48th AAVLD Diagnostic Pathology Slide Session
November 5, 2005; 3:30 - 6:00 pm
Hershey, Pennsylvania

Case/Page #	Base Slide Label	Presenter	Species
1	AHDL MSU 3022804	Scot Fitzgerald	Deer
2	Purdue ADDL	Ingeborg Langohr	Rabbit
3	Ohio ADDL 14601-05-4	Jeff Hayes	Dog
4	A05-60722-G	Elizabeth Howerth	Horse
5	AHDL MSU	Rebecca Smedley	Pigeon
6	AHDL MSU Kiupel	Matti Kiupel	Dog
7	Purdue ADDL	J. A. Ramos-Vara	Horse
8	Purdue ADDL	J. A. Ramos-Vara	Cat
9	A05-43550 UGA	Parag Chary	Cat
10	U of I VDL	Carol Lichtensteiger	Deer
11	05-09453 VMDL U of M-C	Gayle Johnson	Chicken
12	F0501188	H. L. Shivaprasad	Turkey
13	F0503370	H. L. Shivaprasad	Canary
14	T0501528 B 11	John Adaska	Cow
15	05-28466 U of Ga, Tifton	Alan Liggett	Dog
16	DA05-10432	Brad Njaa	Chicken
17	505-392	Brad Njaa	Dog
18	05-360 OSU VET MED	Rob Bildfell	Rabbit
19	U of I VDL	Lily Cheng	Dog

Presenters= names are in **bold** font on the abstract pages.

Meeting coordinators:

Lead: Rob Bildfell, Oregon State University
Assistant: Carol Lichtensteiger, University of Illinois

Cover image: Renal protothecosis, case #19.

48th AAVLD Diagnostic Pathology Slide Session
November 5, 2005; 3:30 - 6:00 pm
Hershey, Pennsylvania

Case/Page #	Presenter	Species	Diagnosis
1	Scot Fitzgerald	Deer	Dermatophilosis
2	Ingeborg Langohr	Rabbit	Cardiomyopathy
3	Jeff Hayes	Dog	Pug dog encephalitis
4	Elizabeth Howerth	Horse	Listerial encephalitis
5	Rebecca Smedley	Pigeon	Circovirus and adenovirus
6	Matti Kiupel	Dog	Hepatoid hyperplasia / dysplasia
7	J. A. Ramos-Vara	Horse	Photosensitization
8	J. A. Ramos-Vara	Cat	Probable Nocardial dermatitis
9	Parag Chary	Cat	Pulmonary fibrosis
10	Carol Lichtensteiger	Deer	Leptospirosis
11	Gayle Johnson	Chicken	Listera encephalitis
12	H. L. Shivaprasad	Turkey	Symmetrical encephalomalacia
13	H. L. Shivaprasad	Canary	West Nile virus myocarditis
14	John Adaska	Cow	Small intestinal hematoma
15	Alan Liggett	Dog	Mushroom poisoning
16	Brad Njaa	Chicken	Intraoccular ossification
17	Brad Njaa	Dog	Cutaneous blastomycosis
18	Rob Bildfell	Rabbit	Mxyomatosis
19	Lily Cheng	Dog	Systemic protothecosis

Presenters= names are in **bold** font on the abstract pages.

Meeting coordinators:

Lead: Rob Bildfell, Oregon State University
Assistant: Carol Lichtensteiger, University of Illinois

Dermatophilosis in a White-tailed Deer (*Odocoileus virginianus*)

Scott D. Fitzgerald[1], Thomas M. Cooley[2], Theresa Mosser,[1] Melinda Cosgrove[2]

[1] Diagnostic Center for Population and Animal Health, Michigan State University, Lansing, MI 48910, USA and [2] Michigan Department of Natural Resources, Lansing, MI 48910, USA

Dermatophilosis has been reported in many domestic and wild animals throughout the world since its first description in 1910. This case report describes a hunter-harvested wild white-tailed deer which presented with variably sized alopecic and scabby patches over its head and all four limbs. Initial differentials included mange and bacterial skin infection. Histopathology demonstrated marked hyperkeratosis, epidermal hyperplasia, thick crusts, and characteristic bacterial colonies. Bacteria consisted of long filamentous and branching forms, which formed parallel rows of cocci, and stained gram positive, consistent with *Dermatophilus congolensis*. Bacteriologic isolation confirmed the histologic diagnosis. This disease is widespread but infrequently reported. When first reported in deer dermatophilosis was limited to fawns and poor-condition possibly immunodepressed individuals, however, this deer was a healthy adult and this likely represents an incidental finding. Dermatophilosis is a zoonotic disease, and should be included in the differential diagnosis when exudative or crusting dermatitis is encountered in virtually any mammalian species.

Right Ventricular Cardiomyopathy in a Rabbit

Ingeborg Langohr[1], J. F. Van Vleet[1], E. J. Northrop[2]

[1]Animal Disease Diagnostic Laboratory, West Lafayette, IN
[2] Northrop Vet Clinic, Frankfort, IN

A 3-year-old mini lop doe, which died with no clinical signs other than lethargy, was submitted for necropsy to the Animal Disease Diagnostic Laboratory at Purdue University. The main gross lesions were indicative of congestive heart failure, including marked dependant edema, hydrothorax, hydropericardium, pulmonary edema and cranioventral atelectasis. The liver appeared slightly enlarged and had an irregular mottled pattern. Fibrin strands formed loose adhesions between the costal and visceral pleura as well as between the liver lobes. The lesions in the heart were restricted to the free wall of the right ventricular apex and outflow tract. The myocardium adjacent to the paraconal interventricular branch of the left coronary artery was firm and pale over an extensive (~4 x 1.5 cm) poorly delimited area. Microscopically, the heart lesions consisted of extensive interstitial fibrosis, associated with myocyte loss, vacuolar degeneration, atrophy and hypertrophy. An ultrastructural study on sections of right ventricle indicated that the sarcoplasmic vacuolation was due to distension of elements of the sarcoplasmic reticulum, consistent with hydropic degeneration. The sections of the left ventricle and interventricular septum were unremarkable. Histologic evaluation of lung and liver confirmed pulmonary edema and atelectasis, and centrilobular congestion with mild hepatocellular atrophy, respectively.

As the cause(s) and the pathogenesis of the myocardial alterations are unknown, the term Aright ventricular cardiomyopathy@ was used to name the disease in this rabbit. To the best of our knowledge, this is the first description of such a condition in this species. Myocardial lesions with similar distribution have been described in arrhythmogenic right ventricular cardiomyopathy (ARVC), Uhl=s anomaly, and right ventricular outflow tract (RVOT) tachycardia as seen in humans (all the above), dogs and cats (ARVC and Uhl=s anomaly), and mink (Uhl=s anomaly). ARVC is a disease in which an area of the myocardium is partially or almost entirely replaced by fat. Despite its name, there is a high prevalence of left ventricular involvement at a later stage of the disease. Uhl=s anomaly is characterized by a parchment-like appearance of the right ventricular wall due to apposition of the endocardium with the epicardium. The pathologic alterations of RVOT tachycardia is difficult to ascertain. The majority of myocardial specimens in patients with this condition are normal yet there may be a high incidence of wall motion abnormalities. Although ARVC, Uhl=s anomaly and RVOT tachycardia appear to be distinct clinical entities, some authors suggest they share a common pathogenesis of myocyte death (most likely by apoptosis). Uhl=s anomaly may be a more severe and rapidly progressive form of ARVC, and RVOT tachycardia may be the initial manifestation of ARVC in a minority of patients. In the rabbit, an injury restricted to the right cardiogenic fold was suspected; however, the etiopathogenesis in this case remains to be established.

