58th AAVLD Diagnostic Pathology Slide Seminar

American Association of Veterinary Laboratory Diagnosticians
Providence, Rhode Island
Saturday, October 24, 2015
3:30-6:00PM

2015 AAVLD Slide Seminar

Case #	Species	Organ	Diagnosis
1	Goat	Liver	Centrilobular hepatic necrosis
2	Bat	Skin	White Nose Syndrome
3	Dog	Heart	Hepatozoonosis
4	Dog	Brain	Pseudorabies
5	Horse	Liver	Tyzzer's/EHV-1
6	Bovine	Brain	Oxalate toxicosis
7	Opossum	Lung	Lungworm
8	Chicken	Multiple	Influenza H5N2
9	Pig	Bone	Vitamin A toxicosis
10	Bovine	Intestine	Enteric listeriosis
11	Goat	Spinal cord	Copper deficiency
12	Horse	Multiple	Streptococcus spp
13	Sheep	Skin	Parapox dermatitis
14	Goose	Bone	Osteopetrosis
15	Deer	Liver	Undetermined granulomatous disease
16	Dog	Multiple	Small cell carcinoma
17	Caique	Liver	Avian polyomavirus

AAVLD 2015 Slide Seminar, Case 1

Death and Severe centrilobular hepatocellular necrosis in Two Goats

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Signalment and History: This 3-year-old castrated male pygmy goat was the second of a pair of goats to die over two weeks. The first animal to die had a pale liver and plastic bags within the rumen. Rumenotomy was performed on the second animal to remove plastic bags, but the animal became ketotic, went into recumbency, and died within 10 days. On gross necropsy, the liver was pale yellow, rumen contents smelled ketotic, and the intestines contained variable yellow and purple to black digesta.

Microscopic Findings: Histologic lesions were similar in both animals. There was extensive centrilobular hepatocellular degeneration and necrosis with extensive loss of hepatocytes and marked centrilobular collapse, mild early fibrosis, and infiltrates of macrophages laden with abundant intracytoplasmic, blue-gray, pigment that was stained with rhodanine. Remaining centrilobular hepatocytes often contained discrete, open, microvacuoles or single macrovacuoles. Occasional individual rounded, hypereosinophilic, necrotic hepatocytes were scattered throughout the remaining parenchyma. In the kidneys, individual and clusters of proximal tubules were degenerate or necrotic, and remaining proximal tubular epithelial cells contained intracellular globules of dark brown hemosiderin pigment. Distal tubules and collecting ducts were often distended by accumulations of orange pigment suggestive of hemosiderin, amorphous eosinophilic, proteinaceous material, or infrequently by basophilic mineral. The cortical interstitium was minimally expanded by fibrosis and edema.

Diagnosis:
Liver: Severe centrilobular hepatocellular necrosis, degeneration, and loss
Kidneys: Marked proximal tubular degeneration and necrosis, intraepithelial hemosiderin accumulation, and intratubular hemoglobulin casts

Additional Findings: Hepatic mineral concentrations were evaluated in formalin fixed liver and kidney specimens using inductively-coupled plasma mass spectrometry. On a dry matter basis, liver and kidney copper concentrations were respectively 780ppm (reference range 20-650ppm) and 13ppm (reference range 9-18ppm). In addition, excessive lead levels in the liver of both animals and in samples of blood from the second animal to die were found.

Comment: Microscopic lesions of centrilobular hepatic necrosis and renal lesions are supportive of hemolytic anemia leading to centrilobular ischemia and hemoglobinuric nephrosis. Such findings in small ruminants are most classically associated with copper toxicosis. Hepatic copper levels were mildly to moderately elevated in this animal, and there was histologically obvious accumulation of copper in centrilobular areas of both goats suggesting a potential role of excessive copper in disease development. The additional finding of elevated lead is unusual as these animals lacked obvious neurologic and gastrointestinal signs typically associated with lead poisoning. Some degree of hemolytic anemia has been reported with lead poisoning in humans; however, the anemia due to lead poisoning is not primarily due to hemolysis. Renal failure has also been reported with lead poisoning in humans, but no reports of renal failure due to hemoglobinuric nephrosis were found in reviewing veterinary and human medical literature. Testing of feed, rumen contents including removed plastic bags, and food buckets failed to identify a source of excessive lead or copper.

References:

1. Nolan CV, Shaikh ZA. Lead nephrotoxicity and associated disorders: biochemical mechanisms. Toxicology. 1992;73(2):127-46.

AAVLD 2015 Slide Seminar, Case 2

Histologic Features of White Nose Syndrome in Hibernating North American Bat Species

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History: White Nose Syndrome (WNS) is a fatal rapidly emerging fungal disease of hibernating bat species which was first recognized in 2006-07 in New York State. The disease is caused by the fungus *Pseudogymnoascus destructans*, a psychrophilic, previously non-pathogenic, environmental species introduced from Europe to North America. In less than 10 years this fungus has swept across Eastern United States and Canada, causing widespread collapse in hibernacula and colonies of hibernating temperate North American bat species. The first outbreaks were recognized in Michigan in early 2014.

Pathologic Findings: WNS is named for large white plaques of fungus growing on the nose, muzzle and face of affected bats. Bats become weak, dehydrated, and unable to fly prior to death. Recently a non-lethal ultraviolet fluorescence screening method has been reported to detect infections in their early stages. While named for the facial lesions, I prefer the submission of wing membranes for histologic examination, as they provide large surface areas to examine for fungal infestation. Periodic acid-Schiff staining is critical in evaluating the fungal hyphal and conidial morphology. Hibernating bats generally exhibit no to minimal inflammatory reaction, while active bats will display varying degrees of neutrophilic and lymphocytic infiltrates. The fungi form thick mats of non-pigmented, septate, branching, and parallel-walled, approximately 2-3 um in diameter hyphae. These hyphae invaded the epidermis and underlying connective tissue, forming small cup-shaped erosions. Superficially, variable numbers of conidia are present above the epidermal surface. These conidia are thicker than the hyphae, 2.5-3.5 um in diameter and up to 7.5 um long, curved, and rounded at both ends.

