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Diseases of the Alimentary Tract in Domestic Animals

- - Case Summaries and Abstracts - -

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Case #1 (BO-04815-19) - Brenda Bryan and Vasyl Shpyrka, Manitoba Agriculture, Veterinary Diagnostic Services Laboratory.

Four Red Angus cows were presented for slaughter at local abattoir. One of them had a firm immovable lump on its ventral mandible, its body condition were from thin to fair. At post mortem inspection multiple nodules were present in the lungs of affected animal. Mandible and lungs were submitted to the diagnostic lab, rest of the carcass was held.

GROSS EXAMINATION: Received fresh lower jaw, ramus removed, and tongue in situ; and lungs with portion of trachea and scant portion base of heart with great vessels.

Mandible: Mandible from mature cow measuring approximately 55cm long x 30cm wide x 20cm thick proximal 1/3. A single submandibular lymph node attached to the jaw was unremarkable. The haired skin of the ventral and proximal 1/3 of the jaw was thickened, multifocally alopecic, and firm with multifocal to coalescing thick folds and thick tan crusts and hairless tan to pink nodules up to 10cm diameter. The oral mucosa under the tongue, along the body of the mandible buccal gingiva and free gingival margin had multifocal round to elongated well demarcated pink to tan, firm ulcers that were slightly raised and measured up to 4cm diameter and up to 7cm long respectively. On the gingival surface between cheek teeth and cheek mucosa, there was a film of bright yellow mucoid sticky exudate. Embedded in the free gingiva between the tongue and mandible were multifocal sharp or spiny elongated pieces of plant material. The ventral aspect of the tongue had multifocal random well demarcated white firm nodules measuring up to 10mm in dimeter. Cut section was abscess/granuloma centered on scant brown material. The jaw was split longitudinally revealing destruction of the normal mandibular architecture. There were multifocal to coalescing red tan grey to black, soft, wide necrotic draining tracts surrounded by bone or thick fibrous tissue, that formed fistula, drained to the ulcers/nodules of the ventral jaw and oral cavity and that effaced and deformed the bony mandible.

Lungs: Multifocally and randomly there were small firm fibrotic abscesses in clusters of up to 5cm diameter. Cut section was well demarcated, fibrous capsule centered on tan to yellow exudate, occasionally with central pigment. Remaining lung was soft, pink and collapsed (normal) and trachea was unremarkable. Bronchial lymph nodes were unremarkable.

HISTOPATHOLOGY:
Gingiva, tongue: in the submucosa and subjacent skeletal muscle there were multifocal to coalescing well demarcated round inflammatory foci up to 5mm dimeter centered on irregularly round clubs of eosinophilic bodies (Splendore Hoeppli material) with variable amounts of irregular pale basophilic centers (tiny coccoid bacterial clusters) and occasionally mineralization, surrounded by degenerate and viable neutrophils and collared by a thick layer of lymphocytes, multinucleated giant cells, and macrophages all encased in fibrous connective tissue (pyogranuloma). Intervening submucosa and skeletal muscle was separated by dense collagenous fibrovascular tissue with scant random and perivascular lymphocytes and plasma cells.
Lung: lung parenchyma had similar multifocal pyogranulomatous inflammatory foci as gingiva/tongue.

Lymph node, bronchial slide A: mild lymphoid hyperplasia, lymphoid follicles indistinct (decomposition) capsule slightly thickened by fibrous tissue. Lymph node, submandibular slide B: lymph node cortex and medulla expanded by mild follicular hyperplasia and plasma cells aggregates.

Red/tan ulcerated button on gingiva slide C: ulcerated surface composed of compressed dark pink cells, nuclei, small vessels and collagen and subjacent multifocal pyogranulomatous inflammatory foci with Splendore Hoeppli material embedded in lymphocytes, plasma cells, macrophages, multinucleated giant cells and fibrovascular tissue.
Bone, mandible: Trabecular lamellar bone lined by thick fibrous tissues and separated by wide inflammatory tracts characterized by multifocal Splendore Hoeppli foci similar and previously described in gingiva embedded in sheets of mononuclear cells and multifocal small vessels, lymphoplasmacytic perivascular infiltrates, occasional hemorrhage and isolated bony spicules.

SPECIAL HISTOCHEMICAL STAINS:
Acid fast: tongue, lung, soft tissue oral pyogranulomas: no acid-fast microbes seen.
Gram stain: Gram-positive bacterial branching filaments prominent at the border of the Splendore Hoeppli reaction foci. Within the Splendore Hoeppli foci, there were gram-positive bacterial branching filaments, gram-positive thin rods and gram-positive cocci.
PAS: Splendore hoeppli granules had purple centers and a bright magenta granular irregular collar lined the inner surface of the outer club-shaped margins possibly reflecting the polysaccharide-protein complex that holds the granule together.

BACTERIOLOGY:
Lung Abscess: Actinomyces bovis 3+, Streptococcus uberis few, Staphylococcus aureus 1col
Mandible bone exudate abscess: Trueperella pyogenes 4+
Tongue abscess: Streptococcus uberis few
Lymph node submandibular: Streptococcus aureus few, Streptococcus dysgalactiae 3+

ANAEROBIC BACTERIOLOGY:
Lung: Fusobacterium 4+
Mandible bone exudate abscess: to follow
Tongue abscess: to date
Lymph node submandibular: to date

DIAGNOSIS: Mandibular Osteomyelitis (Lumpy Jaw), Pulmonary Pyogranulomas
1. Osteomyelitis, mandibular, pyogranulomatous, proliferative and resorptive, severe, with suppurative draining tracts, and mucosal and epidermal fistulation, chronic
2. Ventral glossal, gingival pyogranulomas, miliary, with intralesional bacteria and Splendore Hoeppli material
3. Gingival ulceration, multifocal lateral sublingual with foreign material (plant)
4. Pulmonary pyogranulomas, multifocal, mild, chronic with intralesional bacteria and Splendore Hoeppli material

References:

1. Pathologic basis of veterinary disease/ [edited by] James F. Zachary, sixth edition,2017; pp: 191,942, 926
2. Jubb, Kennedy, and Palmer's pathology of domestic animals / edited by M. Grant Maxie.—Sixth edition, 2017; Vol1; pp: 102-103
3. Large animal internal medicine / editor in chief, Bradford P. Smith.—Fifth edition, 2015; pp: 743-744
4. Veterinary Medicine: A Textbook of The Diseases of Cattle, Horses, Sheep, Pigs, and Goats, Eleventh Edition,2017;Vol1; pp: 531-532

Case #2 (17-1931) – Laura White, Washington Animal Disease Diagnostic lab, Washington State University.

Necrotizing sialometaplasia

A 10 year old FS Golden Retriever presented with a 3 cm in diameter non-painful, firm and freely moveable mass in the right submandibular region. The mass was initially evaluated via needle biopsy with a diagnosis of presumptive squamous cell carcinoma. The entire mass was then removed and submitted.

Histology: The submandibular lymph node is characterized by extensive lobular atrophy and loss with replacement by streams of dense fibrosis. Ducts are prominent and often torturous. Rarely, the ductular epithelium is hyperplastic with piling of layers, or exhibits squamous metaplasia. Occasional ducts contain small numbers of intraepithelial or intraluminal neutrophils. Scattered through the surrounding tissue are low numbers of lymphocytes, plasma cells, macrophages containing intracytoplasmic pigment and occasional neutrophils. Rare ductal epithelial cells are swollen with abundant granular eosinophilic cytoplasm (oncocytic metaplasia).

