:00-6:30 PM**

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November 13, 2010 Minneapolis, Minnesota**

<b>Case #</b>	<b>Slide label</b>	<b>Presenter</b>	<b>Species</b>
1	AAVLD 2010 Case 16	<b>Jeff Hayes</b>	Canine
2	ADDL Purdue University AAVLD 2010 Case #2	<b>José Ramos Vara</b>	Guinea pig
3	VMDL U of Missouri-Columbia AAVLD 2010 Case #1	<b>Sean Spagnoli</b>	Chinchilla
4	AAVLD 2010 Case #3	<b>Scott D. Fitzgerald</b>	Canine
5	AAVLD 2010 Case #4	<b>Kevin Keel</b>	Frog
6	AAVLD 2010 Case #5	<b>Angela Pillatzki</b>	Canine
7	AAVLD 2010 Case #6	<b>Renate Reimschuessel</b>	Fish
8	AAVLD 2010 Case 7	<b>Shivaprasad</b>	Chicken
9	AAVLD 2010 Case 8	<b>Shivaprasad</b>	Chicken
10	AAVLD 2010 Case #9 U of GA Vet Coll Diagn Lab Tifton	<b>Marcia RS Ilha</b>	Duck
11	AAVLD 2010 Case #10	<b>Mahogany Wade-Caesar</b>	Equine
12	Kiupel AAVLD	<b>Matti Kiupel</b>	Canine
13	AAVLD 2010 Case #12 KSVDL	<b>Kyathanahalli S. Janardhan</b>	Gorilla
14	AAVLD 2010 Case #13	<b>Sandra Scholes</b>	Bovine
15	AAVLD 2010 Case #14	<b>Alexander Loretta</b>	Porcine
16	AAVLD 2010 Case #15	<b>L. L. Coffee</b>	Equine
17	AAVLD 2010 Case #17 OSU Vet Med Corvallis OR	<b>Rob J Bildfell</b>	Deer
18	VMDL U of Missouri-Columbia AAVLD 2010 Case #18 2010	<b>Gayle Johnson</b>	Feline

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<b>Case #</b>	<b>Presenter</b>	<b>Species</b>	<b>Diagnosis</b>
1	<b>Jeff Hayes</b>	Canine	Eccrine gland carcinoma
2	<b>José Ramos Vara</b>	Guinea pig	Teratoma
3	<b>Sean Spagnoli</b>	Chinchilla	Hepatic lipidosis/Exertional rhabdomyolysis
4	<b>Scott D. Fitzgerald</b>	Canine	Nephroblastoma
5	<b>Kevin Keel</b>	Frog	Protozoan infestation
6	<b>Angela Pillatzki</b>	Canine	Trypanosomiasis
7	<b>Renate Reimschuessel</b>	Fish	Crystal nephropathy
8	<b>Shivaprasad</b>	Chicken	Cutaneous mucinosis
9	<b>Shivaprasad</b>	Chicken	Avian encephalomyelitis
10	<b>Marcia RS Ilha</b>	Duck	Tuberculosis
11	<b>Mahogany Wade-Caesar</b>	Equine	Glycogen storage disease
12	<b>Matti Kiupel</b>	Canine	Canine distemper
13	<b>Kyathanahalli S. Janardhan</b>	Gorilla	Aortic dissection
14	<b>Sandra Scholes</b>	Bovine	Bone marrow failure
15	<b>Alexander Loretto</b>	Porcine	Mycotic pneumonia
16	<b>L. L. Coffee</b>	Equine	Congenital anomaly
17	<b>Rob J Bildfell</b>	Deer	Verminous pneumonia/ adenovirus infection
18	<b>Gayle Johnson</b>	Feline	Lymphoma

## **Eccrine Gland Carcinoma in a Dog**

**Jeff Hayes**

Animal Disease Diagnostic Laboratory, Ohio Department of Agriculture

**Signalment:** 5-year-old female large mix breed dog

**Clinical History:** Ulcerated tissue was present on the upper pad of the left forelimb, and it extended into the subcutis. The pad was surgically removed, but not all abnormal tissue could be removed. Carcinoma was suspected. Histopathology of formalin-fixed excised tissue was requested.

**Microscopic Description:** An infiltrative mass in the deep dermis is comprised of two cell populations within a moderate fibrous stroma that has abundant collagen and extensive lymphocytic and plasmacytic infiltrates. One cell population consists of acinar and ductular structures lined by low cuboidal cells with moderate amounts of eosinophilic cytoplasm and indistinct cell borders and oval to round nuclei that are either hyperchromatic or have stippled chromatin and a prominent nucleolus. Anisokaryosis is marked in this cell population, and mitoses are moderate, ranging from 0 to 5 per 400x field, mean of 2.5 in ten fields. The second population of cells consists of large polyhedral cells arranged as sheets in various regions of the mass. These cells have abundant eosinophilic cytoplasm, distinct cell margins, and large irregularly round vesicular nuclei with prominent magenta nucleoli. Mitoses in this population are low (none seen). Tumor emboli are noted in multiple lymphatic vessels in the dermis adjacent to the mass, which contains multiple areas of necrosis and mineralization. Areas of squamous metaplasia are noted, also, with small areas of polyhedral cells arranged as small sheets. Neoplastic cells extend to lateral and deep margins (incomplete excision).

**Morphologic Diagnosis:** Skin, eccrine carcinoma

**Comments:** Eccrine carcinoma is an uncommon malignancy in dogs and cats that requires knowledge of the site of origin of the tumor, i.e., from the footpad region. Differential diagnosis includes apocrine adenocarcinoma, acantholytic squamous cell carcinoma from pawpad epidermis, acantholytic variant of nailbed squamous cell carcinoma, and metastatic carcinomas, especially metastatic pulmonary adenocarcinoma in the cat.

### **References:**

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- Goldschmidt MH, Dunstan RW, Stannard AA, et al: 1998, Histological Classification of Epithelial and Melanocytic Tumors of the Skin of Domestic Animals, 2<sup>nd</sup> series, Vol. 3, p.32. Armed Forces Institute of Pathology, Washington, DC.
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## Ovarian teratoma in a guinea pig

*José Ramos-Vara, Margaret Miller*

Animal Disease Diagnostic Laboratory, Purdue University, West Lafayette, Indiana 47907

This skinny adult guinea pig was found abandoned with truncal alopecia and a palpable abdominal mass. Age was estimated at 3 years. Ovariohysterectomy was performed. The right ovary was enlarged at 3.5 cm x 4 cm x 3.5 cm. Numerous fluid-filled cysts up to 7-8 mm in diameter were evident on cross-section as well as areas of mineralization.