Comments: In addition to the fungal presence and morphologic characteristics, a positive PCR for *P. destructans*, and epidermal erosions or ulceration are required to positively confirm infection for the first time in a specific bat colony location.

References:
Courtin F, Stone WB, Risatti G, Gilbert K, Van Kruiningen HJ: Pathologic findings and liver elements in hibernating bats with white-nose syndrome. Vet. Pathol. 47:214-219, 2010.
Meteyer CU, Buckles EL, Blehart DS, et al.: Histopathologic criteria to confirm white-nose syndrome in bats. J. Vet. Diagn. Invest. 21:411-414, 2009.

AAVLD 2015 Slide Seminar, Case 3

Pathology and associated lesions of disseminated canine hepatozoonosis in a dog

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Signalment, history and gross findings: A five-year-old, female, Beagle mix dog, had a clinical history of anorexia, lethargy, increased WBC count, fever, and azotemia. Laboratory tests for Lyme disease, and Ehrlichia were negative. The patient died despite treatment with antibiotics and fluid therapy. Fixed and fresh tissues were submitted to Tifton Veterinary Diagnostic and Investigational Laboratory for diagnosis. Grossly, multifocal areas of hemorrhage and degeneration were observed on the myocardium. Liver, kidneys, heart, and lungs were congested. Liver was attached to the diaphragm. Tissues were negative for canine hepatitis and canine parvovirus by fluorescent antibody (FA) test.

Histopathology: Disseminated "onion skin" cysts were present in various tissues and organs including the heart, diaphragm, skeletal muscle, pancreas, liver, small intestines and abdominal fibroadipose tissue. In the small intestines, the cysts were present in the mucosa, tunica muscularis, and the serosa. There are multifocal areas of chronic portal hepatitis, suppurative interstitial pancreatitis, granulomatous to pyogranulomatous myocarditis, and acute hepatic, splenic, gastric and intestinal necrosis with vascular thrombi in various organs. Mild to severe mineralization was present in the lungs, adrenal cortex, kidneys, intestines and the stomach. Additionally, there was diffuse, severe, intestinal, pancreatic, and renal glomerular amyloidosis.

Morphologic diagnosis: Disseminated canine hepatozoonosis, with secondary mineralization, amyloidosis, thrombi, necrosis and suppurative to pyogranulomatous inflammation in multiple organs.

Comments: American canine hepatozoonosis (ACH) is a highly debilitating, emerging tick-borne disease of dogs mainly in the south-central and south-eastern USA, although it is also documented in other regions of the USA. ACH is caused by *Hepatozoon americanum*, a protozoan parasite, the definitive host of which is the tick *Amblyomma maculatum* (1, 2, 3). Infected dogs are often febrile, stiff, lethargic, and depressed. Grossly, wasting of body mass (marked in temporal muscles), and hypertrophic osteopathy are recorded in chronic disease. Microscopically, "onion skin" cysts will be observed in various organs and tissues of the body. Mature meronts of a well-developed cyst release merozoites, which incite local inflammation and associated systemic reaction and overt illness (1). Dogs may die due to secondary amyloidosis, and glomerulonephritis (4). This is an interesting case in that diagnosis of the disease was challenging clinically; and due to chronic disease process, the pathology and pathogenesis of this emerging disease and associated lesions encompassing several tissues and organs that led to the death of the patient were worth noting.

References

Ewing SAE, and Panciera RJ (2003). American Canine Hepatozoonosis. Clin Microbiol Rev, 16 (4): 688–697.
Allen, KE, Johnson EM, and Little SE (2011). Hepatozoon spp Infections in the United States. Vet Clin Small Anim, 41: 1221–1238.
Cummings CA, Panciera RJ, Kocan KM, Mathew JS, and Ewing, SA (2005). Characterization of Stages of Hepatozoon americanum and of Parasitized Canine Host Cells. Vet Pathol, 42:788-796.
Van Vleet JF, and Valentine BA (2007). Muscle and tendons. Hepatozoonosis. In Jubb, Kennedy and Palmer's pathology of domestic animals, ed. Maxie MG, 5th ed., Vol 1. Pp 270-271.

AAVLD 2015 Slide Seminar, Case 4

Pseudorabies in a Plott hound

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Clinical History: A 6-year-old intact male Plott hound from Pine Hill, Alabama, presented for acute unilateral periocular swelling and intense pruritus. Physical examination revealed marked periocular swelling, facial alopecia, erythema, and excoriations. The patient progressed to a comatose state and passed away within 48 hours of initial presentation. The dog was necropsied and sections of brain, liver, spleen and tonsil were submitted for further evaluation.

Microscopic Description: Cerebrum: Neuropil architecture is disrupted by small, multifocal areas of gliosis. Neurons are occasionally shrunken with hypereosinophilic cytoplasm and hyperchromatic nuclei; affected neurons are surrounded by glial cells (satelliosis). Neurons occasionally contain round to oval, 3-5 um diameter, eosinophilic, homogenous, intranuclear inclusion bodies that marginate the chromatin (Cowdry type A). Virchow-Robin spaces in affected areas contain low numbers of lymphocytes, plasma cells and rare macrophages and endothelial cells lining vessels in these areas are often hypertrophied.

Virology:

- Pseudorabies PCR tonsil - Negative
- Pseudorabies PCR cerebrum – Positive (Ct 30)

Virus isolation:

- Pseudorabies virus was isolated from CNS tissue in porcine kidney-15 cells and confirmed by immunofluorescence staining

Diagnosis: Pseudorabies encephalitis

Comment: Pseudorabies virus (PRV) belongs to the subfamily alphaherspesvirinae of the family herpesviridae. Swine are the only natural host of PRV, although the virus may affect other wild and domestic animals including dogs, cattle, sheep, goats, foxes, rats and wild mice. In non-porcine species, mortality may approach 100% and recovery is unlikely.[1] PRV was eradicated from U.S. commercial swine operations in 2004 but remains in some localized feral swine populations. [2,3] Serologic investigation of feral populations revealed that roughly 20-60% of pigs surveyed from 25 different states were seropositive.[2] The Iowa State University Veterinary Diagnostic Laboratory has noted a recent increase in canine submissions with similar histories involving severe pruritus and feral hog hunting originating from southern states.