Morphologic diagnosis: Submandibular salivary gland: Marked extensive chronic lobular atrophy and loss with fibrosis, duct epithelial hyperplasia and squamous metaplasia

Condition: Necrotizing sialometaplasia

Comments:

Necrotizing sialometaplasia, also known as salivary gland infarction, is a condition reported in the dog, cat, and one rabbit. In dogs, clinical signs most often consistent of variably painful firm swelling of the submandibular salivary gland(s) and may include anorexia and fever. A subset of cases, typically young terrier breeds, present with anorexia, fever, severe nausea, vomiting and regurgitation. Lesions are typically unilateral but may be bilateral and rare reports involve other salivary glands. Early histologic lesions include acute coagulative necrosis, hemorrhage and edema with retention of lobular architecture, fibrinoid vascular necrosis, thrombi and variable mixed inflammation. Subacute to chronic histologic lesions include fibrosis, ductal hyperplasia and squamous metaplasia of glands and ducts. Due to the presence of hyperplasia and metaplasia, this lesion may be misdiagnosed as a malignancy, typically squamous cell carcinoma or mucoepidermoid carcinoma, especially in small incisional biopsies. The cause is unknown but speculated to involve vascular damage secondary to immune-mediated or traumatic etiologies.

References:

1. Brooks DG, Hottinger HA, Dunstan RW. Canine necrotizing sialometaplasia: a case report and review of the literature. *Journal of the American Animal Hospital Association* (1995) 31: 21-25.
2. Munday JS, Lohr CV, Kuipel M. Tumors of the Alimentary Tract. In: Meuten DJ ed. *Tumors in Domestic Animals*. 5th ed John Wiley & Sons, Inc; 2017: 548
3. Schroeder H and Berry WL. Salivary gland necrosis in dogs: a retrospective study of 19 cases. *Journal of Small Animal Practice* (1998) 39:121-125
4. Uzal FA, Plattner BL, Hostetter JM. Alimentary System. In: Maxie MG, ed. *Pathology of Domestic Animals*. 6th ed St Louis, MO: Elsevier; 2016: (2) 30

Case #3 (19-08154) – Erin Zachar, Prairie Diagnostic Services, and Ajaypal Kahlon, WCVM.

A 22Kg, 6 year old, sexually intact, adult female, black and tan German Shepherd dog with current vaccinations, deworming and no travel history. The dog was received for necropsy in a chilled, fresh state of post mortem preservation, with minimal autolytic changes and thin nutritional condition (BCS = 1.5/5). Red-brown fluid was passively draining from the nostrils and mouth. The esophagus was markedly enlarged from approximately 2cm distal to the epiglottis, to the diaphragm. The diameter of the esophagus ranged from 3.5cm to 5cm, with an approximately 10cm x 10cm x 8cm ventral out-pouching just cranial to the thoracic inlet. The intra-thoracic segment of the esophagus was diffusely distended, with an approximate diameter of 17cm at the widest point. The serosal surface of the esophagus was congested, roughened and red on the lateral and dorsal walls, and in ventral areas, the mucosa was thickened, edematous, and often covered by a fibrinonecrotic membrane. The esophageal lumen was filled with liquid tan to brown fluid with multifocal globules of yellow-brown mucus, flecks of white material (rice or finely grated potato?), and small chunks of food (green beans, carrots). The cranioventral areas of the lung were diffusely dark red to purple, thickened and firm, with multifocal miliary to approximately 0.5cm white spots on the serosal surface and within the parenchyma on cut section. The right caudoventral lung lobe was diffusely atelectatic (areas of lung adjacent to distended esophagus, diaphragm and cardia). The cardia, stomach and small intestine were grossly normal. The uterine horns were bilaterally mildly distended by clear, slightly mucousy liquid, and there were multifocal to coalescing small cysts throughout the entire endometrial surface (cystic endometrial hyperplasia). There were no other pathological abnormalities found on gross post mortem examination.

Histophathology: Esophagus: There are multifocal to coalescing areas of mucosal epithelial erosion and edema, often with transmigrating leukocytes. Blood vessels in the lamina propria are diffusely congested, lined by plump (reactive) endothelium, and contain frequent marginating and transmigrating neutrophils. There are often lymphoplasmacytic inflammatory cuffs surrounding lamina propria blood vessels. The lamina propria is diffusely expanded by dilated lymphatic vessels, multifocal areas of hemorrhage and a predominantly mononuclear inflammatory infiltrate. The submucosa is similarly expanded by congested blood vessels, often with lymphoplasmacytic cuffs, and dilated lymphatic vessels. The muscular layers are diffusely thinned, and the skeletal muscle is diffusely affected by one or more of the following changes: small, individual rounded myofibers; large myofibers with multiple centrally located nuclei (fiber splitting); swollen, hypereosinophilic myofibers; loss of cross-striations, central migration of the nucleus, hypercontracted myofibers, fragmented or vacuolated sarcoplasm (myocyte degeneration); mineralized sarcoplasm, and phagocytic cells (macrophages) within the myotube (myocyte necrosis). In some areas, the skeletal muscle is reduced to small (atrophic) rounded, individual, multinucleated myocytes. Myelinated nerves are prominent, but histologically normal.

The following tissues were not on the slide provided.

Lung: Affecting greater than 70% of the examined tissue sections, the tissue is diffusely congested, and there are multifocal to coalescing hypercellular areas, centered on small bronchi and bronchioles. Affected small airways are lined by flattened (attenuated) epithelial cells, and contain an inflammatory coagulum consisting of large numbers of viable and degenerate neutrophils with macrophages and multinucleated giant macrophages surrounding particles of foreign material (plant material, hair, muscle tissue), with necrotic cellular and nuclear debris, areas of hemorrhage, fibrin, high protein edema fluid, and occasionally small aggregates of bacteria. Alveoli adjacent to affected airways also contain similar foreign material surrounded by pyogranulomatous inflammation with the necrohemorrhagic debris, and alveolar septa are diffusely widened by congestion and predominantly neutrophilic inflammation. The

pleura and interlobular septa are widened by edema, congested blood vessels, dilated lymphatic vessels, hemorrhage, fibrin and the previously described neutrophilic inflammation.

Esophageal & Mediastinal Lymph Nodes (2): The capsule is thinned. Subcapsular, paracortical and medullary sinuses are expanded by clear space (edema), and increased numbers of neutrophils and macrophages (often with granular brown cytoplasmic material).

Uterus: Diffusely (affecting uterine horns and uterine body) the endometrium is expanded by cystically dilated and hyperplastic glands lined by cuboidal epithelium and often containing mucin and occasional small numbers of foamy macrophages. Diffuse cystic endometrial hyperplasia, chronic, moderate. This is a common change in older, intact female dogs, and considered incidental in this case.

Morphological diagnosis:
Esophagus: Diffuse, chronic, severe esophageal dilation (megaesophagus), with multifocal epithelial erosions and diffuse skeletal muscle degeneration, atrophy and necrosis.
Lung: Suppurative to pyogranulomatous and necrotic bronchopneumonia with intralesional foreign material and bacterial colonies, subacute, severe. (Aspiration pneumonia).
Esophageal & Mediastinal Lymph Nodes: Suppurative to pyogranulomatous lymphadenitis, subacute, moderate.

Etiology: Congenital idiopathic megaesophagus with secondary aspiration pneumonia

Comments: Congenital idiopathic megaesophagus is a fairly common clinical diagnosis in German Shepherds (as well as a few other breeds including Great Danes and Irish setter), and may be a congenital condition, or develop in mature dogs (this is the most commonly encountered presentation). As the name suggests, the underlying etiology of the disease is unknown, however, (according to JKP, page 34, and the reference paper), "there is strong evidence that CIM results from a selective defect in the distension-sensitive afferent autonomic arm of the reflex that coordinates esophageal function", and is not due to to failure of the lower esophageal sphincter to open or an esophageal blockage. The development of secondary megaesophagus in dogs has been associated with several underlying conditions including myasthenia gravis, administration of cholinesterase inhibitors, immune mediated polymyositis, polyradiculoneuritis, canine distemper, lead poisoning and snake envenomation among others. In this case, the condition was diagnosed when the dog was a puppy. On the provided history, it was stated that the dog had been managed conservatively by the owner since diagnosis. This included feeding the dog on an angled elevated platform, and holding the dog's head elevated for an hour after each meal, as well as preparing and blending the dog's food to a thin watery consistency to improve transit through the esophagus (although, small chunks of recognizable food were still present in the esophagus and stomach on post motem). According to the history, the dog was rarely noted to regurgitate, but "has never had respiratory issues whatsoever". Fatal aspiration pneumonia is the most common serious complication of megaesophagus.