Microscopically, a few ovum-containing follicles are in the periphery of the mass, but most of the specimen lacks recognizable ovarian tissue and instead is composed of neoplastic tissue from all 3 germ layers. The endodermal component includes tubuloacinar glands (lobules of serous acini formed by pyramidal cells with bright eosinophilic cytoplasmic granules) and cystic spaces lined by respiratory type epithelium with numerous ciliated cells and mucus-filled goblet cells. The ectodermal component consists of neuroectodermal tissue with axons, glial cells, and neuronal cell bodies; no skin, hair follicles or cutaneous adnexal glands are observed. The mesodermal component consists mainly of fibrous tissue with scattered plates of well differentiated bone. No lesions were detected in the left ovary or in the uterus. A diagnosis of ovarian teratoma was made.

The presence of all 3 germ layers in a neoplastic gonadal mass was the basis for the diagnosis of teratoma. Reproductive tract tumors account for about 25% of neoplasia in the guinea pig. Though not considered common, teratoma is the most frequently reported tumor of the guinea pig ovary, occurring in juveniles and adults, and accounting for all but 6 of 29 ovarian tumors.<sup>2</sup> Interestingly, testicular teratomas seem not to have been reported in the guinea pig. One of two ovarian teratomas reported by Willis<sup>5</sup> spread to peritoneal surfaces. Of 10 cases of ovarian teratoma found at necropsy of about 13,000 guinea pigs over an 8-year period,<sup>4</sup> none had metastasized, though some cases had resulted in abdominal hemorrhage. Tissues from at least 2 germ layers were found in all 10 tumors; most tumors had all 3 germ layers, and nervous tissue tended to be the dominant ectodermal component. Nervous tissue also figured prominently as the ectodermal component in another case of ovarian teratoma that had spread to the peritoneal surface of the diaphragm.<sup>1</sup> Granulosa cell tumor is reported far less commonly than ovarian teratoma in guinea pigs;<sup>2</sup> however, because of its potentially cystic nature, it should be included in the differential diagnosis for an ovarian mass along with cystic rete ovarii.<sup>3</sup> Though distant metastasis has not been recorded in ovarian teratomas in guinea pigs, a few cases have seeded peritoneal surfaces. In this guinea pig, no recurrence or spread of the teratoma was clinically evident at 10 weeks after ovariohysterectomy. The alopecia had resolved, and the guinea pig had gained weight.

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4. Vink HH: Ovarian teratomas in guinea-pigs: a report of ten cases. *J Pathol* 1970;102:180-182
5. Willis RA: Ovarian teratomas in guinea-pigs. *J Pathol Bacteriol* 1962;84:237-239.

## **Hepatic Lipidosis and Exertional Rhabdomyolysis in a Chinchilla**

**Sean Spagnoli, Keiichi Kuroki**

University of Missouri Veterinary Medical Diagnostic Laboratory

A male, intact chinchilla of unknown age had been housed in a pet store for approximately four months, during which time it had been allowed out of its enclosure for approximately one half hour per day. Following its sale, the owners, whose household included children, reportedly allowed the animal out of the enclosure for a period of four hours. Two days after its sale, the animal was found dead in its cage and was presented for post-mortem examination. On gross examination, the sclera and internal fat stores were diffusely icteric. The liver was diffusely pale tan and friable. Sections of all liver lobes sank in 10% neutral buffered formalin.

Histologically, hepatocytes were diffusely swollen and contained one to multiple, variably sized, well demarcated, clear intracytoplasmic vacuoles that occasionally peripheralized nuclei. Sections of skeletal muscle from the pelvic limb revealed frequent, multifocal myocytes characterized by one or combinations of the following: hypereosinophilic sarcoplasm, fragmented or vacuolated cytoplasm, and loss of striations.

A diagnosis of diffuse, severe hepatic lipidosis was made based on the microscopic lesions in the liver, which explained the icterus observed grossly. The multifocal to coalescing acute myocyte necrosis and degeneration in the pelvic limb was interpreted as exertional rhabdomyolysis.

Hepatic lipidosis in chinchillas can result from prolonged negative energy balance, most commonly secondary to anorexia. Severe hepatic lipidosis can result in hepatocyte dysfunction and cholestasis, leading to liver failure and hepatic icterus. Exertional rhabdomyolysis, also known as capture myopathy, is a relatively common disorder of exotic and wildlife species, particularly large herbivores and avians, that is characterized by acute, widespread, multifocal to coalescing myocyte degeneration and necrosis following a highly stressful event. The pathogenesis is thought to be massive catecholamine and corticosteroid release due to stress, leading to peripheral ischemia. In this animal, the inciting stressful event was considered to be the sale to a family with a child. The stress of the sudden change in environment may have also caused a period of anorexia when, combined with the extended period of exercise reported in the history, could have produced a severe negative energy imbalance, leading to hepatic lipidosis. It is likely that the hepatic lipidosis and rhabdomyolysis collectively contributed to the death of this animal. While both hepatic lipidosis and exertional rhabdomyolysis have both been reported in chinchillas, the diagnosis of both of these diseases in a single animal with an explanatory clinical history provides for an illustrative and instructive example of stress-induced metabolic disease in small, exotic species.

## **An Intradural Extramedullary Spinal Cord Tumor (Nephroblastoma) in a Juvenile English Mastiff Dog**

*Scott D. Fitzgerald, Heather Tillman, Matti Kiupel*

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A 5-month-old, male English mastiff presented with bilateral posterior paresis of one month's duration. Radiographs failed to demonstrate a spinal lesion, and the dog did initially improve on corticosteroid therapy, but relapsed and was euthanized. The referring veterinarian suspected there was vertebral instability leading to chronic spinal cord trauma. On gross necropsy, when the spinal cord had been split, a small (10-12 mm diameter), round, firm tan mass was present on the dura at the level of lumbar vertebrae 1 and 2, which filled the vertebral canal and compressed the lumbar spinal cord.

Histologically, this well-circumscribed mass arose in the meninges, and compressed the adjacent spinal cord resulting in significant rarefaction and spongiosis. The mass was composed of cuboidal epithelial cells forming tubules and acini; a second population of papillomatous structures invaginated into cystic spaces resembling embryonic glomeruli; and a third population of cells separating the previously described structures composed of ovoid to fusiform cells with hyperchromatic nuclei, and indistinct cell borders consistent with blastema cells. There is scant fibrovascular stroma. Immunohistochemical staining for pancytokeratin (MNF116) stained approximately 20-25% of the tubular structures; the immunoreactivity was diffuse throughout the cytoplasm of epithelial cells lining tubular structures; while nearly 100% of the blastema cells had diffuse immunoreactivity to vimentin.