References:

1. Straw B, Zimmerman J, Allaire S, Taylor D. Disease of Swine. 9th edition. 2006: 419-433.
2. Wyckoff AC, et al. Feral swine contact with domestic swine: a serologic survey and assessment of potential for disease transmission. J Wildl Dis 2009; 45: 422-429.
3. Pedersen K, et al. Pseudorabies I Feral Swine in the United State, 2009-2012. J Wildl Dis 2013; 49: 709-713.

AAVLD 2015 Slide Seminar, Case 5

Concurrent Equine herpesvirus 1 and *Clostridium piliforme* hepatitis in a foal

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Signalment: 13-day-old Thoroughbred filly

History: The foal presented recumbent and comatose. A clinical examination indicated leukopenia, hypoglycemia, and sepsis. The foal was hospitalized and exhibited intermittent seizure activity. A day following presentation, the foal went into hypoglycemic shock, cardiac arrest, and subsequently died. The carcass was submitted to the UKVDL for necropsy.

Gross Necropsy Findings: Relevant findings included numerous, faint, tan, miliary foci throughout the hepatic parenchyma and serous fat atrophy of the epicardial adipose tissue.

Histopathology Findings: There were frequent, randomly distributed, multifocal to coalescing, variably sized foci of hepatocellular necrosis. Minimal to low numbers of neutrophils were intermixed with the necrotic debris. Adjacent hepatocytes multifocally contained acidophilic, intranuclear inclusion bodies. Other hepatocytes, adjacent to the regions of necrosis, contained rare stacks and piles of bacterial bacilli. Minimal to low numbers of lymphocytes, plasma cells, and macrophages expanded multiple portal regions. Additionally, there was moderate lymphocytolysis of splenic lymphoid nodules.

Laboratory findings: Pooled tissues were FAT positive for equine herpesvirus 1(EHV-1). A Warthin-Starry silver stain accentuated intracytoplasmic hepatocellular bacilli.

Diagnosis: Severe subacute multifocal to coalescing necrotizing hepatitis with intralesional acidophilic intranuclear inclusion bodies and cytoplasmic bacilli.

Comments: Lesions were consistent with concurrent EHV-1 and *C. piliforme* hepatitis. Both Tyzzer's disease and congenital EHV infections are common in young foals. However, concurrent infections are quite unusual. EHV-1 can result in abortion, stillbirth, or birth of weak foals. Foals may appear normal at birth, but they typically die as neonates. Neonates commonly present with respiratory signs, icterus, fever, and depression due to bronchiolitis, pneumonia, necrotizing hepatitis, and lymphoid necrosis. Immunodeficiency due to lymphopenia and lymphoid necrosis has been documented, and neonates can develop secondary septicemia. Tyzzer's disease is caused by *C. piliforme*, which commonly causes disease in foals 4 to 40 days of age. The incubation period is short, and clinical signs range from acute death to depression, fever, and icterus. Necropsy findings can consist of myocarditis, enterocolitis, and/or necrotizing hepatitis.

The interrelationship between the concurrent infections is not definitively known. However, a congenital EHV-1 infection may have contributed to immunodeficiency and predisposed the foal to Tyzzer's disease.

References:

1. Reed SM, Bayly WM, Sellon DC: 2004, Obstructive Disease of the Urinary Tract. *In*: Equine Internal Medicine, 2nd ed. Saunders, St. Louis.
2. Patel JR and J Heldens. Equine herpesviruses 1 (EHV-1) and 4 (EHV-4)--epidemiology, disease and immunoprophylaxis: a brief review. *Vet J* 2005. 170(1): 14-23.

AAVLD 2015 Slide Seminar, Case 6

Oxalate toxicosis in a 7-month-old Charolais heifer

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Clinical History: The case originated in a beef cow/calf herd from south central Missouri. Staggering and lethargy were reported in multiple calves (2 affected, 3 dead at the time of submission), with adult cows unaffected. The farm had been feeding moldy round hay bales to the affected group for 3-4 weeks prior to the development of clinical signs; however, no feed sample was submitted for analysis. Various tissues were received from a 7-month-old Charolais heifer. A 6 um, H&E stained section of cerebrum is included in the slide set.

Gross Pathology: Per submitting veterinarian, epicardial petechial hemorrhage was the only gross lesion observed at necropsy. Upon further examination of the submitted formalin-fixed brain tissue, slight discoloration of cortical grey matter was observed. Affected areas did not fluoresce when exposed to a Wood's lamp. Mild congestion of the meninges and increased opacity over the sulci were also noted.

Microscopic Description:
Cerebrum: Multifocal cortical blood vessels are surrounded by clear space and occasionally eosinophilic fluid with rare perivascular hemorrhage. The walls of affected vessels are often segmentally expanded by pale yellow, translucent, variably sized, anisotropic crystals that are birefringent under polarized light.

Diagnosis: Cerebrum: Oxalate toxicosis

Comments: Oxalate toxicosis in ruminants most commonly occurs due to the ingestion of plants that contain large amounts of soluble oxalate compounds.[1] Such plants include halogeton (*Halogeton glomeratus*), greasewood (*Sarcobatus vermiculatus*), rhubarb (*Rheum rhaponticum*), sorrels and docks (*Rumex* sp.) and pigweed (*Amaranthus* sp.).[2] Plants with lower amounts of oxalates include,beets, kale, broccoli, cauliflower, lamb's quarters, and Russian thistle. Several relatively common fungi, such as *Aspergillus* and *Penicillium* spp., have also been reported to produce oxalates,[3] and if consumed in large enough quantities, could theoretically result in a toxicity. Oxalic acid-producing *Aspergillus niger* has been previously isolated from a feed source in a case of oxalate nephrosis in sheep.[4] After ingestion, soluble oxalates are metabolized in the rumen or excreted; alternatively they may enter the bloodstream, where they bind to serum calcium. The complexes crystalize in the kidneys, causing blockage of renal tubules, tubular necrosis, and acute renal failure.[5] Oxalate crystals can occasionally be found within the walls of and surrounding blood vessels in the lung, heart, and cerebrum.[6] In this case, oxalate crystals were also present in the kidney, resulting in ectatic cortical tubules lined by attenuated epithelium.