References:

1. Quintavalla, et al. "Sildenafil improves clinical signs and radiographic features in dogs with congenital idiopathic megaesophagus: a randomized controlled trial". Veterinary Record. April 2017. 180 (16) 404; DOI: 10.1136/vr.103832
2. Uzal, FA et al "Alimentary System", pages 33-34. In: Jubb Kennedy and Palmer's Pathology of Domestic Animals, 6th edition, volume 2. Maxie, GM, editor. Elsevier, 2016.

Case #4 (Cytology Case) – Donal O'Toole, Wyoming State Veterinary Laboratory, University of Wyoming.

A 20 year old, well-cared-for afebrile American Quarter horse gelding developed abrupt onset depression, labored breathing, increased heart rate (60/min), and tachypnea (48/min). He was on a senior horse feed. His teeth were floated recently. The clinician aspirated opaque brown fluid she assumed was blood from one jugular furrow. She was puzzled by the in-clinic cytology. She submitted the fluid for analysis. The clinician's history noted that the horse was "kind of thin". A later conversation with the owner, a university student, revealed the horse was a hard keeper.

Cytology: WCVDP participants received photographic images, as it was impractical to submit an adequate number of cytology preparations. Cytology reveals a polymicrobial bacterial population, erythrocytes and effete unidentifiable cells, most likely leukocytes, and rafts of keratinocytes, some decorated by bacteria.

Disease: Choke

Etiology: Loose 1st upper left maxillary molar → pain → partly masticated senior feed → impacted feed bolus in distal esophagus → abrupt onset choke → aspiration pneumonia → welcoming arms of an uncaring God.

Ancillary testing: The horse's condition deteriorated. Following a diagnosis of choke, he was euthanized and a necropsy performed that day. The principal gross finding was acute bilateral anteroventral aspiration pneumonia with intralesional feed material, secondary to choke. Impacted feed material involved the distal 25 cm of esophagus. The underlying esophageal mucosa was essentially unremarkable. There was apical necrosis with incipient tooth loss of the 1st left maxillary molar. The affected tooth could be freed from the alveolus with little effort.

Discussion: Much literature on equine choke is in the clinical realm. Pathologists generally deal with catastrophic choke, including recurrent forms. Yet over the years we've had in a succession of acute choke cases, most terminating in aspiration pneumonia. This case was memorable in several respects. The most common clinical finding of acute choke – discharge of saliva and feed from nostrils – was absent at the time of clinical presentation since the horse already had acute aspiration pneumonia and was going into shock. The veterinarian inadvertently aspirated esophageal contents, which initially puzzled me. I teach a course in equine disease with this clinician, whom I respect - it did not occur to me that distended esophagus might be aspirated inadvertently, rather than jugular vein. The likely underlying problem was a painful tooth. Clinical texts mention as predisposing factors poor dentition (in older horses) and erupting teeth (in young ones). Choke is largely a disease of older horses, with dental problems ("issues" in post-Millennial speak) as prominent risk factor. Others are exhaustion, inappropriate feed, sedation or recovery from general anesthesia, strictures, trauma from nasogastric tubes, diverticulum formation, external compression of esophagus, and rapid ingestion of dry fibrous, pelleted, or cubed feedstuffs. In one large survey of choke in horses, 4 of 27 animals with feed impaction had predisposing dental disease. If you receive the carcass of a horse that died of acute choke, it is useful to check for dental disease since it may have been an important predisposing factor.

References:

Chiavaccini L, Hassel DM. Clinical features and prognostic variables in 109 horses with esophageal obstruction (1992-2009). *J Vet Intern Med*. 2010; 24:1147-1152.

Feige K, Schwarzwald C, Furst A, Kaser-Hotz B: Esophageal obstruction in horses: a retrospective study of 34 cases. *Can Vet J.* 2000; 41:207-210.

Murray, MJ: The esophagus. In: *Equine Internal Medicine*, eds. Reed SM, Bayly WM. WB Saunders Company, Philadelphia. pp. 610 – 612.

Case #5 (15-050596) – Maria Spinato, Animal Health Laboratory, University of Guelph, and Veronique S. Dufour, WCVM.

Clinical history and gross findings: A 6 yo female reindeer presented to the veterinarian with a history of weight loss over several months and a 2-week bout of diarrhea progressing to lethargy and anorexia the last 3 days. She was treated with a steroid, antibiotic, oral electrolytes, kaopectate and Panomec horse dewormer, but subsequently died and was submitted to the AHL for postmortem examination. Three other reindeers had died on this farm over the past 3 years with similar clinical signs. The remaining animals appeared healthy.

At postmortem examination, the reindeer was thin and moderately dehydrated. There was moderate dental disease characterized by uneven wear of molars with some sharp edges noted, in addition to slight wave formation. The rumen contained a 2 kg bundle of cloth admixed with feed and several clumps of hard brown material. Abomasum was empty and mucosa was markedly thickened and had a red-brown, granular to leathery surface. Small and large intestines were relatively empty. Liver was dark brown with sharp borders. There was mild hydropericardium.

Histopathology: Abomasal mucosa is mildly congested and contains elongated glands lined by cuboidal epithelium (mucous neck cells). There is marked depletion and atrophy of parietal cells. Mitotic figures are occasional seen in the upper portion of gland epithelium. A few glands are dilated by cysts or ruptured, filled with neutrophils and occasional eosinophils. The eosinophils can be seen, rarely, surrounding fragmented nematodes. Several of these nematodes are found in tunnels at the base of the mucosa; they contain large intestines lined by simple columnar epithelium and have external cuticular ridges (consistent with Trichostrongyles, such as Ostertagia). A few small lymphoplasmacytic aggregates are seen scattered within the mucosa and a few more lymphocytes are dispersed in the submucosa.

Diagnoses:

1) Moderate subacute abomasitis with glandular hypertrophy, mucous metaplasia, parietal cell atrophy and intralesional nematodes (Ostertagiasis)
2) Ruminal tympany with foreign body and bezoar
3) Abnormal dentition

Ancillary tests: Fecal flotation revealed 2+ GIN ova

Etiology: Parasitic (*Ostertagia* sp.)

Comments: Wild and captive reindeers are susceptible to trichostrongyles, including those found primarily in domestic ruminants (such as *Ostertagia ostergi* in cattle), and cervids may become infected by grazing pastures previously used by other livestock. Infections in these reindeers are often patent, perpetuating presence of infective stages within the environment. Disease due to the parasite, as well as gross or histological findings are often similar to other ruminants; chronic abomasitis, glandular hyperplasia and mucous metaplasia due to larval development within glands. Clinically, animals severely affected are often emaciated, hypoproteinemic and have elevated abomasal pH due to loss of parietal cells (mucosal metaplasia). Larval inhibition may be obligatory in certain environments, and this could potentially magnify emergence damages in affected reindeers.

References:

1) Terio, KA, McAloose, D, St Leger, J. Pathology of Wildlife and Zoo Animals. Elsevier Inc. 2018: 138, 168-169.