These rare spinal cord tumors are most frequently observed in the lower thoracic to lumbar cords of young (6 to 36 months) and predominantly large breed dogs. Histologically they resemble renal nephroblastomas, and have been variously referred to as nephroblastomas, ependymomas, medulloepitheliomas, and neuroepitheliomas. The immunohistochemical staining confirms they exhibit different populations of epithelial and mesenchymal cells. And there is at least one report of positive staining for Wilm's tumor gene product WT1, further strengthening the argument that these are nephroblastomas which arise in young animals from aberrant fetal rests of renal blastema cells.

### References:

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Summers BA, deLahunta A, McEntee, M, Kuhajda FP: A novel intradural extramedullary spinal cord tumor in young dogs. *Acta Neuropathol.* 75: 402-410, 1988.

SYSTEMIC INFECTION OF LARVAL RIVER FROGS (*RANA HECKSSCHERI*) BY A NOVEL ALVEOLATE

Kevin Keel<sup>1</sup>, Michael Yabsley<sup>1,2</sup>, Andy Davis<sup>2</sup> and John Maerz<sup>2</sup>

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On March 5, three river frog tadpoles were collected from a pond in south Georgia. They were brought back to the University of Georgia where they were housed individually. One died and the remaining two were moribund at the time they were submitted for examination. The live tadpoles were euthanized by an overdose of MS-222. The abdomens were swollen and the livers appeared to be enlarged. The livers and kidneys were diffusely tan. Adipose tissue was grossly evident.

Microscopically, both tadpoles were similarly affected. The livers were completely obliterated by sheets of bright-purple, spherical 0.35- to 0.52- $\mu$ m-diameter protozoa. The protozoa were loosely arranged along the remains of the hepatic cords and some were in aggregates that seemed to be limited by the cell membranes of dead hepatocytes. Large veins and some large bile ducts remained but all other tissue had been replaced by the pathogen. The protozoa also greatly expanded the renal interstitium and most tubules were necrotic and/or attenuated. Every tissue was infiltrated by the protozoa but not nearly to the extent that the liver and kidneys were invaded. Only a few could be identified in the central nervous tissue, the least affected tissue type. Epithelial layers were not invaded by the spores though they were abundant in the subcutis and the lamina propria of the gastrointestinal tract. A moderate number of encysted trematode larvae were also scattered throughout the subcutis, skeletal muscle and other tissues.

Phylogenetic analysis of the 18S rRNA gene of the pathogen indicated it was related to, but distinct from, several marine and freshwater alveolates which together form a sister clade to the genus *Perkinsus*.

The protozoan described in this case has been linked to massive mortality events among larval frogs of the genus *Rana* at several locations in the eastern United States. The source population from which these tadpoles were collected was obliterated.

Davis, A.K., M.J. Yabsley, M.K. Keel and J.C. Maerz. 2007. Discovery of a novel alveolate pathogen affecting southern leopard frogs in Georgia: description of the disease and host effects. *EcoHealth*. 4:310-317.

Green, D.E., K.A. Feldman and J. Wimsatt. 2003. Emergence of a *Perkinsus*-like agent in anuran liver during die-offs of local populations: PCR detection and phylogenetic characterization. *Proceedings of the American Association of Zoo Veterinarians*. 2003:120-121



## An Unusual Case of Myocarditis in an Iowa Puppy

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Department of Veterinary Pathology, Iowa State University, Ames, IA (Pillatzki, Fales-Williams) and  
Centers for Disease Control and Prevention, Atlanta, GA (Schmitz, Shieh)

Thoracic viscera from an 18 week-old, female, Alaskan klee kai puppy was submitted to the biopsy service in the Department of Veterinary Pathology at Iowa State University. The puppy had been purchased from a breeder in Texas and had arrived in Iowa approximately 1 week prior to presentation at the referring veterinary clinic. The owner reported progressive lethargy since arrival. Physical exam findings included an irregular heart beat on auscultation. Radiographs revealed pleural effusion and ascites and an ECG revealed an erratic heart rate and irregular QRS complexes. Microscopic lesions included multifocal to coalescing, transmural separation and individualization of cardiac myocytes by moderate numbers of macrophages and lymphocytes, fewer plasma cells and rare neutrophils. A moderate number of cardiac myocytes were shrunken and angular with loss of cross striations, fragmented, dark eosinophilic sarcoplasm and pyknotic nuclei. Many myofibers contained intracytoplasmic, round to oval pseudocysts measuring up to 40 µm wide by 100 µm long containing many round, 2 – 4 µm protozoal amastigotes characterized by a prominent, basophilic nucleus and a single, thin, elongate, dark basophilic kinetoplast. Protozoal organisms were non-immunoreactive for *Toxoplasma* spp. and *Neospora* spp. by immunohistochemistry. Formalin-fixed cardiac tissue submitted to the Centers for Disease Control was positive for *Trypanosoma cruzi* by immunohistochemistry and PCR testing.

American trypanosomiasis or Chagas' disease is caused by the hemoflagellate protozoan *Trypanosoma cruzi* which has an extensive host range including people and many domestic and wild animal species. The life cycle of *T. cruzi* includes fecal deposition of trypomastigotes by a reduviid vector (commonly known as the kissing bug/beetle) at the site where a blood meal is taken from a mammalian host. Trypomastigotes then spread hematogenously within the mammalian host eventually entering a variety of host cell types where they transform into amastigotes and replicate by binary fission. Intracellular amastigotes transform into trypomastigotes which are released from the host cell and enter the circulation where they can be taken up by a reduviid vector. Vector-associated trypomastigotes transform into epimastigotes, undergo binary fission, transform back into trypomastigotes in the vector's hindgut and are passed in the feces.

In the United States, American trypanosomiasis is endemic in the southern states extending from Maryland to California. Principal reservoir hosts include opossums, raccoons, armadillos and various mouse, squirrel and rat species and the most important reduviid vectors are *Triatoma sanguisuga* (eastern United States), *T. gerstaeckeri* (Texas and New Mexico) and *T. rubida* and *T. protracta* (Arizona and California). Clinically affected dogs can either exhibit acute and/or chronic syndromes. Dogs with acute disease are typically < 1 year old and present with sudden onset of clinical signs consistent with right-sided heart failure and /or arrhythmias which are both caused by acute myocarditis. Dogs which survive acute myocarditis may develop chronic myocarditis with progressive myocardial degeneration depending on the type of immune response elicited by the host. Additionally, dogs infected after 6 – 12 months of age often lack clinically evident acute disease, but may develop chronic myocarditis as well. Progressive myocardial degeneration eventually leads to biventricular dilative cardiomyopathy for which the exact pathogenesis remains unclear. Dogs with American trypanosomiasis can serve as both disease sentinels and reservoirs for human infections.