References:

1. Maxie, M. Grant., K. V. F. Jubb, P. C. Kennedy, and Nigel Palmer. Jubb, Kennedy, and Palmer's Pathology of Domestic Animals. Edinburgh: Elsevier Saunders, 2007. Volume 2:471-472.
2. Cheeke, Peter R., and Peter R. Cheeke. Natural Toxicants in Feeds, Forages, and Poisonous Plants. Danville, IL: Interstate, 1998.
3. Dutton, Martin V., and Christine S. Evans. "Oxalate Production by Fungi: Its Role in Pathogenicity and Ecology in the Soil Environment." Canadian Journal of Microbiology 42.9 (1996): 881-95.
4. Botha, C.j., M. Truter, T. Bredell, L. Lange, and M.s.g. Mulders. "Putative Aspergillus Niger-induced Oxalate Nephrosis in Sheep : Clinical Communication." Journal of the South African Veterinary Association. 2009. 80(1): 50-53.
5. Plumlee, Konnie H. Clinical Veterinary Toxicology. St. Louis, MO: Mosby, 2004.
6. Joint Pathology Center: Case U-T06. http://www.askjpc.org/vspo/show_page.php?id=707

AAVLD 2015 Slide Seminar, Case 7

Severe Lungworm Infection in an Opossum

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Signalment: Adult, female intact opossum, (*Didelphis virginiana*).

History: The opossum was found deceased on the porch of a homeowner.

Gross Necropsy Findings: The opossum was in poor body condition weighing 2.1 kg with scant fat stores and was mildly dehydrated. The carcass was moderately autolysed. The most significant findings were multiple renal infarctions, a firm liver with numerous 1 mm white areas throughout the parenchyma, and diffusely pale, firm to meaty, slightly nodular, non-collapsing lungs. Thick brown to yellow material occupied the mainstem bronchi.

Histopathology Findings: Within the bronchiolar and alveolar lumina are numerous cross and tangential sections of nematodes. They measure up to 350 um in width and have a pseudocoelom with gravid reproductive tracts containing embryonated eggs as well as larger numbers of free larval forms. Gonadal and digestive tissues are seen in sections. Adult parasites are surrounded by scant macrophages while juvenile and larval forms incite a moderate to abundant granulomatous and lymphocytic infiltrate which occludes the alveoli and airways. The interstitium is markedly expanded due to alveolar wall thickening by type II pneumocyte hyperplasia, collagen, and lymphohistiocytic infiltration. There is also smooth muscle hyperplasia in the terminal bronchioles and alveolar ducts, and in the medial tunic of some small and medium sized arteries.

Laboratory findings: *Salmonella typhimurium* was isolated from the lungs. A Baermann fecal test recovered numerous larvae consistent with *Didelphostrongylus hayesi*.

Final diagnosis: Severe chronic multifocal to coalescing granulomatous pneumonia with intralesional nematodes (*Didelphostrongylus hayesi*)
Salmonella typhimurium infection
Pulmonary hypertrophic arteriopathy

Comments: *Didelphostrongylus hayesi* is a metastrongyloid nematode that affects only the opossum. It is reported to cause moderate to severe granulomatous bronchopneumonia with significant pulmonary smooth muscle hyperplasia and hypertrophy. These nematodes may cause chronic hypoxia by obstructing airways and inducing architectural changes not conducive with oxygen exchange. The worms are recognized as a cause of mortality and may induce pulmonary hypertension and right-sided heart failure. In this animal, histologic changes in the liver (chronic passive congestion with centrilobular hepatocellular loss) support a diagnosis of right-sided heart failure although thickening of the right ventricular wall was not noted grossly. Opossums are known carriers of *Salmonella spp*. and *Salmonella typhimurium* is commonly cultured from animals with evidence of bacterial septicemia.

References:

1. Duncan RB, et. al. Fatal Lungworm Infection in an Opossum. Journal of Wildlife Diseases. 1989; 25:266-269.
2. Lamberski N1, et. al. A retrospective study of 11 cases of lungworm (*Didelphostrongylus hayesi*) infection in opossums (*Didelphis virginiana*). J Zoo Wildl Med. 2002 Jun;33(2):151-6.

AAVLD 2015 Slide Seminar, Case 8

Histological findings in a natural outbreak of highly pathogenic avian influenza virus H5N2

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Clinical History: Two unrelated commercial layer poultry farms, located in north and central Iowa within a 150 mile radius, described a sudden mortality increase with nonspecific clinical signs including anorexia, reduced water consumption and central nervous signs. A total of 15 birds were submitted to the Iowa State University Veterinary Diagnostic Laboratory for diagnostic investigation

Gross Pathology: External examination revealed marked, diffuse edema of the comb and multifocal to locally extensive areas of hemorrhage. Scant to moderate amounts of mucus partially occluded the proximal portion of the trachea. Other macroscopic lesions included epicardial petechiae and ecchymosis, diffuse necrotizing pancreatitis, and multifocal to locally extensive areas of splenic hemorrhage and necrosis.

Histopathology: There is marked necrotizing bronchiolitis and diffuse lymphohistiocytic interstitial pneumonia. Areas of myocardial degeneration with multifocal lymphohistiocytic infiltration are observed. Occasional lymphohistiocytic perivascular cuffs are apparent in the neuropil associated with scattered neuronal degeneration and gliosis. There are multifocal to coalescing areas of necrotizing pancreatitis and splenitis with variable hemorrhage. The dermal collagen of the comb is diffusely expanded by edema with minimal lymphoplasmacytic perivasculitis.