Necrotizing Meningoencephalitis in a Pug Dog

Jeff Hayes[1], Douglas Poorman [2]

[1] Ohio Animal Disease Diagnostic Laboratory, Reynoldsburg, OH
[2] Petplex Animal Hospital, Buckeye Lake, OH

A 1.5-year-old intact female Pug dog had a history of sudden onset of signs including disorientation, inability to stand, petit mal seizures and increased respiratory rate. Treatment included oral phenobarbital and an injection of diazepam. Rabies vaccination status was uncertain. The dog died spontaneously after a one week course of illness; rectal temperature was >106° F the day prior to death. Necropsy was requested, with requests for ruling out rabies and canine distemper virus infections.

The dog weighed 19 pounds and was moderately overweight. No gross lesions were noted in the brain or internal organs. In each of two sections of cerebrum, there are moderate numbers of lymphocytes with fewer plasma cells, occasional histiocytes and infrequent neutrophils that expand the leptomeninges and cuff multiple vessels within both gray and white matter, and these leukocytes extend into the neuropil. In areas of inflammation, multiple neurons contain shrunken hypereosinophilic cytoplasm and hyperchromatic to pyknotic nuclei (neuronal degeneration and necrosis). Other alterations include proliferation of microglia and astrocytes (gliosis), neurons surrounded by glial cells (satellitosis), multiple variably sized clear vacuoles (spongiosis) and rarefaction of the neuropil (edema). Multiple vessels in areas of inflammation are lined by plump endothelial cells whose nuclei protrude into the lumen (endothelial cell hypertrophy). In one area of the section presented, there is extensive segmental necrosis of neurons within the hippocampus, with no accompanying inflammation. Immunohistochemistry for cell markers revealed a mixed perivascular mononuclear cell population consisting of approximately 50% T-lymphocytes (CD3+) and 30-40% B-cells (CD79a+). Two small focal areas of acute hemorrhage are the only changes within a section of brainstem. No significant microscopic lesions are present in sections of midbrain or cerebellum. No microorganisms (virus inclusions, bacteria, fungi, protozoa or nematode larvae) are observed in any section.

The morphologic diagnosis for cerebral lesions was marked, multifocal lymphoplasmacytic and histiocytic meningoencephalitis, with neuronal degeneration and necrosis, gliosis and spongiosis.

Differential diagnosis for inflammatory lesions included rabies, canine distemper virus (CDV), canine herpes virus type 1 (CHV-1), west Nile virus (WNV), toxoplasmosis, neosporosis and mycotic agents. Rabies, CDV and CHV-1 were discounted by immunofluorescence, and PCR assays were negative for WNV and CHV-1 nucleic acid. Virus isolation attempts to detect CDV were negative. Fungal or other organisms were not detected by Gomori=s methenamine silver (GMS) or Giemsa stains. Immunohistochemistry did not detect antigens of *Toxoplasma gondii* or *Neospora caninum*. Differentials for laminar cortical necrosis included exposure to heavy metals and ischemia secondary to hypoxia during seizure episodes. No elevations in metals including lead, arsenic, cadmium, mercury, boron, cobalt and chromium were detected in a section of liver.

Signalment, clinical signs, nature and distribution of microscopic lesions in the brain and other findings are consistent with a diagnosis of Pug Dog Encephalitis. This is a severe necrotizing meningoencephalitis of young Pug dogs of either sex that is of unknown origin, although one recent report suggests the pathogenesis may involve an autoimmune mechanism. It has been reported in the United States (1989, 1991), Japan (1994), Germany (1996), Switzerland and Italy (1998) and France (2000), and is also known to occur in Canada, Australia and New Zealand. Similar lesions have been reported in other small dog breeds including Maltese, Pekinese, Shi-tzu, Chihuahua and Yorkshire terrier.

1. Cordy DR and Holliday TA. Vet Pathol. 26:191-194 (1989).
2. Uchida K, Hasegawa T, Ikeda M et al. Vet Pathol. 36:301-307 (1999).
3. Kobayashi Y, Ochiai K, Umemura T et al. J Comp Path. 110:129-136 (1994).
4. Beltran WA and Ollivet FFM. J Small Animal Practice. 41:161-164 (2000).
5. Summers, Cummings, de Lahunta. *In:* Veterinary Neuropathology, pp. 111-115. Mosby-Year Book, St. Louis, MO (1995).
6. Jubb, Kennedy, Palmer. *In:* Pathology of Domestic Animals, Volume I, pp. 425-426. Academic Press, San

Diego, CA (1993).

Listerial Encephalitis in a Horse

Parag Chary[1], **Elizabeth W. Howerth**[1], W. Roberts[2], and M. Bonner[3]

[1] Department of Pathology, [2]Athens Diagnostic Laboratory, and [3]Department of Large Animal Medicine, College of Veterinary Medicine, University of Georgia, Athens, GA 30602

A 20 yr old American Saddle Horse gelding died after 8 days of showing nervous signs that included depression, ataxia, deficits of glossopharyngeal, facial, hypoglossal, and vestibulocochlear nerves, difficulty chewing and swallowing, hyperesthesia, and nystagmus. Atrophy of the temporalis muscles was present on physical exam 4 days after the nervous signs began.

At necropsy, the termporal muscles were atrophied and the brain and spinal leptomeninges were very congested.

In brain, significant lesions were confined to the medulla. Multiple to coalescing foci of neutrophilic infiltration with parenchymal liquefaction were present. These areas mostly involved ganglia where there was neuronal necrosis and neuronophagia. A few slender bacterial rods, that were gram-positive on a Lillie-Twort stain, were present in these foci and scattered throughout the neuroparenchyma. In addition, there were multiple foci of neuroparenchymal hemorrhage. In trigeminal ganglia, there were small infiltrations of lymphocytes (B and T cells on immunohistochemistry). There were no significant changes in spinal cord or cauda equina. Scattered myodegeneration in the temporal muscles, centrilobular hepatic atrophy and lipidosis, and mild multifocal necrosuppurative hepatitis were also seen microscopically.

FA tests were positive for *Listeria* and negative for rabies. Culture to determine the species of *Listeria* is pending.

The final diagnosis in this case was listerial encephalitis and trigeminal ganglionitis. *Listeria monocytogenes*, the usual species of *Listeria* associated with disease in domestic animals, is a gram-positive bacillus, which can be a commensal organism of clinically normal animals. It may be present in large numbers in the feces of ruminants and is quite resistant to environmental degradation. Listeriosis most frequently occurs in ruminants, and occurs as three different syndromes: encephalitis, septicemia, and abortion. Of these three forms septicemia is most frequently reported in horses in the literature; listerial keratitis in horses has been reported recently.

Listerial encephalitis occurs most often in adult animals and primarily in ruminants; it is infrequently described in other species. It is believed that the organism spreads to the brain by local invasion of cranial nerves, particularly the trigeminal nerve, rather than hematogenously. *Listeria monocytogenes* is able to polymerize actin into 'comet tail' structures and move within the cytosol of infected cells which may account for it ability to travel from the branches of cranial nerves in the oral mucosa to the brain stem. Trigeminal ganglionitis and caudal brain stem lesions in this case would support a similar pathogenesis in this case.

References

1. Gudmundsdottir KB, Svansson V, Aalbaek B, Gunnarsson E, Sigurdarson S. *Listeria monocytogenes* in horses in Iceland. Vet Rec. 2004 Oct 9; 155(15):456-9.

2 Jose-Cunilleras E, Hinchcliff KW. *Listeria monocytogenes* septicaemia in foals.Equine Vet J. 2001 Sep; 33(5):519-22.

3. Sanchez S, Studer M, Currin P, Barlett P, Bounous D. Listeria keratitis in a horse. Vet Ophthalmol. 2001 Sep; 4(3):217-9.

Pigeon Circovirus and Adenovirus in a 3 Month Old Racing Pigeon

Rebecca C. Smedley, R. Fulton, M. Kiupel

Diagnostic Center for Population and Animal Health, Michigan State University, E. Lansing, MI

Signalment and clinical history: A 3 month old racing pigeon with a history of Agoing light@, anorexia, and watery stool was submitted dead for necropsy to the DCPAH. Similar signs were observed in 15 out of 80 racing pigeons that had been kept in the same enclosure. A few of the birds would not walk.