### References

1. Barr. Trypanosomiasis. In: *Infectious Diseases of the Dog and Cat*. 3<sup>rd</sup> edition, Greene (2006).
2. Cruz-Chan et al. 2009. Immunopathology of natural infection with *Trypanosoma cruzi* in dogs. *Vet Parasitol*. 162, 151-155.

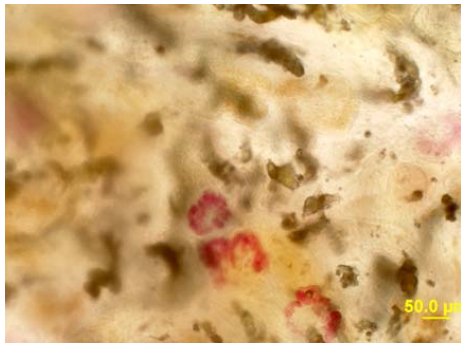
# Crystal Nephropathy in Fish Exposed to Melamine and Cyanuric Acid

*R. Reimschuessel*

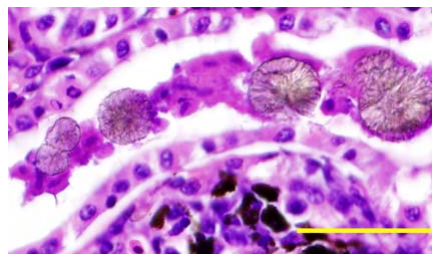
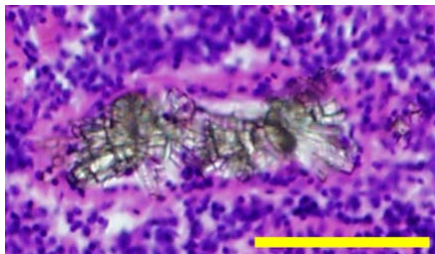
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During 2007, a large number of pets in the US, Canada, and South Africa died of renal failure caused by a crystal nephropathy which was induced by co-ingestion of melamine (MEL) and cyanuric acid (CYA) in contaminated pet foods. Most of the studies demonstrating crystal formation used very high dosages of MEL and CYA. Center for Veterinary Medicine conducted studies in 2007 which showed that fish and pigs also develop melamine cyanurate crystals after co-exposure. We recently conducted a NOAEL study in catfish and trout. The sensitivity of routine histopathologic examination and wet mount tissue samples was compared. Examining fresh tissue for crystal formation was much more sensitive than relying on specimens preserved in formalin and processed for H&E slides. The technique for preparing and evaluating wet mount preparations will be demonstrated. H&E sections will be examined.

The appearance of melamine cyanurate crystals can vary depending on the amount of time between sampling and their formation. Crystals may be present within tubule lumens, or embedded within the tubular epithelium. Some crystals are within protein casts.



*Wet mount of catfish kidney 1 and 3 days post treatment with 400 mg/kg bw Melamine+Cyanuric acid. Bars = 50 um.*



*Crystals within tubules, catfish day 1, salmon day 14 post treatment H&E Bars = 50 um.*

## **2010 AAVID Histopathology Conference.**

**H. L. Shivaprasad and Lucy Anthenill. CAHFS – Tulare and San Bernardino, University of California, Davis.**

### **1. S0902075. Cutaneous mucinosis in Brown Leghorn chickens.**

Fourteen 41 to 42 - week-old Brown Leghorn laying type chickens from a flock of 41,000 were submitted for necropsy with a history of drop in egg production of 20 %, decreased feed consumption, loss of weight and loss of feathers on the head, neck, keel and thigh regions. The incidence of skin and feather lesions in the flock was 70 %.

Supplementation of feed with vitamin A and other vitamins, biotin, and electrolytes did not improve the condition. Chickens of different ages on the same farm on the same feed were not affected. There was no history of spraying in the house and the chickens had received standard vaccinations. Postmortem examination of the submitted chickens revealed loss of feathers, scaly and dry skin with occasional petechiae. Most of the chickens were out of production as evidenced by inactive ovaries.

Histopathology of the skin from various regions of the body revealed accumulation of mucin-like material in the dermis that displaced the collagen. There was mild to severe primarily lymphocytic perivascularitis and vasculitis scattered through out the dermis. There was also acanthosis of the epidermis, hyperkeratosis and occasional cleft formation within the epidermis.

### **Morphologic Diagnoses:**

- 1. Severe diffuse cutaneous mucinosis with multifocal vasculitis, dermis**
- 2. Mild to moderate hyperkeratosis and acanthosis with occasional clefts, epidermis.**

**Comments:** Alcian blue stain of the skin was positive for mucin suggestive of cutaneous mucinosis. Cutaneous mucinosis is a condition where there is excessive deposition of mucin in the dermis replacing collagen. Mucin is a gelatinous substance composed of hyaluronic acid and dermatan sulfate. Mucinosis has been associated with multiple causes including genetics (Shar-pei dogs), hypothyroidism (myxedema), lupus, neoplasia (mastocytoma) and idiopathic. In humans it has been associated with numerous causes including infectious agents.

The cause of this condition in these chickens could not be determined. Routine bacteriology of the skin did not yield anything significant bacteria. Serology for various viruses (Newcastle Disease and Infectious Bronchitis) reflected vaccination titers and were negative for Avian Influenza.

## **2010 AAVLD Histopathology Conference.**

**H. L. Shivaprasad. CAHFS – Tulare, University of California, Davis.**

### **8. F0901597. Myocarditis in broiler chickens.**

Ten live 18 - day-old broiler chicks from a flock of 16,000 were submitted for necropsy. History of the flock included neurological signs in 2.5 % of chicks and increased mortality. Clinical evaluation of the live chicks revealed ataxia, lateral recumbency, occasionally opisthotonus, head shaking and tremors. Postmortem examination of the chicks did not reveal any gross lesions of diagnostic significance.

Histopathology of the heart revealed mild to severe infiltration of lymphocytes and a few plasma cells and macrophages and occasional foci of acute degeneration of myofibers scattered through out but most prominent in the auricles.