Laboratory findings:

- Immunohistochemistry (viral nucleoprotein): heart, spleen, liver, and lung (Positive)
- PCR - Avian influenza

AIV H5; H5 icA: Cloacal swab 10/9; Oropharyngeal swabs; individuals 15/15 (Positive)
Avian influenza matrix: Cloacal swab 10/9, Oropharyngeal pools 2/2 (Positive)
AIV H7: Cloacal swab 10/9; Oropharyngeal swabs; individuals 5/5 (Negative)

- PCR Avian paramyxovirus-1: Matrix gene: Cloacal swab 10/9 (Negative)

Diagnosis: Highly pathogenic avian influenza.

Comment: Highly pathogenic avian influenza virus (HPAIV) causes a multisystemic disease associated with high mortality in poultry. Histological lesions observed in this case varied in severity and distribution amongst birds evaluated. Although histologic lesions observed in this case are similar to those described in experimentally inoculated chickens, infection in field conditions might produce variation in severity, distribution and chronicity due to viral dose and immune status of the flock. Histological lesions caused by H5N2infection are similar to those previously described in chickens naturally infected and experimentally inoculated with an H5N1 AIV isolated from commercial poultry. In this case viral sequencing confirmed H5N2 as causative agent.

References:

Kalthoff D, Breithaupt A, Helm B et al.:2009, Migratory Status Is Not Related to the Susceptibility to HPAIV H5N1 in an Insectivorous Passerine Species. Plos one 4:e6170

Swayne DE:1997, Pathobiology of H5N2 Mexican avian influenza virus infections of chickens. Vet Pathol 34:557-567

D.E. Swayne & D.L. Suarez. Highly pathogenic avian influenza. Rev. sci. tech. Off. int. Epiz., 2000,19 (2), 463-482

AAVLD 2015 Slide Seminar, Case 9

An Unusual Clinical Presentation of Hypervitaminosis A in Swine

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Signalment: 30-40% of Seven week old piglets, two presented.

History and Gross: A few sudden deaths, paddling, dog sitting, goose stepping and reluctance to move. Subsequent examination noted many piglets walking down on pasterns. Pigs were bright and alert. No clinical neurologic abnormalities, pigs were reluctant to move. Moderate consolidation of the lungs was noted. Bone strength and rib crispness appeared normal.

Histopathology: Mild lymphocytic perivascular inflammation was present in the choroid plexus and meninges associated with pineal gland. Lung had regions of suppurative inflammation. Most of lung had lymphohistocytic thickening of alveolar septae. Heart muscle from one piglet had a focus of lymphocytic myocarditis. There were small aggregates of lymphocytes and macrophages seen in periportal. Necrotic debris was seen within tonsillar crypts. The growth plates from the distal femur were discontinuous. The physis consisted of islands of cartilage with endochondral ossification. Large gaps were present in the growth plate. The resting cartilage was minimal and nests of cartilaginous matrix surrounding hypertrophied and proliferation chondrocytes predominated. Cartilage and maturation processes were disorganized.

Diagnosis: Pneumonia, interstitial, lymphohistiocytic, focally extensive, moderate; Bronchopneumonia, focal, lobular, suppurative; Tonsillitis, subacute, moderate: Myocarditis, lymphocytic, multifocal, mild; Meningitis, choroid plexus, focal, mild.
Chondrodysplasia, with segmental growth plate loss (necrosis).

Comment: Vitamin A toxicosis is associated with growth plate necrosis in growing piglets. Necrosis left behind a few islands of cartilage and clinical pain resulted. The death losses are presumed due to concurrent bacterial disease. *Strep suis* and *H. parasuis* were isolated. Vitamin A levels in liver were 1136 and 1242 ppm in livers with normal 36-57 expected. Feed was nearly 100 fold increased at 103,000 IU/gram. Normal. 1-000-5500 IU is normal source: NRC pork central. A later batch of feed was tested at 1570 IU/ gram.

References:

Hypervitaminosis A in the young pig, Anderson MD, Speer VC, McCall JT, Hays VW J Anim Sci. 1966 Nov;25(4):1123-7.
Bone lesions of hypervitaminosis A in the pig. Wolke RE, Nielsen SW, Rousseau JE Jr. Am J Vet Res. 1968 May;29(5):1009-24.
Aust Vet J. 1969 Dec;45(12):570-3.
Local Disappearance of Epiphyseal Growth Plates in Rats with Hypervitaminosis A. Tetsuo Kodaka, Hisashi Takaki, Satoshi Soeta, Ryoichi Mor, Yoshihisa Naito, Journal of Veterinary Medical Science. Vol. 60 (1998) No. 7 P 815-821

AAVLD 2015 Slide Seminar, Case 10

Enteric listeriosis in steers

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Clinical history: One of several 1year old Hereford steers, with a 10 day history of depression, diarrhea, recumbence, and death. The problem started 2 days after the animals were fed spoiled silage.

Gross findings: Moderate amount of translucent yellowish fluid in abdominal cavity; severe congestion of the entire digestive tract; thickening of small and large intestine wall; liquid content in small and large intestine, with multiple fibrin strands loose or adhered to the mucosa.

Histopathology: Significant microscopic lesions were present in the small and large intestine, mesenteric lymph nodes and liver. The small intestinal lesions consisted of moderate diffuse mucosal congestion, multifocal mucosal necrosis, villus blunting and multifocal to diffuse infiltration of viable and degenerate neutrophils and fewer macrophages, lymphocytes and plasma cells in the lamina propria, muscularis mucosa and superficial submucosa. The neutrophilic infiltration was more severe in the muscularis mucosa tan in the other layers of the intestine. Multifocally small and large intestinal crypts dilated and lined by attenuated epithelium that was frequently necrotic; neutrophils and sloughed necrotic epithelial cells were in the lumen of the crypts. Clusters of intralesional Gram positive small rods were seen within the mucosa, muscularis mucosae and submucosa. Mesenteric lymph nodes had extensive multifocal to coalescing areas of neutrophilic infiltration, fibrin exudation and necrosis, that affected large areas of the cortex, subcapsular sinuses and capsule. Intralesional clusters of Gram positive small rods similar to those seen in the intestine were also present in the affected lymph nodes.