2) Hrabok, JT, Oksanen, A, Nieminen, M, Rydzik, A, Uggla, A, Waller, PJ. Reindeer as hosts for nematode parasites of sheep and cattle. Veterinary Parasitology 2006, Vol 136: 297-306

3) Hoar, BM, Eberhardt, AG, Kutz, SJ. Obligate larval inhibition of *Ostertagia gruehneri* in *Rangifer tarandus*? Causes and consequences in an Arctic system.

Case #6 (18-25991) – Erin Zachar, Prairie Diagnostic Services Inc., and Divya Jose, WCVM.

Clinical history:
An 18 month old bearded dragon was presented with one month history of not eating and doing poorly. Blood work showed high glucose, increased wbc counts and bacteremia. Euthanasia was elected due to poor prognosis.

Gross necropsy findings:
Received for necropsy was a 0.5 kg adult, male bearded dragon measuring 50.5 cm from nose to tail, which is consistent with a sexually mature adult over 1 year of age. The animal was submitted in a chilled state of post-mortem preservation and good nutritional condition (BCS = 3/5). There was approximately 2.3×1.5× 1.5 cm, firm, white to tan, nodular mass in the anterior stomach, approximately 2 cm distal to the heart. On cut section, the mass was multilobular, firm and white. There were similar, multifocal white masses within the kidneys and adhered to the coelomic surface and on the left abdominal fat pad. There were no other abnormal pathological findings on gross post-mortem examination.

Histopathology:
Stomach: The gastric mucosa is focally ulcerated and transmurally effaced by a poorly demarcated, unencapsulated, multilobulated, densely cellular, infiltrative neoplasm, which extends into the serosa. The cells are arranged in packets and nests saparated by thin strands of fibrovascular stroma. Cells are pleomorphic, polygonal with indistinct cell borders and scant to abundant granular eosinophilic cytoplasm. They have a central, hyperchromatic, round, oval or polygonal nucleus with finely stippled chromatin and 1-3 inconspicuous nucleoli. There is 2-3 times anisocytosis and anisokaryosis, frequent karyomegaly and 18 mitotic figures (a few bizzare) in 10 random high power (400X) fields. Occasional perivascular pseudorosette arrangement and frequent vascular invasion of the neoplastic cells were observed.

Variably sized nodules of cells with similar morphology (metastasis) were found within the liver, kidney, spleen, coelomic surface and the ventral abdominal fat.

Special stain (Grimelius) indicated strong positivity for neuroendocrine origin of the tumor.
The following tissues were examined with no significant histopathological lesions found: brain, heart, lung, trachea, esophagus, tongue, skeletal muscle, testis, hemipenis, small intestine and colon.

Morphologic diagnosis: Metastatic gastric neuroendocrine carcinoma

Comments:
The poor condition and anorexia in this bearded dragon was due to a metastatic neuroendocrine carcinoma, which has likely originated from the stomach. Case descriptions of a couple of case reports closely resembles the cell morphology and pattern observed in the present case.

References:

- Ritter JM et al. Gastric neuroendocrine carcinoma in bearded dragons (Pogona vitticeps). Vet. Path. 2009.46:1109-16 and

- Lyons JA et. al. A gastric neuroendocrine carcinoma expressing somatostatin in a bearded dragon (Pogona vitticeps). J. Vet. Diagn. Invest. 2010.22:316-320).

Case #7 (18-2934-16) – Gavin Fenske, Melissa Behr, Dale Miskimins, Joy Scaria, Veterinary & Biomedical Sciences Department, South Dakota State University, Brookings, SD

Enterotoxigenic Escherichia coli as the cause of Diarrhea and Deaths in Mink

A mink farm experienced illness and losses in adult mink during the winter of 2017-2018. Multiple submissions were made to the Animal Disease Research and Diagnostic Laboratory at South Dakota State University. The mink were noted to have a brief episode of diarrhea, anorexia and lethargy before death. Treatment with fluids and antibiotics was helpful if the signs were recognized quickly. Dead mink often demonstrated gastric hemorrhage and scant blood tinged intestinal contents upon post mortem examination. Hemolytic Escherichia coli (E. coli) were consistently isolated from intestines and other tissues. No other common mink diseases were identified. Small bacterial rods or coccobacilli were colonizing the intestinal epithelium, in which mild, degenerative and necrotic changes were noted at tips of some villi, with focal, mild villous atrophy. The bacterial isolate was sent to the E. coli Reference Center at The Pennsylvania State University for virulence testing. The isolate had estA and estB genes, indicating that the strain was an enterotoxigenic strain. The isolate also showed the presence of the colonization factor antigen operon (*cfaABC).* This syndrome would appear to be a new emerging and previously unrecognized disease of farmed mink.

Case #8 (19-2051 30-3) – Kelly Ramsay, Washington Animal Disease Diagnostic lab, Washington State University.

Clinical History: Two pigs from a 4-H herd of over forty 3-month-old Yorkshire pigs, died after one to two days of bloody diarrhea. The herd was dewormed two weeks prior.

Necropsy Findings: The ileum is diffusely, severely, and transmurally thickened with a prominent cerebriform pattern of projections and depressions as observed from the serosal aspect and thickened folds from the mucosal aspect. This change continues but attenuates in the caudal aspect of the jejunum. The submucosa is mildly expanded by clear, gelatinous fluid (edema). The mucosa is hyperplastic, with prominent ridges that are multifocally covered by organized, mottled dark red to bright red clots. The lumina of the cecum, spiral colon, distal colon and rectum are severely distended by large amounts of red, opaque, gelatinous material (hemorrhage).

Histopathology:
Ileum: The mucosa is diffusely and markedly thickened by crypts and glands that are increased in number and lined by epithelial cells that are tall and piled in disorganized layers (hyperplasia). The lamina propria is infiltrated by moderate numbers of lymphocytes and plasma cells and fewer eosinophils. Large numbers of crypts contain sloughed, brightly eosinophilic and karyorrhectic debris (crypt abscesses). Peyer's patches are mildly depleted of small, mature lymphocytes, and occasionally contain herniated crypts. Replicate sections stained with Warthin-Starry, large numbers of epithelial cells contain small numbers of silver-positive, approximately 1.5 micron long and less than 0.5 micron thick, curved, intracytoplasmic bacteria at the apical aspect. Free within the lumen and loosely adhered to epithelial cells are very large numbers of mixed bacteria (Gram-negative rods, Gram-positive rods and Gram-positive cocci on replicate sections stained with Brown-Hopps Gram).

Cecum and Colon: Multifocally, crypts contain sloughed, brightly eosinophilic and karyorrhectic debris (crypt abscesses). Superficial glands multifocally contain red, granular material (digested blood) with mixed bacteria. Goblet cells are moderately increased in number. The lamina propria is infiltrated by moderate numbers of lymphocytes and plasma cells. Free within the lumen and loosely adhered to epithelial cells are very large numbers of mixed bacteria, including many silver-positive spirochetes (on replicate sections stained with Warthin-Starry).

Morphologic Diagnoses:

1. Ileitis, proliferative and hemorrhagic, multifocal, subacute, severe with severe adenomatous hyperplasia and intracytoplasmic silver-positive short curved bacilli
2. Colitis, lymphoplasmacytic, multifocal, subacute, moderate with intralesional spirochetes

I**mmunohistochemistry**: There is positive immunoreactivity to *Lawsonia intracellularis and Brachyspira sp.* within replicate sections of the ileum and colon*, respectively.*

Etiologic diagnoses:
1. Lawsonial enterocolitis
2. Brachyspiral colitis

Comments: History, gross findings, histologic lesions and immunohistochemistry confirm that the cause of diarrhea and death in this case is due to the involvement of multiple pathogens of herd significance.