Gross and microscopic lesions: A small amount of yellow, liquid, bilateral, nasal discharge was noted. There were no other significant gross lesions. Microscopically, sections of bursa were characterized by moderate to marked centrofollicular depletion and numerous large, basophilic, intracytoplasmic, botryoid inclusions within histiocytes consistent with pigeon circovirus inclusions. Sections of small intestine exhibited mild to moderate crypt necrosis. Scattered crypts were mildly dilated and contained small amounts of necrotic cellular debris. Occasional crypts were mildly attenuated but most were moderately hyperplastic. Small numbers of crypt epithelial cells and very occasional villous epithelial cells contained amphophilic to basophilic intranuclear inclusion bodies that filled the entire nucleus. These inclusions were suggestive of adenovirus inclusions.

Morphologic diagnosis:
1. Bursa: Moderate to marked, diffuse, lymphoid depletion with numerous intrahistiocytic, intracytoplasmic, basophilic, botryoid pigeon cirocovirus inclusions.
2. Small intestine: Mild to moderate, multifocal, crypt necrosis with intranuclear amphophilic to basophilic inclusion bodies suggestive of adenovirus.

Discussion: AGoing light@ syndrome in pigeons was originally attributed to infection with pigeon adenovirus (PAV); however, many cases actually involve dual infection with pigeon circovirus (PiCV) and PAV. Circoviruses are small, non-enveloped, icosahedral, DNA viruses (15 to 24 nm in diameter). PiCV infects lymphocytes and macrophages, causing interference with antigen processing and immunosuppression in young birds. Basophilic, botryoid, 5-25 µm in diameter, intracytoplasmic inclusions are found within histiocytes and epithelial cells exclusively in primary and secondary lymphoid organs. Clinical disease results from secondary bacterial, viral, fungal, or parasitic disease. The pigeon presented here had concurrent PiCV and PAV infections as well as severe pulmonary aspergillosis. Usually, PAV does not cause lesions or clinical disease in pigeons. However, under stress conditions and with concurrent PiCV infection, PAV can be associated with two clinical disease entities: classical adenovirosis and necrotizing hepatitis. It is not known whether these disease entities are caused by the same or different types of adenoviruses. Adenoviruses are non-enveloped, icosahedral DNA viruses (65 nm in diameter). In classical adenovirosis, large, basophilic, intranuclear inclusions (often surrounded by a halo) are noted within the intestinal epithelial cells or within hepatocytes. In this pigeon, adenovirosis resulting in crypt necrosis was most likely secondary to immunosuppression caused by PiCV. Dual infection with both of these viruses may be more common than previously reported and should be considered in pigeons with Agoing light@ syndrome. There are no specific treatments or vaccines for either PiCV or PAV. Immunity to both viruses develops within a flock over approximately a two year period, but initial concurrent infections can cause devastating losses.

1. Abadie, J., Nguyen, F., Groizeleau, C., Amenna, N., Fernandez, B., Guereaud, C., Guigand, L., Robart, P., Lefebvre, B., Wyers, M. 2001. APigeon Circovirus infection: pathological observations and suggested pathogenesis.@ Avian Pathology 30: 149-158.
2. Smyth, J.A., Carroll, B.P. February 18, 1995. ACircovirus infection in European racing pigeons.@ The Veterinary Record 136, 173-174.
3. Vereecken, M., de Herdt, P., and Ducatelle, R. 1998. AAdenovirus infections in pigeons: a review.@ Avian Pathology 27:333-338.
4. Woods, L. W., Latimer, K. S., Barr, B. C., Niagro, F. D., Campagnoli, R. P., Nordhausen, R. W., and Castro, A.E. 1993. ACircovirus-like infection in a pigeon.@ J Vet Diagn Invest 5:609-612.

Hepatoid Gland Hyperplasia with Post-Surgical Dysplasia in a Dog

Matti Kiupel[1], D. A. Rehagen[1] 1, M. Ritter[2], P. Dominguez[2], B. A. Steficek[1]
Diagnostic Center for Population and Animal Health[1] and Veterinary Teaching Hospital[2], Michigan State University

Signalment and history: A 14 year old spayed female Tibetan Spaniel dog was presented to the Veterinary Teaching Hospital at Michigan State University with a 5-cm mass in the right anal sac and a 3-cm mass at the right side of the tail base. A punch biopsy of the mass at the base of the right side of the tail was performed to exclude a malignant neoplasm and to determine the extent of future surgery. The biopsy from the right tail base mass was composed of closely packed, polygonal shaped cells with distinct cell borders. The cells contained abundant eosinophilic, cytoplasm with round, heterochromatic nuclei containing stippled chromatin. Mitoses were rare (less than 1/HPF). There was extensive infiltration by numerous neutrophils throughout the mass as well as multifocal areas of necrosis. The histiologic findings were most consistent with hepatoid (perianal, circumanal) hyperplasia or adenoma (the size of the biopsy precluded a more accurate distinction) with marked suppurative inflammation. Following this diagnosis, the anal sac mass and the right tail base mass were surgically removed 5 days after the original biopsy had been taken. Clinical examination of the dog 2 months after successful surgery was unremarkable and there has been no evidence of local recurrence or distant metastases.

Microscopic lesions: The lesion in the right anal sac was consistent with an anal sac rupture with severe, locally extensive, chronic-active, granulating, pyogranulomatous and lymphoplasmacytic cellulitis. The submitted slide contains the original punch biopsy of the hepatoid gland (tail base) and sections of the surgically removed gland. Sections of hepatoid gland were characterized by multilobular glandular hyperplasia with a focal area of sharply demarcated tissue loss rimmed by granulating fibrosis. This area corresponded to the previous punch biopsy site. The reactive stroma surrounding the surgical site extended into the surrounding glandular tissue separating and isolating gland lobules. There were multiple islands of glandular epithelial cells that had undergone dysplastic changes. Furthermore, individualized dysplastic epithelial cells, which appeared to arise from the adjacent circumanal glands were noticed throughout the reactive stroma. These cells were polygonal with scant amphophilic cytoplasm and plump vesiculate nuclei containing prominent nucleoli. In areas of dysplasia 1-2 mitoses/HPF were observed. The previous biopsy site was not contained within submitted tissue sections.

Morphologic diagnosis: Hepatoid (perianal, circumanal) gland hyperplasia with previous biopsy site surrounded by granulating fibrosis and glandular epithelial cell dysplasia.

Discussion: This case was of interest to us due to the unusual dysplastic changes observed in the hepatoid gland epithelial cells following surgical manipulation. It is rare that we, as veterinary pathologists, have the opportunity to examine the effect of surgery on glandular tissue within a 5 day time period. When the slide with the dysplastic changes was examined by a number of pathologists that had not been provided a complete history, a hepatoid gland carcinoma was discussed as one of the differential diagnoses. However, 2 months after surgery the dog shows no clinical evidence of a malignant neoplasm. It has been documented in rats that stress and surgery-induced suppression of natural killer cell activity is sufficient to cause enhanced tumor development. However, we found no reports of direct effects of surgery resulting in dysplastic changes or neoplastic transformation. Rather, in regards to mammary neoplasia, surgical reduction of glandular tissue has been associated with a decreased risk of carcinoma development.

Recurrence of benign proliferative hepatoid gland lesions is common following surgical removal. Difficulties related to complete removal of these tumors may account for some of these cases. Hyperplastic hepatoid glands are commonly observed in the periphery of hepatoid adenomas, and these hyperplastic glands may progress to form new tumors at the surgery site. Hepatoid glands in dogs have long been considered as modified sebaceous glands. Interestingly, recent studies suggest that the lobules of the hepatoid glands have many characteristics of the epidermis (a basal layer, a polyhedral or "spinous layer," a granular layer, and a keratinized layer) and should not be classified as glandular tissue. The cysts in lobules have been interpreted as "closed hair canals."