**Morphologic Diagnosis:** Subacute multifocal moderate to severe myocarditis.

**Etiology:** Avian Encephalomyelitis virus (Picornavirus: genus-*Tremovirus*)

**Comments:** Myocarditis is a common lesion found in chicks suffering from AE but is not reported commonly. Other lesions in the chicks included severe encephalitis characterized by neuronal swelling and central chromatolysis, gliosis and perivascular cuffing in the brain. Also, lymphocytic infiltration forming lymphoid nodules was prominent in the muscular layers of the proventriculus and gizzard, esophagus and intestine. Other lesions included pancreatitis, myelitis, neuritis, myositis, adrenalitis, and occasionally cataract formations in the eye.

Avian Encephalomyelitis (AE) is an infectious viral disease of chickens, turkeys, quail and pheasants caused by Picornavirus, genus *Tremovirus*. AE is characterized by neurological signs such as ataxia and paralysis in young chickens and transient drop in egg production in layers. The disease is often called “epidemic tremor” because of the characteristic head movements in young chickens. Survivors often develop cataracts later in life. The disease can be effectively controlled with proper vaccination of breeders. In this instance the breeders were not vaccinated properly. Serological titers for AE were positive in the presented chicks.

## Avian tuberculosis in two hooded merganser ducks (*Lophodytes cucullatus*)

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Two adult hooded merganser ducks (*Lophodytes cucullatus*) with a clinical history of lethargy and dyspnea were presented for necropsy. Both ducks were in fair to poor body condition. At necropsy, the inner surface of the air sacs was thick and covered by an irregular layer of yellow exudate. Lungs were diffusely consolidated, grey to red, and firm. Additionally one of the ducks had multifocal, random, firm, white, 1 to 6mm in diameter nodules throughout the hepatic parenchyma.

Microscopically, the wall of the air sacs was diffusely thick due to proliferation of fibrovascular tissue and infiltration of large numbers of epithelioid macrophages, plasma cells, and lymphocytes. A layer of fibrin, cellular debris, occasional heterophils, and numerous intralesional acid-fast bacilli were seen on the mucosa of the air sacs. The pulmonary parenchyma was replaced by multifocal to coalescing large areas of caseous necrosis, fibrosis, numerous epithelioid macrophages, plasma cells, and lymphocytes, and lesser heterophils. The mucosa of larger airways was necrotic or lined by hyperplastic epithelial cells with squamous metaplasia. Airways were filled with fibrin and cellular debris. Numerous intralesional acid-fast organisms were seen in the areas of necrosis and in the lumen of bronchi and parabronchi. Diffuse amyloidosis was seen in the spleen and liver of both birds. GMS, acid-fast, and gram stains failed to identify microorganisms within areas of necrosis in the liver of one of the ducks. No significant bacterial or fungal growth was seen with aerobic and fungal cultures. Mycobacteria culture of lungs yielded *Mycobacterium avium* subspecies *avium* (confirmed by IS901PCR).

From 2008 to 2010, five ducks, including these two hooded merganser ducks (*Lophodytes cucullatus*), two blue winged teal ducks (*Anas discors*), and a wood duck (*Aix sponsa*) died of avian tuberculosis in an aquarium exhibit. Both hooded merganser ducks had extensive involvement of the respiratory system affecting the air sacs and lungs. The other ducks had disseminated and intestinal lesions. In birds in general, avian tuberculosis has been considered as an intestinal disease. Some studies have demonstrated that respiratory organs are also often affected in disseminated avian tuberculosis and primary pulmonary disease may occur more frequently than primary intestinal disease in captive birds<sup>3</sup>. White-winged ducks (*Cairina scutulata*) with disseminated avian tuberculosis often have massive involvement of air sacs and lungs<sup>1</sup> as seen in these two hooded merganser ducks. Variation in susceptibility of affected organ systems, pathogenesis, and port of entry to *Mycobacterium avium* may exist between species of birds. A single case of avian tuberculosis in a hooded merganser duck reported in the literature describes lesions in the liver, lung, and pleura<sup>2</sup>. Probably hooded merganser ducks are susceptible to extensive respiratory involvement as seen with other avian species; however, the limited number of ducks necropsied (only 2) precludes further conclusion.

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## ***Glycogen Storage Disease in a Quarter Horse Foal***

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A 2-day-old female Quarter Horse with contracted tendons of the front limbs was nonambulatory, hypoglycemic and developed uncontrollable seizures despite anticonvulsant therapy. An irregular heartbeat was auscultated at birth and elevated liver enzymes were reported.

On gross examination, this 25 kg foal had mottled tan and pink mucous membranes. The trachea contained abundant red tinted foam and the lungs were diffusely rubbery in consistency, reddened, wet, and heavy with interlobular edema. Abundant red tinted fluid exuded from the airways on cut surface. The liver was mildly friable. Pale tan streaks were identified in the skeletal muscle of the left gluteal region.

Microscopically, 7 to 20 um diameter, spherical, amphophilic, homogenous, globules expand the sarcoplasm of 25-50% of the myocytes. Occasional myofibers were fragmented with slightly granular sarcoplasm and few vacuoles (degeneration). Similar intracytoplasmic inclusions were identified in the brain, heart, lung (airway epithelium), bile duct epithelium, and renal tubular epithelium. These intracytoplasmic inclusions stained positive with periodic acid-Schiff (PAS) stain. A diagnosis of Glycogen Branching Enzyme Deficiency (GBED) was made. A mild acute diffuse suppurative interstitial pneumonia (not thought to be associated with this condition) was also identified.

GBED is a fatal, inherited glycogen storage disease recognized in Quarter Horse foals and related breeds. These foals are unable to store sugar molecules because they lack the glycogen branching enzyme needed to store glycogen in its branched form. Affected animals exhibit generalized muscle weakness due to altered function (dysfunction) of the heart and skeletal muscles. Other clinical signs include hypothermia, elevated respiratory rate, contracted tendons in all four limbs, inability of the foal to stand up, fatality, and recent research suggests that at least 3% of abortions in Quarter horses are due to GBED.

This disease is not commonly diagnosed in the North Carolina Veterinary Diagnostic Laboratory system, as only 3 cases were reported in the past 8 years. To my knowledge, genetic testing was not performed on the first two cases; however in this case, a section of liver was referred to University of California-Davis for GBED testing. The foal tested homozygous positive (G/G) for the GBED gene.

Genetic testing to detect the GBE1 mutation can be performed at VetGen or the University of California Davis. The test can also be used to determine carrier status of stallions and mares. Through genetic testing, it is confirmed that the sire of this foal is a carrier of the Glycogen Branching Enzyme Deficiency gene.