Comments: Several strains of *Listeria monocytogenes* were isolated from the gallbladder and from the silage fed to the animals. *L. monocytogenes* was detected in intestinal wall and mesenteric lymph nodes by immunohistochemistry (IHC). Clinical history and signs, gross and microscopic findings, bacterial isolation, and IHC results confirmed a diagnosis of enteric listeriosis. The source of infection was likely the spoiled silage. The inflammation of the lamina propria with intralesional bacteria is a hallmark finding in this disease.[1,2]

References:

1-Fairley RA, Pesavento PA, Clark RG. 2012. Listeria monocytogenes infection of the alimentary tract (enteric listeriosis) of sheep in New Zealand. J Comp Pathol 146:308-313.
2-Gracia JA, Micheloud JF, Campero CM, Morrell EL, Odriozola ER, Moreira AR. 2015. Enteric listeriosis in grazing steers supplemented with spoiled foodstuff. J Vet Diag Invest. Forthcoming.

AAVLD 2015 Slide Seminar, Case 11

Spinal Cord Degeneration in a Young Goat Associated with Cerebellar Abiotrophy

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Signalment: A goat kid 3.5 months of age, unknown breed.

History: The goat was examined by the submitting veterinarian with an initial complaint of ataxia in its rear limbs. By the time of clinical evaluation, the kid was recumbent, blind and grinding its teeth. The owner of the flock reported that 4 additional young kids had a clinically similar disease during the spring.

Gross Necropsy Findings: Grossly, no lesions were noted.

Histopathology Findings: Sections of spinal cord contain swollen, chromatolytic neurons with reduced or absent Nissl substance, affecting neurons at random in Clarke's column and the ventral horns throughout the length of the cord. Rare shrunken red neurons have pyknotic nuclei at these sites. Chromatolytic neurons retain cell-body neurofilaments compared to non-degenerate cell bodies and dilated axons are also intensely stained. Additional affected neurons occurred in the red and vestibular nuclei of the brain. This change is accompanied by spinal cord white matter degeneration that is most severe in the lateral columns (spinocerebellar and spinomedullary tracts) and along the ventral median fissure. Affected white matter is characterized by distended axons and axon sheaths, mixed with normal-appearing fibers. The areas of white matter degeneration are best demonstrated with stains that detect perivascular microglia.

Also present is segmentally severe loss of cerebellar Purkinje cells that involves some tissue in all longitudinally and cross-sectioned lobes (not present on submitted slides). Degenerating Purkinje neurons are bright pink in color, and have dilated axons near the cell body; nearby internal granular neurons are pyknotic.

Laboratory Findings: The copper concentration of the liver sampled from this kid was 2.6 ppm (wet-weight basis, Reference range 25-150 ppm).

Final Diagnosis: Myelopathy characterized by long tract axonal degeneration and neuronal degeneration and necrosis (enzootic ataxia)

Comments: Copper deficiency, or more properly copper unavailability, has a spectrum of microscopic lesions depending on timing and severity of disease. Ruminants are at particular risk, due to potential binding of dietary copper to thiomolybdate complexes in the rumen. Cerebellar lesions have been reported in about one third of affected goats and were extensive in this kid. It is interesting that a lamb with histologically similar spinal cord lesions, low liver copper and high liver molybdate levels was examined at the VMDL within 10 days of the diagnosis of this case. Copper deficiency is not historically an important disease in Missouri, but perhaps is under-reported.

References

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AAVLD 2015 Slide Seminar, Case 12

Vegetative valvular endocarditis with bacterial emboli in a horse due to non-hemolytic streptococcus infection

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Signalment and History: A 7-year-old, intact female, Appaloosa horse had a history of recurrent high fever, anorexia, and weight loss despite antibiotic treatment. Examination and echocardiography were suggestive of aortic valve endocarditis. Subsequently, the animal started coughing and leaked foam from the nostrils. The horse was euthanized due to lack of improvement.

Gross findings: The primary lesions were found in the heart. All leaflets of the aortic valve were severely thickened, mottled pink to dark red, firm, and had deep purple, friable, irregular, cauliflower-like rims projected into the lumen. Both kidneys had multiple infarcts characterized by slightly indented, mottled pink to purple, sharply demarcated areas, ranging from 2-8-cm in maximum dimension, on the capsular surface. On cross sections, these infarcts were sharply demarcated, dark red to deep purple, wedge-shaped areas extending from medulla to cortex associated with infarct areas on the cortical surface. The lung was diffusely dark red, soft, wet, non-collapsed and oozed pink foam on cut surface.

Histopathologic Findings: The aortic and mitral valves were severely thickened by diffuse myxomatous degeneration and mostly effaced by severe necrotizing to fibrinous inflammation with numerous intralesional bacteria. Necrotizing endocarditis comprised of a thick mat of bacterial cocci on a thick layer of eosinophilic necrotic debris admixed with severe infiltrates of viable and degenerate neutrophils. The lesion was sharply demarcated and bordered by a thick layer of granulating fibrosis segregating from the underlying myocardium, which exhibited locally extensive coagulative necrosis. In both kidneys, there was severe multifocal embolic necrotizing vasculitis with perivascular and intramural neutrophilic infiltrates and numerous intralesional bacteria. Multiple blood vessels in the kidney were occluded by fibrin thrombi, resulting in severe multifocal renal infarction. There also was severe locally-extensive necrotizing vasculitis with vascular thrombosis and numerous intralesional bacteria within the meninges. Affected blood vessels had fibrinoid necrosis and were often occluded by fibrin thrombi admixed with large aggregates of viable and degenerate neutrophils. There was locally extensive rarefaction of the granular and molecular layers of the subjacent cerebellum with marked infiltrates of foamy macrophages within the neuropil and white matter.