1. Atoji Y, Yamamoto Y, Komatsu T, Suzuki Y, Tsubota T. Circumanal glands of the dog: A new classification and cell degeneration. Anat Rec 1998, 250: 251-267. **2.** Ben-Eliyahu S, Page GG, Yirmiya R, Shakhar G. Evidence that stress and surgical interventions promote tumor development by suppressing natural killer cell activity. Int J Cancer 1999, 80: 880-888. **3.** Boice JD Jr., Friis S, McLaughlin JK, Mellemkjaer L, Blot WJ, Fraumeni JF Jr., Olsen JH. Cancer following breast reduction surgery in Denmark. Cancer Causes Control 1997, 8: 253-258. **4.** Brivio F, Gilardi R, Bucocev R, Ferrante R, Rescaldani R, Vigore L, Fumagalli L, Nespoli A, Lissoni P. Surgery-induced decline in circulating dendritic cells in operable cancer patients: a possible explanation of postoperative immunosuppression. Hepatogastroenterology. 2000, 47: 1337-1339. **5.** Goldschmidt MH. Sebaceous and hepatoid gland neoplasms of dogs and cats. Am J Dermatopathol, 1984 6: 287-293.

Photosensitization Secondary to Liver Disease in a Horse

J.A. Ramos-Vara[1], A. Sánchez[2], M.A. Miller[1]

[1] Animal Disease Diagnostic Laboratory, Purdue University, West Lafayette, IN. ramosja@purdue.edu
[2] Large Animal Veterinary Teaching Hospital, Purdue University, West Lafayette, IN

This haired skin and liver were from a 6-year-old, gelded Tennessee Walking horse with a history of liver disease and necrosis of lightly pigmented skin of the limbs and head. Gross findings included extensive necrosis and ulceration of lightly pigmented skin over canon bones, fetlocks, pasterns of both hind legs and the face. The conjunctiva and sclera were slightly yellow. The liver was enlarged and discolored brown to green with marked lobular pattern.

Microscopically, there was marked epidermal hyperplasia (acanthosis) and hyperkeratosis. Acanthosis ranged from regular to irregular, psoriasiform with prominent rete pegs. Hyperkeratosis was usually compact and orthokeratotic or parakeratotic. Apoptotic keratinocytes were rare. There were extensive areas of epidermal necrosis and ulceration with the defect covered by a thick layer of fibrin, cell debris, extruded collagen, mixed inflammatory infiltrates with a preponderance of neutrophils, and bacterial colonies. The underlying superficial dermis was edematous, with newly formed collagen and numerous immature vessels. The deep vessels, particularly veins but also some arteries, sometimes had partially organized thrombi associated with mural fibrosis and proliferation of adventitial vessels. The intima of affected veins had plump endothelial cells that in some areas appeared stratified. There was also basophilic amorphous material beneath the endothelium. Affected areas of the deep dermis also had extensive fibrosis. Dermal inflammation was mild and mostly mononuclear. The liver changes were periportal fibrosis, hepatocellular megalocytosis and giant syncytia, cholestasis, and bile duct proliferation. Megalocytosis was characterized by overall increase in the size of hepatocytes due mainly to enlarged nuclei. Syncytial cells contained numerous and irregularly distributed nuclei surrounded by abundant cytoplasm. Other changes were occasional centrilobular hemorrhage, multifocal necrosis, and fibrosis, and mild to moderate mononuclear inflammatory infiltrates, more common in the periportal and centrilobular areas. The hepatocytes of some lobules were vacuolated (usually a single and clear vacuole, lipid type). Small nodules of regeneration were rarely seen. Lesions were present in most hepatic lobules examined and were moderate to severe.

Morphologic Diagnosis: Necrotizing and ulcerative dermatitis, dermal vascular proliferation and thrombosis. Hepatopathy with hepatocellular necrosis, periportal fibrosis, bile duct proliferation, hepatocellular megalocytosis and syncytia.

Cutaneous lesions were most likely the result of photosensitization secondary to liver disease. The metabolism of chlorophyll ingested by herbivores produces phylloerythrin which is detoxified in the liver and excreted into bile. A damaged liver has reduced ability to process phylloerythrin which accumulates in tissues including skin, acting as a photosensitizing agent. Causes of secondary hepatic photosensitization in horses include the ingestion of plants from the *Senecio, Crotalaria, Brassica* and *Heliotropum* genera amongst others as well as blue green algae, neoplastic diseases, and other causes of diffuse, chronic, and severe liver damage. Although the specific cause of hepatic and cutaneous disease in this horse was not determined, prominent hepatocellular megalocytosis suggests pyrrolizidine alkaloid hepatotoxicity. The presence of hepatic giant cell syncytia is rare in the liver of horses. Giant cell (syncytia) hepatitis has been reported in young humans and rarely in animal species (equine fetuses, a foal, and 2 cats). In humans it has been associated with viral, autoimmune, and toxic conditions and appears to be more likely the result of fusion of the cytoplasm of several hepatocytes rather than incomplete division of proliferative hepatocytes. Syncytial hepatocytes and megalocytosis have been reported in horses surviving pyrrolizidine alkaloid toxicosis.

Cutaneous Nocardiosis in a Cat

J.A. Ramos-Vara, C.C. Wu**,** M.A. Miller

Animal Disease Diagnostic Laboratory, Purdue University, West Lafayette, IN.
Email: ramosja@purdue.edu

This section of haired skin is from an adult cat with a 4 cm long, ulcerated lesion on the dorsal aspect of its tail. Within the dermis and deeper tissues, but not involving the coccygeal vertebrae, there is a diffuse inflammation with ill-defined and confluent granulomas around mats of filamentous organisms that are weakly stained with hematoxylin (especially the periphery). Foreign body multinucleated giant cells and/or neutrophils surround these organisms. There is an outer layer of epithelioid macrophages. Mild fibrosis separates groups of granulomas. Microorganisms are rarely seen within multinucleated giant cells. These filamentous organisms are strongly Gram-positive and acid-fast (Fite-Faraco modification), and weakly PAS positive.

Diagnosis: Pyogranulomatous dermatitis and panniculitis with intralesional filamentous, Gram-positive and acid fast bacteria.

Differential diagnosis of this cutaneous lesion includes deep mycotic infections, mycobacteria, botryomycosis, and other deep bacterial infections (e.g., *Actinomyces* and *Nocardia* sp.). The morphologic and tinctorial characteristics of the organisms in this lesion are highly characteristic of *Nocardia*. The strong acid fastness of these microorganisms is unusual for *Nocardia,* which is usually only partially stained with the Fite-Faraco modification of the acid-fast stain. Sequencing of the 16s rRNA of nucleic acid extracted from paraffin sections yielded 99.5 % homology with *Nocardia* sp.

Nocardiae cause suppurative to granulomatous inflammatory syndromes in humans and animals, including cutaneous nocardiosis, pulmonary nocardiosis, solitary extrapulmonary nodules and disseminated nocardiosis. Cutaneous nocardiosis can clinically present as a mycetoma, cellulitis, subcutaneous abscesses, and granuloma. In this case, the presence of a nodular lesion is compatible with mycetoma (actinomycotic type). Several species of *Nocardia* have been reported in cats: *N. africana*, *N. asteroides*, *N. brasiliensis*, *N. tenerifensis*, and *N. otitidiscaviarum*. *Nocardia* sp. are found in the soil and transmission to animals or human beings is usually through skin trauma (either by contact with contaminated soil or by trauma with infected claws).