Lawsonia intracellularis is a gram-negative, curved rod, obligate intracellular bacterium. *L. intracellularis* infects a variety species and is the causative agent of porcine and equine proliferative enteropathy (PE), as well as wet-tail in hamsters. While the clinical presentation may vary, the characteristic histologic lesion in all animals is proliferation of the crypt epithelium within the distal small intestines and/or large intestines, with or without hemorrhage and necrosis. In pigs, animals from 3 weeks old to adults may be affected, though disease is most common in the postweaning period. Symptoms range from diarrhea to anorexia to failure to thrive, resulting in significant economic losses. Pigs can die peracutely from massive intestinal hemorrhage or after protracted diarrhea and weight loss. *L. intracellularis* replicates within the apical cytoplasm of immature, mitotically active enterocytes. Bacterial replication is directly associated with the observed epithelial hyperplasia. The precise mechanism is unknown, but is likely related to a host response, as *in vitro* infection does not result in cell proliferation. PE is endemic to all major swine industries globally. Subclinical shedders in combination with poor hygiene or improper environmental disinfection are thought to led to recurrent PE outbreaks in some swine operations.

According to the lab at which the immunohistochemistry was performed, the antibodies used for *Brachyspira sp*. are not necessarily specific to *B. hyodysenteriae*, and differentiation from *B. pilosicoli* would require PCR. Both bacteria are gram-negative, anaerobic, pathogenic spirochetes that infect the large intestines. *B. hyodysenteriae* is the causative agent of swine dysentery, while *B. pilosicoli* results in the syndrome intestinal spirochetosis. With both bacteria, transmission most commonly occurs via ingestion of infected feces from an asymptomatic carrier, though rodent vectors are another potential route. Both result in economic losses due to reduced growth rate. There is overlap in the signalment, clinical signs, gross findings and histologic lesions, with *B. pilosicoli* infections tending to be milder and less frequently affecting the cecum. Mortality is uncommon with *B. pilosicoli*, while it can reach up to 50% with *B. hyodysenteriae*. One unique but inconsistent feature of *B. pilosicoli* is the appearance of a false brush border on the apical surface of enterocytes caused by palisading of bacteria. This feature was not appreciated in this case. In addition, while *B. hyodysenteriae* is swine-specific, *B. pilosicoli* can infect several species, including chickens, rodents and humans. Finally, a novel pathogenic *Brachyspira* species, *B. hampsonii*, was recognized relatively recently that causes disease in pigs that is clinically indistinguishable from swine dysentery produced by B. *hyodysenteriae*.

References:

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2. Page, A.E., N.M. Slovis, and D.W. Horohov, *Lawsonia intracellularis and Equine Proliferative Enteropathy.* 2014. **30**(3): p. 641-658.
3. Vannucci, F.A. and C.J. Gebhart, *Recent Advances in Understanding the Pathogenesis of Lawsonia intracellularis Infections.* Veterinary Pathology, 2014. **51**(2): p. 465-477.
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Case #9 (17-10887 5-5) – Rachel M. Bone, Washington Animal Disease Diagnostic lab, Washington State University.

Clinical history and gross findings: An 11 week old, Yorkshire puppy with an initial limited history of sinus infection, and antibiotic administration was found dead by the owner and submitted for necropsy. Gross findings were limited and included diffuse transmural thickening of the jejunal wall and firm, dark pink, mottled lung lobes. After the necropsy, personal communication with the submitter revealed that this puppy had previous bouts of diarrhea and additional puppies in the litter had diarrhea at the time of death of this puppy.

Histopathology:

Small intestine: Crypts are multifocally, mildly to moderately dilated, lined by hyperplastic epithelium typically 2-3 layers thick, and occasionally filled by cross and longitudinal sections of adult nematodes, larvae, and eggs. Adult nematodes are folded within dilated crypt lumina and are ~25 um in diameter, with a smooth cuticle, platymyarian musculature, an esophagus, bilateral uteri that frequently both contain an egg, and a prominent ovary. Larvae are ~10-15 um in diameter and contain basophilic circular cells, and a linear intestinal tract. Eggs are ~20-25 um, shelled, and contain abundant magenta, granular material. The villi are occasionally blunted, with frequent individual necrotic enterocytes. The surrounding mucosal epithelium is infiltrated by moderate numbers of macrophages, and few lymphocytes and plasma cells. Occasional crypts are ectatic and filled by abundant sloughed epithelial cells and neutrophils (crypt abscess).

Lung (not submitted): Multifocal to coalescing alveoli contain abundant extravasated red blood cells, clots of fine fibrillar eosinophilic material (fibrin), and hemosiderin laden macrophages (siderophages). Occasional foamy macrophages are scattered throughout alveoli. Rare tangential sections of the previously described larva are seen in alveolar septa (approximately 2 in 100 40x HPF). There are few alveolar capillaries that are occluded by fibrin thrombi.

Morphologic diagnosis:

1. Enteritis, histiocytic, lymphoplasmacytic, acute, multifocal, moderate with crypt hyperplasia, crypt abscesses, and intralesional nematode- adults, larvae and eggs consistent with *Strongyloides* sp
2. Pulmonary hemorrhage, subacute, multifocal to coalescing, severe, with nematode larva consistent with *Strongyloides* sp.

Etiology: *Strongyloides* sp. enteritis with pulmonary migration

Comments: The cause of death in this puppy was attributed to multiple disease processes, with *Strongyloides* sp. infection and migration presumed to be the proximate cause of death. *Strongyloides* sp. are rhabditoid nematodes in which the parasitic female is characterized by having a paired genital tract, a prominent intestine lined by cuboidal cells, platymyarian musculature, and produce uninucleate eggs [1]. While the parasite in this case was not identified to the species level, one of the most important *Strongyloides* sp. known to undergo migration and cause hyperinfecton within immunosuppressed dogs and humans alike is *Strongyloides stercoralis*. This zoonotic pathogen is able to complete its whole lifecycle in a single host, leading to immense worm burdens[2,3]. The heavy worm burden coupled with the migratory nature of the larval stage can lead to catastrophic consequences for the host, such as life-threatening pulmonary hemorrhage[4,5]. The immune status of this animal may only be theorized, as no

known immunosuppressive drugs were administered, and only a presumed secondary bacterial rhinotracheitis was noted histologically.

Interestingly, within two months of submission of this puppy, an additional young Yorkie puppy was submitted to our lab with similar severe Strongyloides sp. infection. As the puppies were slightly different ages, and submitted from a different area of western Washington, it is unclear if the two cases were related.

References:

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Case #10 (15-046425) - Maria Spinato, Animal Health Laboratory, University of Guelph, and Pini Zvionow, WCVM.

Canine Intestinal Pseud-Obstruction: Lymphocytic enteric leiomyositis

Clinical history and gross findings: A 4-year-old female Bernese Mountain dog was presented with dilated intestines and suspected foreign body obstruction or pyometra. Animal was treated with Benazepril, Cerenia, and Famotidine prior to surgery.
An exploratory laparotomy was performed, and intestines were noted to be grossly dilated with air and liquid fecal matter. Two formalin-fixed full-thickness small intestinal biopsies and a biopsy of mesenteric lymph node were submitted to the AHL for histopathologic examination.

Histopathology: Three pieces of biopsied tissue, each measuring approximately 3 mm x 2 mm in size, were received and sectioned for histopathologic evaluation.