Comments: Bacterial isolation yielded non-hemolytic streptococcus from both the aortic valve and the kidney. Based on the gross findings, histopathology, and microbiology a diagnosis of severe chronic vegetative valvular endocarditis with septic emboli due to non-hemolytic Streptococcus infection was made. Dislodging of septic emboli from the valves also caused severe multifocal embolic vasculitis in the kidney and meninges, resulting in bilateral renal infarction and encephalomalacia. In general, bacterial endocarditis in horses is rare. The mitral valve, aortic valve, or both are most commonly involved. Echocardiography is the most specific and sensitive imaging system for the diagnosis of bacterial endocarditis in horses. Antemortem bacterial culture of blood samples, when attempted, is not always successful in identifying an etiologic agent. No breed or sex predilection is recognized for bacterial endocarditis; however, young horses are at increased risk due to increased incidence of bacteremia, possibly from underlying viral infection and lack of vaccination. Bacteria most commonly associated with endocarditis include P*asteurella sp, Actinobacillus sp,* and *Streptococcus sp* and less commonly*Escherichia coli, Corynebacterium sp, Pseudomonas sp, Bacillus sp, Erysipelothrix rhusiopathiae,* and *Borrelia burgdorferi*.

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AAVLD 2015 Slide Seminar, Case 13

Proliferative dermatitis in a Suffolk ram

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Signalment and History: In February of 2015, a 1-year-old Suffolk ram presented with an acute history of swollen and bleeding hind limbs. At the time of examination, the ram had a rectal temperature of 104.5F and the skin over both fetlocks was friable and warm on palpation. There were large, sharply demarcated, vegetative, proliferative lesions over the dorsal and lateral surface of the left distal hind limb, the dorsal surface of the right distal hind limb, and the lateral surface of the left distal forelimb.

Histopathologic Findings: The submitted slide represents a biopsy of skin from the raised edge of the right hind limb lesion. Extending from the epidermis, there is a well delineated exophytic proliferation of well differentiated papillated squamous epithelium with marked overlying serocellular crusting admixed with surface bacteria and fibrin. The papillated epithelium is segmentally severely eroded and ulcerated. Intact regions of epidermis are irregularly thickened and lined by mild parakeratosis. There is elongation of the rete pegs, multifocal intracorneal pustules, and ballooning degeneration of numerous keratinocytes. Occasional intracytoplasmic eosinophilic inclusion bodies are within cells undergoing ballooning degeneration in the stratum spinosum and granulosum. There is multifocal intracellular edema of basilar cells and occasional dyskeratotic cells are noted. The superficial connective tissue core within papillary projections is loosely expanded by edema and focal areas of hemorrhage, and contains numerous small capillaries and reactive fibroblasts as well as marked infiltrates of neutrophils and lesser numbers of lymphocytes and plasma cells. The subepithelial stroma is multifocally expanded by a marked proliferation of polygonal to spindloid cells arranged in cohesive bundles that extend from the epidermal-dermal junction to the superficial dermis. These proliferative cells have a small to moderate amount of wispy pale basophilic cytoplasm and indistinct cell borders. Nuclei are round to ovoid and densely stippled. Anisokaryosis is moderate, and there are 0-2 mitoses per 10 high power fields (400x). Inclusion bodies are not within this cell population.

Diagnosis: Severe proliferative and ulcerative dermatitis with epidermal intracytoplasmic eosinophilic inclusion bodies and atypical superficial dermal dendritic cell proliferation

Additional Findings: A diagnosis of Parapoxvirus was confirmed by PCR.

Comment: Although variable numbers of dendritic cells within the dermis of ovine skin infected with Parapoxvirus has been well described, the plump atypical mesenchymal cells noted in this case caused some concern for the more virulent Sheeppox virus. Molecular testing, however, was negative for Sheeppox virus. Parapoxvirus infection was confirmed; thus, a diagnosis of contagious ecthyma, or Orf was made. These antigen presenting cells, albeit atypical in appearance, are responsible for the initiation and maintenance of the host adaptive immune response following viral infection. It is proposed that these cells accumulate beneath the affected epidermis due to this virus's ability to disrupt dendritic cell maturation. Thus, viral clearance and subsequent healing time is a reflection of the dynamic relationship between viral immune modulatory proteins and the host immune response.

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AAVLD 2015 Slide Seminar, Case 14

Osteopetrosis in a Domestic Goose

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Signalment: 2-year-old, female intact, African goose

History: The goose presented for weakness, inappetance and lethargy. The owner also reported ataxia following the production of a clutch of 2 eggs and had been hand-feeding the goose for the past 4 months. On presentation, the goose was unable to stand or walk unassisted, but was quiet, alert and mentally appropriate. On radiographs, there was bilateral severe bone lysis of the femoral heads and necks and there were fractures of the right ulna and radius with surrounding periosteal proliferation. Bloodwork was none remarkable.

Gross Necropsy Findings: The articular surfaces of the left femoral head were irregular with patchy, dark, subchondral discoloration. The joint capsule was moderately thickened. Within the right humerus, the endosteum was distorted by irregular, multinodular to coalescing, 1-3mm in diameter proliferations that extend into and span the marrow space. There was a smooth, hard periosteal callus surrounding the radiographically-identified fractured area of the proximal right ulna and radius.

Histopathology Findings: In both femurs and the right humerus, the marrow cavity is filled with trabecular bone extending from the endosteal surfaces of the diaphysis, metaphysis and epiphysis with numerous tidemarks. Trabeculae are lined by a 1-3 cell-thick layer of osteoblasts. Numerous lacunae are present, most containing viable osteocytes. There is moderate periosteal proliferation in the metaphysis. Remaining marrow spaces contain variable amounts of erythrocytic and myeloid cell precursors admixed with fat. In the left femoral head, the articular surface is irregular with fragmentation of the cartilaginous matrix, proliferation and cloning of chondrocytes and proliferation of chondroid matrix.