References:

1. Astudillo et al. (2001) Cat scratch responsible for primary cutaneous *Nocardia asteroides* in an immunocompetent patient. Br J Dermatol 145:667-670
2. Davenport and Jonson (1986) Cutaneous nocardiosis in a cat. J Am Vet Med Assoc 188:728-729
3. Edwards DF (1998) Actinomycosis and nocardiosis. In: *Infectious Diseases of the Dog and Cat*, Greene CE (ed.)., 2nd edit., pp. 303-313, W.B. Saunders Co., Philadelphia
4. Hattori et al. (2003) *Nocardia africana* isolated from a feline mycetoma. J Clin Microbiol 41:908-910 5. Luque et al. (2002) *Nocardia otitidiscaviarum* infection in a cat. Vet Rec 151:488
6. Tilgner and Anstey (1996) Nocardial peritonitis in a cat. Aus Vet J 74:430-432

Idiopathic Pulmonary Fibrosis-like Condition in a Cat

Parag Chary[1], Monica Kucher[2] and Jagannatha Mysore[1]

[1]Department of Pathology, CVM, The University of Georgia, Athens, GA 30602
[2] Good Hands Veterinary Hospital, Athens, GA 30606

A 10-year-old, Domestic Longhair, white-brown tabby, spayed cat was presented to the hospital with increased inspiratory/expiratory stridor and rapid heart rate. Radiographs of chest cavity revealed severe generalized interstitial pattern of radiopacity through all lung lobes, the cardiac silhouette was difficult to differentiate and there was a large amount of gas in the intestinal tract. Despite treatment, the animal died showing signs of severe dyspnea. The carcass was presented to Athens Veterinary Diagnostic Laboratory for necropsy. On gross examination, both the lungs had multifocal, small (<1mm), poorly circumscribed, pale to tan, firm nodules. Both kidneys had multiple old infarcts. Other organs were unremarkable.

Histologically, both lungs had multifocal to coalescing areas of marked alteration of terminal bronchioles, alveolar ducts and alveoli with predominant peribronchiolar distribution. Architectural alterations were characterized by fibroplasia and fibrosis of variable intensity within alveolar septa leading to disruption of bronchiolar wall and adjacent BALT with minimal to none inflammatory infiltration or exudates. Occasionally, areas of alveolar congestion/hemorrhage and hyperplasia of cuboidal type II pneumocytes were also noticed. In less affected areas, alveoli contained small numbers of attached and free macrophages and fibrinous exudate. Polarized light exhibited anisotropic birefringence, indicating fibrillar collagenous matrix within fibrotic areas. Representative lung sections were stained with Gomori's methnamine silver (GMS) and there was no evidence of fungi. Masson=s trichrome stain demonstrated collagenous matrix within fibrotic alveolar septa. Immunohistochemically, affected alveolar septa showed positive reactivity for α-smooth muscle actin (α-SMA).

Radiographic and histochemical evidence of extensive interstitial fibrosis resulting in alteration of pulmonary alveolar architecture, minimal inflammatory response, immunoreactivity of α-SMA within interstitia and type II pneumocyte hyperplasia are highly suggestive of idiopathic pulmonary fibrosis (IPF)-like condition.[1] IPF is a poorly understood disease in cats and has been recently documented in veterinary literature.[1,2] Current findings on etiopathogenesis indicate an association with prolonged inhalation of bentonite (cat litter) particles resulting in pulmonary fibrosis.[3] Current hypothesis suggests micro-injuries to alveolar epithelium resulting in promotion of deposition of extracellular matrix without concomitant degradation of the matrix. Further, defect in type II pneumocyte characterized by abnormal cytoplasmic lamellar body-like inclusions have also been demonstrated suggesting a heritable form of IPF. [2,4] Feline asthma and paraquat toxicity were also considered in our differential diagnosis.

Special Note: Digital copies of radiograph and color photomicrographs of this case report can be accessed at this URL: http://www.vet.uga.edu/vpp/mysore/index.htm {Accessed on 7/5/05}

References:

1. Cohn LA, Norris CR, Hawkins EC, Dye JA, Johnson CA, Williams KJ: 2004, Identification and characterization of an idiopathic pulmonary fibrosis-like condition in cats. J Vet Intern Med. 18:632-641.
2. Williams K, Malarkey D, Cohn L, Patrick D, Dye J, Toews G: 2004, Identification of spontaneous feline idiopathic pulmonary fibrosis: morphology and ultrastructural evidence for a type II pneumocyte defect. Chest. 125:2278-2288.
3. Williams KJ, Lewandowski RP, Hotchkiss JA, Patino LC: 2004, Microscopic and spectroscopic evidence that feline idiopathic pulmonary fibrosis is associated with bentonite pneumoconiosis. *In*: Proceedings of 55th Annual meeting of ACVP, Orlando, FL, pp. 575.
4. Williams K: 2005, Comparative pulmonary pathology in feline and human idiopathic pulmonary fibrosis. *In*:

Proceedings of 23rd ACVIM Forum, Baltimore, MD, pp. 762-764.

Leptospirosis in Farmed Deer

Carol A. Lichtensteiger[1], Carol W. Maddox[1], Cliff F. Shipley[2], and Virgina E. Bayer[1]

Department of Pathobiology and Veterinary Diagnostic Laboratory,[1] and Veterinary Clinical Medicine and Veterinary Teaching Hospital,[2] University of Illinois

Leptospira disease is seldom recognized in deer and seroconversion without disease is apparently common. We recently diagnosed, via lesions and PCR assay, acute leptospirosis in farmed deer. The case deer, from a herd of 28, was a 5 month old male fawn presented dead after a one day history of anorexia and deworming. The fawn was 15 kg body weight, small in stature, and emaciated. No gross changes were found to explain the death. Histologic changes in the kidney and liver included a moderate renal tubular necrosis with neutrolymphocytic tubulointerstitial nephritis and mild to moderate individual hepatocellular necrosis and drop out. With these changes, leptospirosis was a top differential. Immunohistochemistry for leptospira antigen was suggestive but the background staining hindered interpretation. Steiner=s silver stain labeled a few elongate structures in a rare tubule compatible with a spirochete. By PCR assay, leptospira genome was detected in both the kidney and liver. Other changes in the deer included reactive lymph nodes and splenic white pulp. The intestines were poorly preserved; however, the Strongyloides eggs found on fecal exam, the history of diarrhea in the herd, and the general poor body condition of the herd with adequate nutrition, suggested inadequate parasite control.

With enteric parasitism as a possible predisposing risk factor, the proximate cause of death was diagnosed as acute leptospirosis. A second fawn from this herd presented at the same time and, after three days of hospitalization, was euthanized. It had similar renal changes plus an acute superficial suppurative rumenitis and a secondary fungal hepatitis. The kidney, but not the liver, was PCR positive for Leptospira. A few months prior to the case deer, a fawn from another local herd had the hemolytic form of leptospirosis (PCV 7); renal changes were milder and both the kidney and liver were PCR positive for leptospirosis.

Deer are considered accidental hosts for Leptospira. The source of Leptospira in farmed deer are likely small mammals such as rodents, opossums, skunks, or raccoons, which are maintenance host for some serovars. The serovar infecting the case deer is unknown as neither culture isolates nor serum were available for testing. Of concern for both herd and public health is the unknown length of time deer shed Leptospira in the urine and no method to clear an infection.

References:

1. Bolin CA. (2003) Leptospirosis. In: Zoo and Wild Animal Medicine. Editors: ME Fowler and ER Miller. 5th edition. Saunders, St. Louis, MO. Chapter 68, pg 699-702.

2. Borst, LG and CW Maddox. (2004) Quantitative real-time PCR assay for the detection of pathogenic Leptospira spp. AVMA Meeting, Philadelphia, PA.

3. Mackintosh C, JC Haigh, and F Griffin. (2002) Bacterial diseases of farmed deer and bison. Rev Sci tech Off int Epiz 21:249-263.

4. Mèrien F, P Amouriaux, P Perolat, G Baranton, and IS Girons. (1992) Polymerase chain reaction for detection of *Leptospira* spp. in clinical samples. J Clin Microbiol 30:2219-2224.

5. Shotts EB. (1981) Leptospirosis. In: Disease and Parasites of White-Tailed Deer. Editors: FA Hayes, VF Nettles, and FE Kellogg. Southeastern Cooperative Wildlife Disease Study, University of Georgia. Chapter 13, pg 138-147.

6. Smythe LD, IL Smith, GA Smith, MF Dohnt, ML Symonds, LJ Barnett, and DB McKay. (2002) A quantitative PCR (TaqMan) assay for pathogenic *Leptospira* spp. BMC Infect Dis 2: article #13.