Small intestine (1): Two full-thickness small intestinal biopsies of duodenum and jejunum are examined. There are no differentiating features present. One biopsy contains tall villi lined by intact enterocytes and an adequate number of mucous goblets. Crypts appear slightly elongated with equivocal attenuation of epithelial cells. Several villi contain markedly dilated lymphatic vessels, and a marked, uneven increase in infiltrating leukocytes, including abundant neutrophils, some eosinophils and plasma cells. Crypt bases are separated from the muscularis mucosae by a cell-poor layer of collagenous stroma that contains a few dispersed eosinophils. Several dilated ruptured crypts at the edge of the biopsy may be due to squeezing artifact. The submucosa is mildly edematous. Myenteric ganglion cells appear viable. The tunica muscularis is significantly expanded by an inner layer of edema, with atrophy of myocytes. A few ganglion cells are visible at the junction of the muscle layers. There are numerous dispersed clusters of small lymphocytes, occasionally admixed with neutrophils throughout the tunica muscularis. A single large aggregate of approximately 50 neutrophils is situated within the outer layer of the tunica muscularis. The second biopsy is fragmented and contains scattered mucosal hemorrhage. Large strands of mucus containing colonies of bacteria are present overlying the luminal surface. Villi contain increased infiltration plasma cells and eosinophils; a dispersed accumulation of degranulating eosinophils is visible in the mid-mucosa. The tunica muscularis is similarly infiltrated by numerous dispersed small lymphocytes with admixed neutrophils. Rare mitotic figures are visible in the population.
Lymph node (1): The jejunal lymph node is markedly congested and has mild paracortical expansion. The subcapsular sinus is expanded by numerous hemosiderin-laden macrophages, karyorrhectic neutrophils and occasional eosinophils. A similar population of cells is distributed throughout medullary sinuses.

Diagnoses: Lymphocytic enteric leiomyositis

1) Moderate eosinophilic, plasmacytic and suppurative mucosal enteritis
2) Marked lymphocytic intestinal leiomyositis with localized myofiber atrophy
3) Mild suppurative and hyperplastic mesenteric lymphadenitis

Ancillary tests: None

Etiology: Suspected cell-mediated auto-immune disorder

Comments: Canine Intestinal Pseud-Obstruction Disorder (CIPOD) is a rare functional intestinal motility (ileus) disorder in dogs. It is well characterized in human medicine and can be classified as

congenital/acquired and primary (idiopathic)/secondary. Clinical signs include vomiting, regurgitation, anorexia, small intestinal diarrhea, abdominal cramps, dehydration, lethargy and wasting. Usually blood work and biochemistry are within normal limits. CIPOD has three histopathologic forms: neuropathic, mesenchymopathic affecting Cajal cells and myopathic. In this case, the myopathic forms seemed to predominate. Definite diagnosis is made by sampling full-thickness intestinal biopsies from multiple sites. The prognosis is poor and survival rate after diagnosis is short, usually few days to few weeks. Animals are usually euthanized due to poor performance and because they are refractory to treatment. Several immunohistochemistry markers could be used to further the diagnosis; for example, for the neuropathic form: neurone-specific-enolase (NSE) could be used to highlight neurons, S100 and GFAP for enteroglial cells and synaptophysin for degenerating neurons; KIT marker for the mesenchymopathic form and α1-smooth muscle actin filaments (SMA) for the myopathic form. Other markers such as CD3 for T cells, CD79α or CD20 for B cells, MAC387 or CD18 for macrophages, lysozyme (Lys) for neutrophils, and Van Gieson's (a special stain) for fibrosis and connective tissue.
On histology, the circular layer is more severely affected than the longitudinal muscular layer. The main infiltrative inflammatory cells are T lymphocytes, followed by B lymphocytes and histocytes. More chronic lesions may have fewer inflammatory cells, increased fibrosis and attenuated tunica muscularis layer. One case report showing a more pronounced B cell infiltration with fibrosis and angiogenesis was probably a more advanced form of the disease (Gianella, P., et al.).

References:

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Gianella, P., et al. "An unusual case of intestinal leiomyositis in a Bernese mountain dog." *Schweizer Archiv fur Tierheilkunde*157.10 (2015): 563-567.

Case #11 (17-094301) - Maria Spinato, Animal Health Laboratory, University of Guelph.

***Yersinia pseudotuberculosis* enterocolitis and lymphadenitis**

Clinical history and gross findings: A 2-year-old Boer doe in late pregnancy developed diarrhea, became recumbent and died. The veterinarian performed an on-farm postmortem and noted diarrhea and dehydration externally. Internally, the mesenteric lymph nodes were markedly enlarged, and scant pasty ingesta was present in the colon. A tentative diagnosis of Johne's disease was made, and formalin-fixed and fresh tissues were submitted to the AHL for diagnostic testing.

Histopathology: Multiple sections of **jejunum** are typified by subtotal villous atrophy and focal superficial mucosal erosion, accompanied by effusion of neutrophils into the lumen. There are numerous large colonies of bacterial coccobacilli within the lamina propria that are encircled by abundant degenerate neutrophils. The lamina propria is edematous and also contains a few thrombosed capillaries. The submucosa and serosa are expanded by edema, congested vessels and dilated lymphatics. **Colonic** luminal epithelium is sloughed due to autolysis; glands are lined by intact epithelium. One to two bacterial microabscesses are present in the mucosa of each cross-sectional profile, often associated with superficial erosion.
Mesenteric lymph nodes are lightly populated by lymphocytes. Numerous bacterial microabscesses are distributed throughout the node, and there is regional filling of the subcapsular sinus by bacterial coccobacilli, neutrophils and hemosiderin-laden macrophages.
Liver (not shown) contains several random foci of parenchymal necrosis and a few colonies of bacterial coccobacilli that are surrounded by degenerating neutrophils, mononuclear leukocytes and karyorrhectic cells.

Ancillary tests:
Bacteriology: Heavy (3+) growths of Yersinia pseudotuberculosis were isolated from cultures of jejunum and a swab of mesenteric lymph node.

Morphologic diagnoses:
1) Multifocal erosive and suppurative enterocolitis with intralesional bacterial colonies
2) Multifocal suppurative mesenteric lymphadenitis with intralesional bacterial colonies
3) Multifocal necrotizing and pleocellular hepatitis with intralesional bacterial colonies

Etiology: *Yersinia pseudotubercuolosis*

Comments:
Yersinia pseudotubercuolosis is a member of the gastrointestinal flora of several species of animals. Infection is thought to be fecal-oral following the ingestion of food or water suspected to be contaminated by wildlife, most commonly rodents and birds. The term "pseudotuberculosis" describes the tuberculoid appearance of granulomas in affected tissues. Clinical disease in domestic species usually affects only individual animals. Mastitis and abortion have also been reported in goats. Sporadic cases of ovine abortion due to *Y. pseudotuberculosis* have been diagnosed at the AHL. In other geographic regions such as California, cases of enterocolitis and/or abortion have been reported in cattle, llamas, water buffalo and sheep. This organism is a documented cause of food-borne illness in humans, and outbreaks have occurred following ingestion of contaminated milk, meat, fresh vegetables, and water.

Reference:

1) Giannitti, F, Barr, BC, Brito, BP, Uzal, FA, Villanueva M, Anderson M. Yersinia pseudotuberculosis infections in goats and other animals diagnosed at the California Animal Health and Food Safety Laboratory System: 1990-2012. JVDI 2014, Vol 26(1):88-95.

Case #12 (15-019828 B-6) - Maria Spinato, Animal Health Laboratory, University of Guelph.

Enteritis due to *Cryptosporidium sp*. and enteropathogenic *E. coli* (EPEC)

Clinical history and gross findings: A herd of approximately 420 Saanen dairy goats was experiencing diarrhea in 10-day-old nursery kids. Two kids were submitted to the AHL for full enteric work-up. Postmortem findings included dehydration, thin body condition and depleted internal fat stores. Small intestine were regionally filled with clear fluid and were minimally congested. Mesenteric lymph nodes were slightly enlarged. Large intestines contained semi-fluid to pasty yellow ingesta.