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## **Necrotizing cryptitis in a dog: It has to be canine parvovirus, right?**

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**History:** A fifteen month old, female Shih Tzu with a history of mucopurulent ocular discharge, vomiting, hemorrhagic diarrhea and neurologic deficits was euthanized and submitted for a complete necropsy. The dog had recently been vaccinated against canine distemper virus and canine parvovirus.

**Gross Pathology:** The Shih Tzu weighed 4.25 kg and was in good body condition. The primary lesions were observed in the gastrointestinal tract. Throughout the length of the small and large intestines the luminal contents were semi-solid to loose and watery and variably hemorrhagic. The duodenal and jejunal mucosa had numerous scattered petechiae. No significant lesions were observed in other organs.

**Histopathology:** Sections of brain, liver, lung, heart, spleen, kidney, small intestine, pancreas, and eye were examined microscopically. Lesions were limited to the intestines. Within multiple intestinal segments there was enteritis that was centered on mucosal crypts. Affected crypts displayed a variety of changes including mild to marked ectasia, segmental to circumferential epithelial attenuation, loss, and regeneration. Crypt lumina variably contained necrotic cellular debris (cryptal abscesses) and/or amphophilic flocculent material (mucin). The villous lamina propria was infrequently expanded by hemorrhage. No infectious organisms or intracellular inclusions were observed. All other organs appeared microscopically unremarkable.

**Morphologic Diagnoses:** Small intestine: Subacute, segmental, hemorrhagic enteritis with crypt epithelial attenuation, necrosis, and regeneration

**Laboratory findings:** PCR results on sections of small intestine: Positive for canine distemper virus and canine parvovirus; Sequence analysis of PCR amplicons confirmed the distemper virus as a field strain; IHC results on sections of small intestine: Canine parvovirus antigen not detected, strongly positive for canine distemper virus antigen.

**Final Diagnosis:** Enteritis due to infection with canine distemper virus

**Comments:** Based on gross, microscopic and PCR results, a diagnosis of canine parvovirus infection was originally made. Especially since intestinal crypt necrosis is one of the hall marks of canine parvoviral disease and the absence of pulmonary or nervous tissue lesions made an infection with canine distemper virus less likely. However, immunohistochemical testing identified positive labeling for canine distemper viral antigen in areas of enteric inflammation, and specifically within epithelial cells of necrotic crypts and no labeling for canine parvovirus. Following sequence analysis of PCR amplicons, the canine distemper virus was confirmed as a field strain and not a vaccine strain, thereby confirming a post vaccination canine distemper field virus infection as the cause of the described lesions. This case illustrates that being a diagnosis on histopathology and PCR results alone without further knowledge of the vaccine status or sequence analysis of the PCR amplicons can easily lead to the wrong conclusion. Demonstration of viral antigen by FA or IHC within microscopic lesions should be the preferable diagnostic approach.

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## **Aortic dissection with cardiac hypertrophy in a Western Lowland Gorilla (*Gorilla gorilla gorilla*)**

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A 17 year and nine months-old male Western Lowland Gorilla was presented for necropsy. The gorilla was found dead in its holding and had no known clinical problems.

Grossly, the pericardial sac was filled with approximately 1.2 liters of frank blood with multiple large blood clots. The heart weighed 1.15 kilograms and the heart:body weight (HW:BW) ratio was 0.54. The entire ascending aorta and the aortic arch were circumferentially dilated and thin. The adventitial surfaces of these portions were mottled dark red and yellow (hemorrhage). The aorta was split into two layers with multifocal areas of hemorrhage in between. There were multifocal loose adhesions between the layers (Dissection of aorta). Approximately 2 cm from the aortic root, there was an approximately 2.5 cm linear tear in the outer layer of the aorta (from where it bled into the pericardium). The edges of this tear were slightly thick and dark red. Within the aortic lumen, there was a 0.7 cm tear at the level of origin of brachiocephalic trunk. The dissection of aorta involved the entire thoracic- and abdominal aorta. The splitting was less severe in the distal half of the abdominal aorta. The split extended into the brachiocephalic artery and subclavian arteries. The aortic endothelium was grossly normal. There were no other significant gross findings.

Microscopically, significant changes were present in the heart and aorta. In the left ventricular free wall and interventricular septum there was marked hypertrophy of the cardiomyocytes with karyomegaly. The nuclei ranged from 15- 30 microns in diameter. In the aorta, there was a split resulting in dissection of the tunica media. The dissected area contained fibrin and hemorrhage. Hemorrhage was also present around the tunica adventia. A diagnosis of Aortic dissection at the level of tunica media with aortic tear and hemopericardium was made and considered to be the cause of death.

Aortic dissection occurs when tunica media is separated by flow of blood through a tear in the intimal layer. In adult humans more than 90% of the aortic dissection cases are associated with hypertension and the frequency is more in males.<sup>3</sup> Aortic dissection has been well documented in captive Western Lowland Gorillas.<sup>1,2</sup> Like in this case, 6 of the 8 were males in the previous report. Five of the eight had evidence of ventricular hypertrophy with a mean HW:BW ratio of  $0.54 \pm 0.13$ . In the present case also, the ratio was 0.54 with histological evidence of ventricular hypertrophy. We do not know if this gorilla had hypertension or when the dissection initially started. In most instances inability to record blood pressure in non-anesthetized gorillas is a major limitation in monitoring and adopting measures to prevent aortic dissection. In humans, cystic medial degeneration, characterized by elastin fragmentation and areas devoid of elastin, is the most frequent histologically detectable change.<sup>3</sup> No such change was observed in the current case. In humans, DeBakey classification system classifies aortic dissection as type I (Originates in ascending aorta and propagates distally), type II (originates and confined to ascending aorta) and type III (originates in descending aorta). Type I and II are more common and dangerous. Using the human classification the present case can be classified as type I aortic dissection which resulted in hemopericardium and cardiac tamponade in this gorilla.

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## Bone marrow failure (bovine neonatal pancytopenia) in a young calf

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A one week old male Red Poll calf was euthanased on welfare grounds and submitted for necropsy examination. The calf was reported to be the second calf in the herd to have a progressive clinical history of apparent abdominal distress, intermittent collapse and recovery throughout the first week of life. External examination revealed dark faecal material adherent to the perineum and thighs. At necropsy, petechiae were present on the oral mucosa and ecchymotic and more extensive haemorrhages were detected in the subcutis, multiple skeletal muscles and on the serosal aspects of the rumen, small intestine and spleen. Blood clots were present in the peritoneal cavity, pericardial sac, pyloric lumen and distended the lumen of an approximately 1 metre segment of small intestine. Caecal contents were markedly blood-stained and the rectal faeces were formed and reddish black. Pale miliary foci were present in liver. *Listeria monocytogenes* was isolated from liver and brain.