Focal Encephalitis in the Brainstem of a Chicken

Gayle C. Johnson, DVM, PhD, DACVP, Alex Bermudez, DVM, MS, DACPV

Veterinary Medical Diagnostic Laboratory, University of Missouri

An adult chicken was purchased at a livestock auction and brought into a flock of other birds. All of the purchased birds died of respiratory disease, which then spread to the owner=s laying hens. The animal had torticollis for about 1 week and then died. It was submitted to the VMDL for necropsy as representative of the respiratory disease problem observed in other members of the flock. Postmortem revealed a small area of localized softening in the brainstem. Microscopically, this area was characterized by focally extensive necrosis that was infiltrated by heterophils and surrounded by granulomatous inflammation. Perivascular cuffs peripheral to this lesion and in the leptomeninges were dominated by lymphocytes. Numerous small bacterial rods were seen in the center of the lesion. Immunohistochemical testing of fixed brain revealed Listeria within the lesion, both in the neuropil and within macrophages. Sections of liver, kidney, spleen, lung and intestine did not have any microscopic lesions. *Listeria monocytogenes* can produce necrosis of multiple visceral organs secondary to septicemia in newly hatched chicks, and in broilers it has been reported as a cause of low level disease (characterized by torticollis) and mortality. It is thought that the organism is introduced into the skin of the face traumatically and travels to the brain via cranial nerves, a pathogenesis analogous to mammalian encephalitic listeriosis. Although *Listeria monocytogenes* is a common organism in the intestinal tract of poultry, and has been recorded as causing disease in cattle that were stabled on recycled poultry litter, it is an uncommon cause of avian encephalitis that has been reported in chickens, turkeys and geese.

Symmetrical Encephalomalacia in Turkey Poults

H. L. Shivaprasad, R. P. Chin
CAHFS B Fresno, University of California, Davis, CA

Five and 7 day-old turkey poults were submitted with a history of sudden increase in mortality around 2 to 3 days of age. Clinical signs included down on their sides and unable to get up, roll on their backs, bob or roll heads and weakness. There were 25,000 poults in the house; 25 % exhibited clinical signs and there was 10 % mortality. Birds were treated with vitamins B, D and E but no improvement was seen. Postmortem examination of the birds did not reveal any significant gross lesions in the brain or other organs.

Histopathology of the brain revealed symmetrical malacia and necrosis of neurons (in some birds) in nuclei of the brain stem and cerebral cortex accompanied by perivascular cuffing and increased number of glial cells.

Comments: We have seen this condition sporadically in turkey poults over several years but a cause has not yet been determined.
On the above case, feed analysis revealed normal concentrations of vitamin E and salt. A water sample was analyzed for salt and it was found to contain 3600 ppm of chloride (normal, 14 B 200 ppm). Brains from the poults contained normal cholinesterase activity. Sodium concentrations in the brains of four birds were 1520, 1660, 1740 and 2080 ppm, respectively. In mammals, brain sodium of 1800 ppm and above is considered toxic but the toxic level in poultry is not known. Salt toxicity has been known to cause encephalomalacia in turkey poults but lesions like those observed in the nuclei of these birds have not been described. Salt toxicity in chickens can cause right heart failure and ascites.

Myocarditis associated with West Nile Virus in Canaries

H. L. Shivaprasad, P. Cortes
CAHFS ʙ Fresno, University of California, Davis, CA

Two canaries, approximately one-year-old and kept indoors, died in a span of two days. One bird appeared ill in the bottom of the cage. Except for mild hepatosplenomegaly there were no significant gross lesions upon necropsy. Microscopically, there was minimal to severe multifocal degeneration of myofibers and infiltration of lymphocytes and plasma cells scattered in the heart of the two canaries. Immunhistochemistry (IHC) for West Nile virus revealed antigen in the cytoplasm of myofibers, mononuclear inflammatory cells and endothelial cells of the heart and smooth muscle cells of the aorta. In addition mononuclear inflammatory cells in the sinusoids of the liver and spleen and renal tubular epithelial cells were also positive for WNV by IHC. West Nile virus was also confirmed in the kidney of the two canaries by PCR.

Comments: West Nile virus (WNV), a Flavivirus, caused serious disease in birds and mammals, including humans, during 1999 in the eastern USA. Since then, the disease has spread westward and arrived in California during 2003. WNV infection is well known in certain avian species such as Corvids and Raptors and it has been identified in about 300 species of birds thus far. However, very little is know about the epidemiology, susceptibility, clinical signs, pathology and diagnosis in such birds as canaries, finches, psittacines, *etc.* CAHFS has diagnosed WNV in more than 35 psittacines and in several canaries and finches during 2004 and 2005. One aviculturist lost 60 canaries out of 160 birds in one week; WNV was diagnosed in one canary submitted for necropsy. Lethargy, anorexia and loss of weight or sudden death were the primary clinical signs observed.

Jejunal Hematoma in an Adult Holstein Cow

John Adaska

CAHFS B Tulare, University of California, Tulare, CA

A 4.5 year old cow was presented to the California Animal Health and Food Safety Laboratory System BTulare laboratory alive but moribund. The animal had a history of being normal the previous day and then showing signs of melena, bloating and going down in less than 24 hours. Necropsy revealed the carcass to be in excellent postmortem condition and good nutritional condition. The carcass was markedly pale but not appreciably dehydrated. The rumen and abomasum both contained very large amounts of roughage feed and intermixed fluid. Within the mid jejunum, there was a marked, hemorrhagic expansion of the intestinal sub-mucosa which occluded the intestinal lumen. The hematoma within the submucosa was approximately 45 cm long. The small intestine proximal or oral to this point contained large amounts of watery, green material, while the distal portion of the small intestine contained watery, bloody fluid with moderate amounts of intermixed feed. The cecum and spiral colon both contained small amounts of very dry, hard, dark green feces. The uterus was well involuted.

Histologically, there was massive hemorrhagic expansion on the submucosa of the small intestine and necrosis of the mucosa that had been separated from the muscularis mucosa by the hemorrhage. The luminal surface of the hemorrhage has very large numbers of densely arranged neutrophils and occasional Aghosts@ of necrotic mucosal epithelium. The submucosa is expanded by abundant edema with frequent variable size fibrin aggregates, scattered, loosely arranged neutrophils and scattered small foci of hemorrhage. The submucosa also has small numbers of scattered lymphocytes and plasma cells and lymphatic vessels are often dilated. In adjacent areas where the mucosa has not been separated, the intestinal lamina propria has a slightly increased population of lymphocytes and plasma cells. Within the lumen of the intestine there is a mixed population of bacteria including large bacterial rods.

Grossly and histologically the lesions are consistent with jejunal hematoma or hemorrhagic bowel syndrome of cattle[1,2]. This is a poorly understood entity for which both *Aspergillus fumigatus* and *Clostridium perfringens* type A, with or without the beta2 toxin gene, have been suggested as possible causes. The acute nature of the clinical signs and the relative lack of significant inflammation in most cases are suggestive of an underlying non-infectious cause for this syndrome. In some cases of jejunal hematoma we have found small numbers of neutrophils and/or eosinophils in the submucosa adjacent to the area of massive hemorrhage. Rarely, we have seen single or small numbers of blood vessels undergoing fibrinoid necrosis in these same areas. Whether these are in response to the underlying cause or are secondary to the hemorrhage is not known.

References:

1. Dennison AC, Van Metre DC, Morley PS, Callan RJ, Plampin EC, Ellis RP. Comparison of the odds of isolation, genotypes, and in vivo production of major toxins by Clostridium perfringens obtained from the gastrointestinal tract of dairy cows with hemorrhagic bowel syndrome or left-displaced abomasum. J Am Vet Med Assoc. 2005 Jul 1;227(1):132-8.

2. Berghaus RD, McCluskey BJ, Callan RJ Risk factors associated with hemorrhagic bowel syndrome in dairy cattle. J Am Vet Med Assoc. 2005 May 15;226(10):1700-6.