Histopathology:
Jejunum is typified by moderate villous atrophy, with abundant bacterial coccobacilli and cryptosporidial organisms visible overlying the brush border surface of enterocytes. Clusters of neutrophils are present in several crypts. **Ileum** has similar features with numerous cryptosporidial organisms attached to both villous and crypt enterocytes. Villi are stumpy with occasional fused villi evident. Peyer's patches are centrally depleted with lympholysis evident.
Colonic luminal and glandular epithelium is intact with an adequate number of mucous goblets. Abundant bacterial coccobacilli are attached to luminal enterocytes, often in association with clusters of neutrophils that are infiltrating superficial proprial stroma and transmigrating into the lumen. Also present within colonic sections are multiple cryptosporidial organisms that are attached to the brush border of luminal and colonic gland epithelial cells. Occasional clusters of neutrophils are present within dilated lumina of the more extensively parasitized glands.

Ancillary tests:
Parasitology: Sucrose wet mount of pooled feces revealed 3+ *Cryptosporidium spp*.
Virology: PCR of pooled small intestine was negative for rotavirus.
Bacteriology: *E. coli* was isolated from small and large intestines in relatively pure and heavy growths (2+ to 4+). *E. coli* agglutination serotyping was negative for F5/K99. Genotyping was positive for the presence of eaeA, EPEC, hlyA and negative for Shiga toxin 1, Shiga toxin 2 and VTEC. This genotyping pattern is compatible with enteropathogenic attaching and effacing *E. coli*.

Morphologic diagnosis: Moderate atrophic enteritis and mild suppurative enterocolitis with adherent protozoa and bacteria

Etiology: *Cryptosporidium sp*. and enteropathogenic *E. coli (*EPEC*)*

Comments: AHL submissions from 2010-2018 indicate that cryptosporidiosis and attaching and effacing strains of *E. coli* (AEEC) are the most commonly-diagnosed causes of enteritis in kids between 1-3 weeks of age. Both are best diagnosed by histologic examination of intestines fixed in formalin immediately following euthanasia or recent death (<6 h), followed by genotyping analysis of a pure culture of *E. coli* isolated from affected tissue, usually the colon. Several layers of AEEC bacteria attach to the microvilli covering the surface of enterocytes and injure or efface these important structures that are involved in fluid absorption and electrolyte transport, resulting in diarrhea. Compared to enterotoxigenic *E. coli* (ETEC), AEEC cause more severe mucosal damage and inflammation. AEEC are further subcategorized into 2 groups: enteropathogenic *E. coli* (EPEC) and verotoxigenic *E. coli* (VTEC). VTEC strains elaborate specific toxins, termed verotoxin or shiga-like toxin, the prototypical species being E coli O157:H7. Verotoxin is absorbed systemically and may produce severe clinical signs such as hemorrhagic diarrhea and septicemia. Some, but not all, strains of VTEC are potentially zoonotic.

Reference:

1) Merck Veterinary Manual:
http://www.merckvetmanual.com/mvm/digestive_system/intestinal_diseases_in_ruminants/diarrhea_in_neonatal_ruminants.html

Case #13 (94-4801) Tara Funk, WCVM.

Intestinal infarction caused by bovine histophilosis

Clinical history: A 5-month-old, male Hereford X calf was euthanized and submitted for post-mortem examination, following a history of depression, inappetence and joint swelling, with further deterioration to lateral recumbency.

Gross pathologic findings: Upon gross examination, many joints, involving all four legs, were markedly swollen and contained large amounts of fibrin. In the distal duodenum and jejunum, a small number of randomly distributed, well-delimited, raised, plaque-like lesions were identified on the mucosal surface. These areas were dull, red to brown and the surrounding intestinal mucosa was occasionally eroded or erythematous. Multiple irregular, dull, greenish foci were scattered throughout the myocardium, and the left ventricular papillary muscle was most severely affected. A scant number of small, hemorrhagic foci were present within the brain parenchyma.

Histopathology:
Intestine (ileum): The normal architecture of the ileum is focally disrupted and replaced by large accumulations of eosinophilic, fibrillar material (fibrin), numerous inflammatory cells, predominantly neutrophils and fewer macrophages, hypereosinophilic cells with pyknotic nuclei (necrotic cells), karyorrhectic debris and marked hemorrhage, all extending transmurally (hemorrhagic infarction). Within the adjacent submucosa, the lumen of several blood vessels is filled with fibrin admixed with similar inflammatory cells (fibrin thrombi) and the wall of multiple blood vessels is hypereosinophilic, disrupted and obscured by moderate numbers of degenerate neutrophils and karyorrhectic debris (fibrinoid necrosis) infiltrating the vessel wall. Rare intestinal crypts are filled with or replaced by karyorrhectic debris (crypt necrosis) and amphophilic, granular material (mineralization). The Peyer's patches are unaffected.

Diagnosis:
Intestine (ileum): Intestinal infarction; severe, acute, focal, transmural, hemorrhagic, pyogranulomatous, with multiple fibrin thrombi and marked necrotizing vasculitis

Ancillary test: Bacterial culture and Immunohistochemistry for *Histophilus somni*

Etiology: *Histophilus somni* (formerly *Haemophilus somnus*)

Comments:
Histophilus somni is an opportunistic bacterial pathogen of cattle and is a major cause of morbidity and mortality in beef and dairy calves across Canada. *H. somni* infection often results in a septicemia and can target multiple organ systems, including the lungs, heart, brain, joints and larynx. In order to cause disease, bacteria adhere to the vascular endothelium, resulting in contraction of endothelial cells. The underlying collagen is exposed, leading to platelet adhesion, thrombus formation and potential infarction, due to interruption of the blood supply. The development of clinical signs is associated with the extent of involvement of the affected organs.

References:

1. Chapter 4: Cantile C, Youssef S. Nervous System. In: Jubb, Kennedy & Palmer's Pathology of Domestic Animals. 6th ed (Volume 1). St. Louis, Missouri: Elsevier Ltd.; 2015:364-365

Case #14 (17-34191) – Roman Koziy and Enrique Aburto, WCVM.

Septicemic salmonellosis in a calf

History and gross findings: A 2-week-old, Holstein Friesian calf sick since 3 days of age; got progressively depressed and febrile (104F rectal T) despite treatment. This calf was euthanized, had loose stools but not fluid diarrhea. On gross examination, the calf was in fair body condition with normal muscle mass and moderate body fat stores. The eye globes were moderately sunken (dehydration). Moderate amounts of yellow-brown fluid feces stained the fur around the perineum, tail, and hocks. The lungs were diffusely wet, heavy, and non-collapsed with cranioventral areas of red discoloration and consolidation. The intestinal tract was flaccid, and distended with abundant tan watery fluid.

Histopathology: Liver: There are multifocal to coalescing variably sized areas of coagulative to lytic necrosis of the liver parenchyma. These areas are often partially surrounded or infiltrated by histiocytes, karyorrhectic debris, and fibrin deposits. The portal areas are often infiltrated by moderate numbers of histiocytes admixed with fewer lymphocytes and occasional neutrophils. Scattered blood vessels and sinusoids are occluded fibrin thrombi. Colonies of rod-shaped bacteria are also seen within the sinusoids.

Lung: Diffusely, the alveolar capillaries are congested. The bronchovascular interstitium and interlobular septa are mildly expanded by oedema and low to moderate numbers of lymphocytes and histiocytes. There are multifocal areas of necrosis and inflammatory infiltrates scattered throughout the parenchyma and usually centred on blood vessels. The infiltrates are composed of histiocytes, fewer lymphocytes, occasional neutrophils and necrotic cellular debris usually admixed with fibrin. Occasional capillaries and larger blood vessels are occluded with fibrin thrombi and colonies of rod-shaped bacteria (bacterial emboli). The alveolar spaces often contain small numbers of macrophages, red blood cells, cell debris and scant fibrin.

Similar areas of inflammation and/or necrosis were also seen in small intestine, kidney, spleen, mesenteric lymph nodes, epicardium and meninges.