Haemopoietic cell clusters and megakaryocytes were rare or absent in sternal bone marrow (A), compared with unaffected calves in which these cells were evenly distributed (B). Multiple variable sized foci of lytic and coagulative hepatocellular necrosis contained numerous gram positive bacilli, particularly at the margins, and karyolytic debris, however a neutrophil response was not apparent. Numerous gram positive bacilli were present in meninges and especially multifocally in subpial neuroparenchyma where they were associated with fibrinoid exudation, incipient neuropil necrosis and variable inflammatory response.

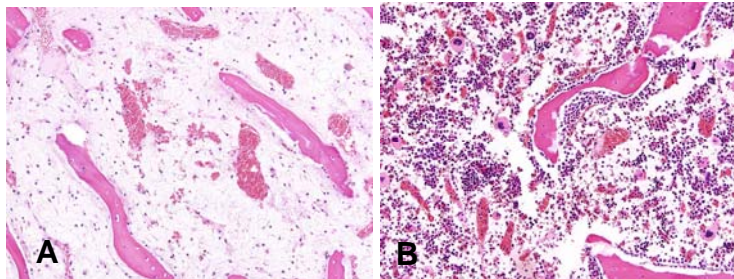
This lesion of trilineage hypoplasia (TLH; defined as less than 25% of cellular marrow occupied by megakaryocytes, erythroid and myeloid series cells) is, at the time of writing, considered to be typical of bovine neonatal pancytopenia (BNP; bleeding calf syndrome, 'blood-sweating disease', idiopathic haemorrhagic diathesis of young calves). This syndrome has been reported in several European countries since 2007/8 (Promed-mail, 2009). The cause/s have not been established. TLH is the histological criterion for diagnosis of aplastic anaemia. Viral, toxic, genetic and immunological (T-cell mediated) causes of aplastic anaemia are recognised; to date no convincing evidence of viral infection or potential toxic exposure has been found and a wide range of breeds are affected. There is some evidence for a colostral factor in the pathogenesis of this syndrome. An association between BNP and the use of a particular BVDV vaccine in the dam during pregnancy has been suggested but to date not proven. Two case reports of calves with similar clinical and pathology presentations from Canada (Ammann et al, 1996) and Japan (Shimada et al, 2006) precede the introduction of the implicated vaccine.

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Promed-mail archive number 20091207.4168, 07-DEC-2009

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## ***Conidiobolus incongruus* pneumonia in a sow**

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In October 2006, a 2-year old Yorkshire sow from a small swine herd in the State of California was submitted to the UC Davis CAHFS Davis Lab for necropsy. This animal had a clinical history of respiratory distress of 2 weeks duration. At necropsy, there was a hard, well-circumscribed, round mass approximately 30 cm in diameter replacing most of the left diaphragmatic lung lobe, and surrounded by a thick, fibrous, white, shaggy capsule. This mass had a homogeneous, light brown cut surface with some cystic, cavernous areas 0.5-2 cm scattered throughout. Attached to the external, capsular surface of the larger, firm mass there was a multilocular, elongated mass formed by many thin-walled cysts 0.5-1.5 cm in diameter filled with yellow or green colloid, or yellow friable material. Severe subpleural and interlobular edema were present in the remaining parenchyma from this lung lobe. Marked pleural effusion was also noted. Histologically, the larger lung mass consisted of abundant, well-vascularized, dense fibrous connective tissue with multifocal to coalescing discrete foci composed mostly of large numbers of necrotic eosinophils which surrounded numerous hyphae. These intralobular fungal elements, unstained on HE but GMS positive, were irregularly shaped, short or elongated, had non-parallel, thin walls, were infrequently and randomly branched, were occasionally septate, and had bulbous enlargements. This mass was walled off from an adjacent rim of pulmonary parenchyma by a thick, avascular fibrous capsule. DNA was extracted from a frozen sample of this lung mass, and a PCR reaction was run with primers directed to the 5' end of the 28S rRNA gene. Sequence analysis was performed on the amplified product which showed greater than 99% homology with the fungus *Conidiobolus incongruus*. No significant microorganisms were cultured from a fresh sample of this lung mass.

Conidiobolomycosis is a highly invasive zygomycosis caused by fungi of the genus **Conidiobolus**, class Zygomycetes, order Entomophthorales. It has been reported worldwide (e.g. in Australia, Canada, USA, India, Africa, and Brazil) as a cause of skin, respiratory, or generalized disease affecting humans and animals (sheep, dogs, horses, llamas, and deer). Species of **Conidiobolus** are **C. coronatus**, **C. incongruus**, and **C. lamprauges**. **Conidiobolus** is present in soil, decaying vegetation, and insects in tropical and subtropical areas. **Cases of zygomycosis have been reported in swine causing lymphadenitis, gastrointestinal disease, or disseminated infection, and include proven cases of mucormycosis by Absidia and Rhizopus, and presumed but not confirmed cases of entomophthoramycosis by Conidiobolus or Basidiobolus.** To the authors' knowledge, this is the first confirmed case of conidiobolomycosis in a **pig** with *Conidiobolus incongruus* being identified by PCR. A literature review failed to reveal any published reports of conidiobolomycosis by *C. incongruus* in swine. **In the US**, there are only two reports of **C. incongruus infection – one in a white-tailed deer (with a lung mass resulting in hypertrophic osteopathy), and one** in a granulocytopenic person (with pulmonary and pericardial disease).

**Acknowledgments:** We thank Dr. Robin L. Skillman, (veterinarian, Lincoln-CA) for submitting the case.

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## **Bilateral Ocular Anomaly in a Standardbred Foal**

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A female, neonatal standardbred foal presented to Cornell University for necropsy. The foal was born blind. The left eye was microphthalmic and the right was buphthalmic. The cornea were opaque, and a fundoscopic exam could not be performed. She was found dead in the stall that she shared with the dam.

Death was due to a concussive injury in the frontal bone that resulted in a severe, contrecoup, subdural hematoma around the brain stem. The eyes were not traumatized. Other than size, gross changes in both eyes were similar. There was deep stromal opacification, with a central pale tan zone surrounded by dark pink, dull, granular tissue that extended to the limbus. Multifocally, the corneal surface was elevated by botryoid clusters of well demarcated, variably sized, 0.3 to 1.0 mm diameter, white nodules and contained patches of brown, stippled discoloration. On section, the lens was absent and the anterior chamber was shallow. The iridial stroma was uniformly thick and ended abruptly at the ora serrata. A pupil and ciliary body were not detected.