Final Diagnosis: Osteopetrosis with osteochondrosis of the left femoral head

Comments: Osteopetrosis in birds has been reportedly mainly in association with retroviral infection, most commonly avian leukosis virus in chickens, which causes a proliferation of osteoblasts. There are sporadic reports of geese being infected with avian leukosis virus, but none associated with osteopetrosis. There is a single report of a naturally occurring outbreak of reticuloendotheliosis virus in geese causing widespread lymphoproliferative disease that was experimentally transmissible. One bird in this outbreak was reported to have “osteopetrosis-like lesions”, but these were never characterized. The birds in this reported outbreak were also 16-22 weeks of age, whereas the bird in the current case was an adult with a more chronic course of disease. There has been no evidence of tumors in this flock or other birds with similar bone pathology.

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AAVLD 2015 Slide Seminar, Case 15

Disseminated granulomas in a white-tailed deer

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SIGNALMENT: 3.5-year old, female, white-tailed deer

History: This white-tailed deer was harvested in December of 2014 in Kansas. During field dressing the hunter noted numerous nodules throughout the skeletal muscle and organs of this deer, and submitted tissue samples to a wildlife biologist at the Kansas Department of Wildlife, Parks, and Tourism. Tissues were then submitted to the Southeastern Cooperative Wildlife Disease Study for evaluation.

Gross Necropsy Findings: Samples of lung, heart, liver, kidney, omentum, and skeletal muscle from an adult female white-tailed deer were received for evaluation. Hundreds of multifocal to coalescing, 0.2 – 1.0 cm, smooth, firm, raised, white nodules were present on the pleural surface of the lungs, over the epicardial and endocardial surfaces of the heart and extending into the myocardium, over the capsular surface of the liver, and throughout the omentum. Nodules are also present within the parenchyma of the liver and skeletal muscle. On cut surface, the nodules have tan to yellow firm cores. The kidneys were not affected. There were no other significant findings.

Histopathology Findings: Scattered throughout the omentum, liver, heart, lung, and skeletal muscle are multifocal to coalescing granulomas composed of central cores of eosinophilic debris surrounded by rings of epithelioid macrophages and hundreds of multinucleate giant cells with a peripheral layers of reactive fibroblasts, collagen bundles, and intermixed eosinophils, lymphocytes, and plasma cells. Granulomas predominate on the serosal surfaces or fascial connective tissues of all evaluated organs, but occasionally are present within the parenchyma. No bacterial or fungal organisms were identified with gram, Grocott's methenamine silver (GMS), or acid fast staining.

Final Diagnosis: Granulomatous and eosinophilic serositis, hepatitis, myocarditis, and myositis, multifocal to coalescing, moderate, chronic

Comments: The etiology of the widely disseminated granulomas in this deer was not determined. *Mycobacterium bovis*, the causative agent of bovine tuberculosis, was not detected by polymerase chain reaction, culture, or acid fast staining. No other bacterial, fungal, or parasitic agents were identified in the lesions microscopically. Similar lesions were recently reported in a llama in association with *Heterobilharzia americana* infection, and *H. americana* infection remains a differential in this case. Other potential differentials include an atypical immune response to parasite migration, potentially in response to the abdominal worm *Setaria yehi*, immune mediated disease, or hairy vetch (*Vicia villosa*) poisoning.

References:

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AAVLD 2015 Slide Seminar, Case 17

Avian Polyomavirus infection in two white bellied caiques (*Pionites leucogaster*).

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History: Two, 6-month-old male white bellied caiques with history of sudden death. The owner has a history of *Pseudomonas* septicemia/death in various species of parrots they had raised. Recently cultured *Pseudomonas spp*. in baby bird isolate.

Gross findings: Both birds were in good body condition. The liver was slightly swollen with a very fine reticular pattern. The heart was pale with focal paint-brush hemorrhages. Kidneys were diffusely pale and swollen.

Histologic findings: In the liver, there was multifocal to coalescing hepatic necrosis, characterized by the presence of random areas of parenchyma displaying marked cytoplasm eosinophilia and pyknotic or karyorhectic debris, together with mild infiltrates of heterophils and discrete to extensive hemorrhages. In viable hepatic areas, hepatocytes display an enlarged nucleus containing a one glassy amphophilic inclusion body. Periportal areas have small infiltrates of lymphocytes, plasma cells and few macrophages.

Ancillary testing: Avian Polyomavirus PCR: positive; Avian Influenza virus PCR: negative; no *Salmonella* spp detected from liver or small intestine; no bacterial growth from the liver after 48 hours of aerobic culture; Liver mineral screen (Pb, Mn, Fe, Hg, As, Mo, Zn, Cu, Cd) was within normal limit.

Comments: Avian Polyomavirus (APV) have been associated with acute and chronic diseases in several species of birds, predominantly psittacines birds. One characteristic inflammatory condition of young parrots is Polyomavirus disease. It was first described in 1981 as the etiologic agent of budgerigar fledgling disease, characterized by high mortality (close to 100%), hepatitis, ascites and hydropericardium. The disease affects predominantly to nestling parrots less than 16 weeks of age; macaws, coenures, caiques, eclectus and ring-necked parrots are the most susceptible. In adults, caiques are particular susceptible to the disease. Acute disease is characterized by ascites, hydropericardium, hepatomegaly with variable degree of mottling and serosal hemorrhages. Multifocal to coalescing hepatic necrosis to some degree is present in nearly all cases, and when severe, the periportal areas are spared. Nuclei of hepatocytes and Kupfer cells are markedly enlarged (karyomegaly) and contain characteristic intranuclear, basophilic to amphophilic inclusion bodies. Splenic and renal lesions are also common in this disease. Differential diagnoses for necrotizing hepatitis in parrots include Pacheco's diseases (Psittacid herpesvirus), Adenovirus, Reovirus, *Chlamydophila* infection and disseminated bacterial disease (*E.coli, Proteus spp, Proteus spp, Enterobacter spp, Salmonella spp, Pseudomonas spp* and *Yersinia pseudotuberculosis*).

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