Mycetismus (Mushroom Poisoning) in a Dog

Alan Liggett[1], Eloise Styer[1], and Larry Branch[2]

[1]UGA, College of Veterinary Medicine, Tifton Veterinary Diagnostic and Investigational Laboratory, Tifton GA 31793 and [2]Quailwood Animal Hospital, Tifton, GA 31793

The owner observed their 4 month old Yorkshire Terrier/Poodle puppy ingest a mushroom during a morning walk. Late that afternoon the puppy started vomiting and developed a loose malodorous stool. The puppy was presented to the referring veterinarian that evening. The puppy was alert and not dehydrated. It was treated conservatively for vomiting. She was found dead in her cage the following morning.

At necropsy, the stomach and upper small intestine were partially filled with mucoid opalescent material. Segmental congestion and hemorrhage were noted in the serosa of distal ileum. The mucosa of the terminal ileum was hemorrhagic.

Increased aptosis was noted in the superficial gastric mucosa. Sloughing of sheets of enterocytes, necrosis of tips of villi, and mucoid exudates were noted in the jejunum and upper ileum. Widespread necrosis with hemorrhage and collapse of the mucosa was present in the distal ileum. Necrosis of lymphoid cells was observed in all sections of the GI tract, especially in Peyer=s patches. Sloughed columns of necrotic enterocytes and mucus filled the colon. Dissociation of hepatocytes, with degeneration of most, and necrosis of random hepatocytes and Kuppfer cells was observed in all lobules. Edema and hemorrhage were noted around central veins, with sparing of the portal tracts. The hallmark lesion of nucleolar fragmentation (loss) was observed in hepatocytes and enterocytes.

Eight mushrooms were collected on a field trip to the site where the mushroom was ingested. All were identified as *Amanita bisporigera*. The mushrooms were dug with a trowel to preserve the cup and placed in individual paper bags. They should never be hand-picked or placed in plastic bags. *Mushrooms of North America* by Roger Phillips is an excellent reference for identification of mushrooms.

A. bisporigera contains amitoxins. *Alpha-aminitin* is the main constituent and it causes cell death by damaging nuclear RNA, resulting in cessation of protein syntehesis.

This case is unique in that the patient died within 24 hours of ingestion of the mushroom. This is interpreted to be the result of the comparatively large amount of mushroom ingested by a small dog. Most of the cases of mushroom poisoning diagnosed at our laboratory involved retriever puppies that usually had body weights ten times greater than that of this puppy.

References:

1. Liggett, AD, Weiss RW. Liver necrosis caused by mushroom poisoning in dogs. J Vet Diagn Invest 1:267-269, 1989.
2. Frazier KS, Liggett AD, Hines, ME, Styer EL. Mushroom toxicity in a horse with meningioamatosis. Vet Hun Toxicol 42:166-167, 2000.

Intraocular Ossification in a Mature Chicken

Brad L Njaa[1,2], B. Lucio-Martinez[2]

[1]Department of Biomedical Sciences, and [2]Animal Health Diagnostic Center (AHDC), Department of Population and Diagnostic Sciences, Cornell University, Ithaca, New York, 14853

Nine birds from a mixed-breed layer flock of 65 became blind the previous summer. The flock was otherwise healthy. Most affected birds were white rock chickens and by the time they were examined, they were about 1 1/2 years old. Gross lesions seen were characteristic of ocular Marek's disease or >Pearly-eye=.

Heads were collected from the spent hens and placed in Bouin=s fixative for further histologic characterization. Gross examination of one bird in particular resulted in eyes that appeared normal. Following removal from the skull, both eyes were extremely hard. Sectioning each through the optic nerve proved no easy task as the knife blade barely cut through the globes. All eyes examined grossly from the remaining three heads were normal.

Histologically, both eyes were similar comprising slightly shrunken globes with near complete filling of the posterior segment and anterior chamber by cartilage and bone. This intraocular infiltration compressed the lens rostrally against the endothelium of the cornea and distorted the retina diffusely. This proliferative bone appeared to either extend to or extend from the pigmented choroid. This choroid, in fact, appeared thicker than normal. Focally through the bony tissue were scattered collections of cells that were reminiscent of the retina. Bone marrow was present in a few locations. Also scattered throughout the intraocular mass were aggregates of pigmented cells. Retinal pigmented epithelium and the pectin could not be definitively identified in the sections examined.

Intraocular ossification in humans is most often associated with atrophic bulbi or phthisis bulbi as end stages to intraocular inflammation. The retinal pigmented epithelium (RPE) is a highly reactive tissue which may undergo hyperplasia or metaplasia. Intraocular ossification chickens has previously been reported[1] in a particular breed of chicken predisposed to retinal degeneration. Although the pathogenesis is not known, it was associated with progressive retinal degeneration with alternating hyperplasic and atrophic RPE and choroidal inflammation. First signs of ossification came at 18 months of age when vitreous came into contact with degenerate and atrophic retina. This exposure of the choroids to the vitreous facilitates migration of reparative fibroblasts that lay down matrix. Varying rates of ossification were thought to be associated with varying rates of retinal degeneration. Other birds from this flock with clinical blindness had evidence of retinal degeneration.

In the human condition, it is reported to occur without previous formation of cartilage, yet in the case presented today, there is evidence of initial formation of a cartilage model followed by ossification. This was a unique lesion to me but after speaking with a couple of seasoned veterinary ophthalamic pathologists, they assured me it was not uncommon. The interesting features about this case were the concurrent retinal degeneration in others in the flock. Whilst it has been previously reported, this is a nice specimen to add to your collection.

1. Kelley KC, Ulshafer RJ, Ellis EA. 1987. Intraocular ossification in the *rd* chicken. Av Pathol 16: 189-197.
2. Lee W.E. 2002. AThe Traumatised Eye,@ in Ophthalmic Histopathology, 2nd Edition, Springer-Verlag, London, England.
3. Shivarasad HL. 1999. APoultry Ophthalmology,@ in Veterinary Ophthalmology, 3rd Edition, Lippincott, Williams and Wilkins, Baltimore, MD, pp. 1201, 1202.

Cutaneous Blastomycosis in a Dog

Brad L. Njaa [1,2], M. Rozmanec[1], V. Patten[2], K. Ramstedt[3]

[1]Department of Biomedical Sciences, [2]Animal Health Diagnostic Center, Department of Population and Diagnostic Sciences, and [3]Department of Clinical Sciences, Cornell University, Ithaca, NY, 14853

Java, an approximately 7 year old, female-spayed, Laborador retriever dog from northern New York presented with a history of cyclic fever, lethargy, inappetence, and stiffness for 2 weeks duration. A respiratory stridor and cough were initially noted two weeks earlier. She had received repeated prescriptions of antimicrobials but here fever and symptoms never completely resolved. There had been no history of travel out of the area. Based on evaluation of the chest radiographs, it was determined that there were mild, diffuse, lung infiltrates. Blood was collected to rule out Ehrlichiosis, Leptospirosis, and Rocky Mountain Spotted Fever. In addition, tracheal washes and lymph node aspirates were performed for cytologic evaluation and culture. Urine and fecal samples were also submitted for culture. While the results from this testing were pending, Java was placed on broad spectrum antibiotics and systemic steroids in an attempt to control the fever, and coupage was employed to help manage the respiratory distress. During one of her coupage treatments, a veterinary student felt a cutaneous lesion rupture and exudate had to be cleaned from her ungloved hand and the skin over right thorax. Assuming this was a fistulous tract formed from a previous injection site, the area was surgically excised and submitted for histologic evaluation. A section of skin from this Afistulous tract@ was submitted for this case presentation.