Histologically, several structures were confirmed to be absent, including Descemet's membrane, the lens, ciliary processes and ciliary muscle. The superficial corneal epithelium formed infoldings into the corneal stroma that recapitulated primitive adnexal structures. Scattered throughout the corneal stroma were islands of well-differentiated sebaceous glands and adipocytes. The trabecular meshwork of the filtration angle was diffusely collapsed. The anterior surface of the iris was covered by an abnormal endothelium, continuous with the posterior corneal endothelium. At the center of the iris there was a well-demarcated proliferation of heavily vacuolated epithelial cells organized into small acini and interspersed with small caliber ducts lined by columnar, ciliated epithelial cells (lacrimonasal choristoma). The neurosensory retina was pathologically detached from the retinal pigmented epithelium and was multifocally degenerative.

Microphthalmia, aphakia, corneal and iridial choristomas, anterior segment dysgenesis, and persistent hyperplastic primary vitreous can occur in concert owing to shared pathways of organogenesis. The lens orchestrates migration, differentiation and proliferation of the neuroectoderm and is pivotal in normal development of the optic cup. Eyes that have dysregulated optic cup formation due to aphakia are prone to choristomas, anterior segment dysgenesis and congenital glaucoma. In this case, anterior segment dysgenesis contributed to unilateral congenital glaucoma due to incomplete remodeling of the filtration angle and an aberrant iridio-corneal endothelium. Similar anomalies have been reported in two other foals (1, 2). Further, among young domestic animals, foals are predisposed to corneal dermoids, which can be accompanied by anterior segment dysgenesis. Thoroughbreds and American thoroughbred crosses seem to be overrepresented in the literature, and a genetic predisposition has been postulated (3). This case illustrates the complement of ocular malformations that can accompany dermoids in foals. These lesions may be missed clinically due to total corneal opacification. Changes in this foal were more severe than those usually reported, which could be attributed to the absence of a lens. The primary cause of this group of malformations in foals remains enigmatic.

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## **Verminous pneumonia and adenovirus infection in a Black-tailed deer (*Odocoileus hemionus columbianus*)**

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This 3 month old female fawn was found dead in a suburban area of western Oregon. A field necropsy was performed and only formalin-fixed samples were collected for analysis. Body condition and hydration status appeared adequate. The small intestinal content was bloody. Tarry unformed feces were present in the rectum. No gross pulmonary lesions were observed.

Microscopic changes in this section of lung include randomly located granulomata, sometimes containing nematode eggs that range from primitive multicellular blastocyst forms to vermiform larvae. Close inspection of the endothelial cells of some of the medium-sized arterioles (and other vessels) sometimes may reveal amphophilic intranuclear inclusions, consistent with adenovirus. There is generally minimal vasculitis although endothelial cells tend to be hypertrophied throughout the section. Occasionally perivascular cuffs of a mixed population of leukocytes are seen.

This slide illustrates that adenoviral hemorrhagic disease (AHD) may not always cause the pulmonary edema often associated with fatalities due to this disease. Some cases seem to have more pathogenic effects on gastrointestinal vessels, causing death via massive hemorrhage into the gut. This infection is considered an important cause of mortality in cervid populations of California and Oregon, and has been identified in various other western locales. Within 5 days of this necropsy four other deer were found dead near this location but necropsies were not performed.

The eggs and larvae seen in this section are developmental stages of *Paraelaphostrongylus odocoilei*, an extrapulmonary lungworm common in Oregon. A gastropod intermediate host is required for the life cycle of this parasite. Adult worms are located in fascia and soft tissue of the limbs and trunk. Eggs are deposited into lymphatics and veins, with embolism to the lungs (and sometimes lymph nodes). Larvae develop and hatch in this location, then are coughed up and pass into the digestive tract. Prepatent period is thought to be about 2.5 months.

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## **Angiocentric Lymphosarcoma of T cell Origin as the Cause of Vestibular Disease in a Cat**

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A 12.5 year old, neutered male, domestic short haired cat presented with a 1 month history of lethargy and a 1 week history of right vestibular, facial and trigeminal nerve deficits. On examination, the cat also had multiple oral nodules, histologically diagnosed as lymphoma, and several enlarged lymph nodes. Magnetic resonance imaging of the brain revealed a right trigeminal nerve enlargement extending into the brain, as well as other small parenchymal lesions and meningeal infiltrates. Due to poor quality of life, the cat was euthanized. Fixed specimens were submitted for microscopic examination. On gross examination of the fixed brain, the root of the right trigeminal nerve was enlarged to roughly four times the diameter of the left trigeminal nerve. Within the brainstem, a firm, 2 mm wide bundle of pale tissue extended along the outer aspect of the right brainstem, near the trigeminal motor nucleus, vestibular nuclei and the reticular formation. Microscopically, the trigeminal nerve, trigeminal tract and adjacent brain were infiltrated by large polyhedral cells, located predominantly within and around small and medium-sized blood vessels. Infiltrates were surrounded by areas of edema and necrosis. Similar, less extensive perivascular infiltrates occurred in other regions of the brain and infiltrated the leptomeninges, usually staying close to vessels. Neoplastic cells were pleomorphic, with large oval to indented nuclei, dispersed chromatin and large nucleoli. Occasional binucleate cells were present. Cells had abundant amphophilic cytoplasm with well defined margins. In some areas, extensive tumor cell necrosis was apparent. Additional neoplastic infiltrates occurred in at least one lymph node (mitotic index 33), and in the oral cavity. These microscopic lesions are consistent with angiocentric or intravascular lymphosarcoma (lymphomatoid granulomatosis). Immunohistochemistry of involved brain revealed that neoplastic cells were CD3 positive. Angiocentric lymphosarcoma is an uncommon neoplasm that may be of either B or T cell origin in humans. B cell tumors have been associated with Epstein-Barr viral infection, but PCR failed to reveal herpesviral sequences in formalin-fixed, neoplastic tissue from this animal. Most canine cases involving the brain are derived from T cells (CD3 positive), and extraneural involvement is also often present. Two feline cases of angiocentric lymphosarcoma have been previously reported: one was characterized by severe pulmonary infiltrates, and the neoplasm could not be phenotyped immunohistochemically. The other cat presented for a recurrent subcutaneous mass and had multi-organ involvement at necropsy; tumor cells were positive with both B and T cell reagents. This is the first case of angiocentric lymphosarcoma reported in a cat with documented nervous system involvement.

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