The lesion consists of an area of cavitation within the panniculus or at the junction of panniculus and deep dermis. Within and peripheral to this area are abundant inflammatory cells including neutrophils, macrophages, lymphocytes, plasma cells, and multinucleate giant cells. This infiltrate often surrounds pannicular adipocytes, myocytes, and more superficial adnexal structures. Moderate numbers of yeast organisms are present within the pyogranulomatous exudates, either extracellularly or within the cytoplasm of macrophages. These organisms have a thick, partially refractile cell wall, contain a central, condensed nucleus, and range in diameter from 13 to 25 microns. On closer inspection and with the aid of special stains, broad-based budding is seen. Multifocally, the superficial dermis contains moderate numbers of lymphocytes, neutrophils, and macrophages that vary from perivascular to interstitial, accompanied by moderate superficial dermal edema, hemorrhage, and numerous, prominent capillaries that are often lined by very plump, reactive endothelial cells.

Following histologic confirmation of the etiology for the fistuluous tract, impession smears of the draining exudates were re-examined and few yeast organisms consistent for *Blastomyces dermatitidis* were seen. Several days later, and following subculture, the histologic and cytologic indentification was confirmed.

Blastomyces dermatitidis is a dimorphic saprophytic fungus that uncommonly causes systemic infections even in endemic areas (Java was from Lake Placid, a region considered endemic for Blastomycosis). Blastomycosis in the dog is typically a systemic infection, most often beginning as a pulmonary infection that subsequently disseminates throughout the body with preferred sites including eyes, bone, lymph nodes, subcutaneous tissues, brain, and testicles. Reportedly, up to 40% of dogs with Blastomycosis have skin lesions. Preferred sites include nasal planum, face, and clawbeds, however cutaneous involvement can be anywhere. While the case is not terribly unique, it is an excellent example of this disease and a wonderful example of how a rather simple instrument, the microscope, saved the day in the end. We reported this spectacular finding to the unsuspecting clinician at our morning read-outs in the clinic and immediately, Java was placed on antifungal medication (itraconazole) and maintained on steroids at the upper limit of anti-inflammatory dose range. Java continues to improve and it goes to show you that pathologists can save lives!

1. Legendre A.M. 1998. AChapter 59: Blastomycosis@, in Infectious Diseases of the Dog and Cat, 2nd Edition, Editor: Craig A. Greene, W.B. Saunders Company, Philadelphia, PA.
2. Scott DW, Miller WH, Griffin GE 2001. AChapter 5: Fungal Skin Diseases@, in Muller and Kirk=s Small

Animal Dermatology, 6th Edition, W.B. Saunders Company, Philadelphia, PA, pp. 391-394.

Myxomatosis in Domestic Rabbits in Oregon

CV Löhr[1], **Rob J Bildfell**[1], BJ Cooper[1], JR Heidel[1], BA Valentine[1], M Kiupel[2] and JP Teifke[3]
[1]Oregon State University, Corvallis, OR; [2]Michigan State University, Lansing, MI; [3]Friedrich-Loeffler-Institute, Riems, Germany

Signalment: This 3 month old female Rex rabbit was noted to be anorexic and have facial edema 24 hrs before death. No treatment was attempted. This is one of 7 mortalities in the rabbitry (total population of 21 animals) over the previous 2 weeks. Affected rabbits ranged from 2 months to 5 years of age. Facial and anogenital edema were also noted in several of the other mortalities.

Gross findings: There was mild bilateral swelling of the eyelids. No internal abnormalities were noted.

Microscopic findings: Tissues on the slide include facial skin, anogenital skin, sacculus rotundus and appendix. Noteworthy features in skin include the edematous to mucinous character of the dermis and swollen, abnormal keratinocytes which often contain a single, eosinophilic cytoplasmic inclusion body. These inclusions can also be found in sebocytes and some follicular epithelial cells. Small numbers of large, polygonal to angular cells with an abundant cytoplasm may also be seen in the superficial dermis. In some sections, dermal capillaries are blocked by bacterial thrombi. There is marked lymphoid depletion and lymphocytolysis in the mucosal follicles of the gut sections. Close examination of dome epithelial cells may reveal cytoplasmic, eosinophilic inclusion bodies.

Comments: Periodic outbreaks of myxomatosis are seen in domestic rabbits housed outdoors in Oregon during the summer months. The occurrence of such outbreaks is likely influenced by the populations of the passive mosquito vectors for this leporipoxvirus and by the density and immune status of the reservoir species, the Californian brush rabbit, *Sylvilagus bachmani*. Rabbits are generally either found dead, or are culled/die after showing lethargy and anorexia for 24 -72 hrs. Skin nodules (myxomas) do not develop with the Californian strain, which is isolated from cases along the Pacific Coast of North America. Swelling of the eyelids and/or the genital region is an important necropsy finding, but is not consistently present. A hemorrhagic to necrotizing appendicitis and/or ileitis of the sacculus rotundus is due to necrosis of gut associated lymphoid tissue. Severe lymphoid depletion is also seen in the spleen and lymph nodes. This targeting of the immune system (MHC II-positive cells and T cells are infected) (1) is an important feature of the pathogenesis of myxomatosis. Secondary infections with Gram-negative bacteria are common and are likely the cause of death in most cases. Although not shown on this slide, an uncommon finding is bronchial epithelial dysplasia with cytoplasmic inclusion bodies.

Reference:

Coevolution of host and virus: cellular localization of virus in myxoma virus infection of resistant and susceptible European rabbits. S.M. Best, S.V. Collins and P.J. Kerr. *Virology* **277**;76-91 (2000)

Disseminated Protothecosis in a Dog

Lily I. Cheng, Marie E. Pinkerton
Veterinary Diagnostic Laboratory, University of Illinois, Urbana-Champaign

A 23.4 kg, 4 year old, spayed female, brown boxer dog was presented to the University of Illinois Veterinary Teaching Hospital in July 2005 for clinical signs of diarrhea, neurologic deficits and blindness. The dog had resided in Louisiana approximately two years prior to moving to Illinois. Due to a poor response to treatment, the dog was euthanized and submitted to the Veterinary Diagnostic Laboratory for necropsy.

Significant gross findings included miliary granulomas of the heart, kidney and spleen. Bilaterally, the kidneys had marked chronic infarcts with few medullary cysts.

Histologically, cardiac myocytes are multifocally effaced by discrete, circumscribed nodules composed of concentric layers of dense fibrous connective tissue surrounding a central area of pallor, with loss of differential staining and cellular detail (caseous necrosis). The center of the nodules contain myriad, pale amphophilic, round to oval, 8-20 µm in diameter organisms with a clear 2-4 µm thick wall. The organisms are often segmented by thin septa into wedge-shaped endospores (Apie slices@ or AMercedes Benz emblem-like@) that contain a 2-4 µm, brightly eosinophilic nucleus. Occasionally, organisms are degenerate and deeply basophilic (mineralization) or consist solely of an empty capsule wall following release of endospores. Similar organisms are in the kidney where the renal pelvis is largely effaced by sheets of organisms within the stroma admixed with degenerate and nondegenerate inflammatory cells.

The presenting clinical signs were well-explained by histologic evidence of organisms in the eye, brain, liver, spleen and the small intestine. *Prototheca zopfii* organisms were isolated from heart, kidney and spleen tissue on Columbia agar with 5% sheep blood agar after 48 hours incubation at 37_ C. The organism was identified using UNI-YEAST-TEK panel (Lenexa, KS).

Disseminated protothecosis is a rare infection caused by a microscopic colorless, achlorophyllic algae of the Family Chlorellaceae. *Prototheca spp*. are ubiquitous in nature and may be found in sewage, soil and water. The organism affects both humans and animals, including cats, dogs and cattle. Most cases of protothecosis in the United States are restricted to the Southeast and an increased incidence is cited in female dogs and Collies. Organisms may occasionally be recovered from animals with no clinical significance, however, disease is most often associated with host immunosuppression. Dogs with disseminated protothecosis normally present with a history of bloody diarrhea that is unresponsive to treatment. Disseminated and cutaneous protothecosis occurs in the dog (usually *P. zopfii*) while only the cutaneous form is seen in the cat (*P.wickerhammii*).

Morphologic diagnosis: Severe disseminated subacute to chronic pyogranulomatous and necrotizing myocarditis and pyelonephritis with intralesional algae organisms (consistent with *Prototheca zopfii*).