Etiology: Bacterial culture from intestine, liver, and lung yielded significant growth of *Salmonella enterica* serovar Dublin.

Morphological diagnoses:
1. Liver: Hepatitis, necrotizing and histiocytic, multifocal to coalescing, severe, subacute with fibrin thrombi and bacterial emboli.
2. Lung: Pneumonia, necrotizing and histiocytic, multifocal to coalescing, moderate, subacute with fibrin thrombi and bacterial emboli.

Comments: The histomorphological features and bacterial culture are consistent with septicaemia caused by *S.* Dublin. In calves this cattle-adapted serovar is primarily associated with septicaemia, sometimes with localization to the liver, joints, bones, lungs and meninges. The characteristic paratyphoid nodules in the liver are prominent in this case, and may also be present in the spleen, kidneys (as in this case), lymph nodes and bone marrow. Lung lesions may include congestion, edema, interstitial thickening of alveolar septa by mononuclear cells, and thrombosis of septal capillaries in acute cases. In subacute salmonellosis, there may be cranial bronchopneumonia, usually with adhesions and abscessation. *Salmonella* is a facultative intracellular pathogen which can cause three major clinical signs in cattle: enterocolitis, septicemia and abortion. Pneumonia is commonly seen in calves with *S.* Dublin. *Salmonella* Dublin has the ability to persist in the herd for a long time. Some animals become life-long carriers and are important

sources of infection. Multiple tissue samples were negative for BVD virus by RT-PCR. Skin samples also tested negative for BVD by immunohistochemistry.

References:

- Uzal, FA et al: "Alimentary System", pages 174-175. In: Jubb Kennedy and Palmer's Pathology of Domestic Animals, 6th edition, volume 2. Maxie, GM, editor. Elsevier, 2016.

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Case #15 (19-08436) – Michael Zabrodski and Helene Philibert, WCVM.

Bovine Enteric Coronavirus and Rotavirus

Clinical History and Gross Findings:
Fresh and fixed tissues from a calf of unknown age were received. The producer was reported to be having an outbreak of neonatal diarrhea. On post-mortem exam, eyes were sunken, straw was present in the rumen, and some curdled milk and electrolytes were in the abomasum.

Histopathology:
Sections of colon, ileum and duodenum. There is marked villous atrophy with attenuation and frequent vacuolation of the surface epithelium along the tips of the villi, most prominent in the ileum. Underlying lamina propria contains necrotic debris. Crypts are hyperplastic with occasional acute necrosis and luminal debris. Peyer's patches are depleted with large number of apoptotic bodies. There is severe necrosis of colonic glands with gland drop-out and epithelial attenuation.

Morphological Diagnosis: Necrotizing enterocolitis, acute, severe with villous atrophy and necrosis of crypts/glands

Ancillary Testing:
Real time PCR was positive for Bovine Coronavirus and Rotavirus (CT values of 13.03 and 26.67, respectively). Immunohistochemistry for Bovine Coronavirus showed strong cytoplasmic immunoreactivity in epithelial cells of the villi in the ileum, glands of the colon and, to a lesser extent, in the crypt epithelium of the ileum. Immunohistochemistry for Rotavirus showed modest cytoplasmic immunoreactivity within the lamina propria of the villus tips in the ileum. Bacteriological culture was not significant and parasitology for, including fluorescent antibody for Cryptosporidium sp., was negative.

Etiology: Bovine Coronavirus and Rotavirus

Comments: Bovine coronavirus (BCoV) is associated with respiratory disease, winter dysentery syndrome in adult cattle, and diarrhea in 1 – 3-week-old calves. Other etiological agents may be seen in combination with BCoV in cases of calf diarrhea, including Bovine Viral Diarrhea Virus, Rotavirus, *Escherichia coli*, and Cryptosporidium. Findings at autopsy are generally consistent with undifferentiated calf diarrhea. Microscopic lesions of Rotavirus and BCoV in the small intestine are indistinguishable; villus atrophy with attenuation of surface epithelium are common resulting in maldigestion and malabsorption. BCoV infects the mature absorptive epithelial cells of the villi and is rarely reported to replicate in crypt epithelial cells. However, in field cases, severe villous atrophy and crypt necrosis in the ileum accompanied by severe depletion of Peyer's patches and severe necrosis in the glands of the colon appears to be the most common presentation. Rotavirus lesions, in contrast, remain limited to the small intestine. It remains uncertain if the depletion of Peyer's patches is attributable directly to viral activity or non-specific stress.

References:
Clark MA. Bovine coronavirus. *Br Vet J* 1993; 149: 51-70.

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Case #16 (19-15816-3) – Christiane Loehr, Oregon State University.

***Case Introduction**:*
A koi was found dead in its exhibit (outdoor pond) after a short period of showing exophthalmia, a swollen abdomen and buoyancy issues. On necropsy, the female, 11kg koi was in fair postmortem condition. The body / coelomic cavity was markedly expanded; the vent was very swollen and bright red. Multifocally, scales were elevated by edema and discolored dark grey (hemorrhage). Both eyes severely protruded from the sockets (exophthalmia) and had disseminated petechiation throughout the sclera. The gills were pale and filaments appeared swollen. The kidney and heart were covered with thick layers of white, soft material; the kidney was also severely enlarged and had numerous white nodules up to 5mm in diameter. The lumen of the digestive tract (stomach and intestine) contained mucoid red fluid and the mucosa was bright red and edematous. The spleen appeared enlarged, the liver uniformly tan. The swim bladder was within normal limits. *Histopathology:* The stomach has massive submucosal edema and perivascular histiocytic and lymphocytic infiltrates, and extensive ulceration. A diffuse mild to moderate histiocytic infiltrate expands the submucosa of the intestines. Mesentery has multifocal areas of fibrosis extending to swim bladder duct.

Other histological findings in this fish included severe necrotizing to granulomatous nephritis with multinucleate giant cells was present with bacterial colonies surrounded by Splendore Hoeppli material. Diffuse moderate histiocytic branchitis had infiltrates in primary and secondary filaments and the stroma of the gill arch. The skin had multifocal cutaneous hemorrhage and edema in the hypodermis; the heart mild multifocal lymphocytic to histiocytic perivascular epicarditis (panniculitis). Other organs including spleen and brain were within normal limits.

***Ancillary Tests**:* Culture of the intestine and kidney yielded large numbers of Aeromonas hydrophila, and large numbers of *A.* sp., *Bacteriodes* spp. including *A. fragilis*, *Actinomyces* sp., and *Clostridium* sp.

***Morphological Diagnosis**:*
Severe necrohemorrhagic gastroenteritis and caseating (granulomatous) nephritis (grossly also exophthalmia, cloacitis and coelomitis)

***Comments**:*
Gross necropsy showed hemorrhagic gastroenteritis and granulomatous nephritis. Histopathology identified widespread inflammation ranging from lymphoplasmacytic to granulomatous and necrotizing, especially in the kidney and digestive tract. While *Aeromonas* sp. can be commonly cultured in small numbers from most septic fish, the culture of large numbers of *Aeromonas hydrophila* from internal organs are a relevant finding in this case. *A. hydrophila* can cause widespread bacterial infections with hemorrhage, coelomic swelling and exophthalmia - as seen in this fish. Unfortunately, *A. hydrophila* is resistant to many antimicrobials. *A. hydrophila* is often a secondary invader; underlying predisposing conditions include stress and primary infections such as KHV. PCR for koi herpesvirus did not detect viral DNA. In this particular case, the introduction of this koi into a pond with chronic low-level losses due to "bacterial dropsy" may be a caused by insufficient immunity against a pond-specific bacterial strain or a sign of particular virulence of this *A. hydrophila*.

***References**:*

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Abdelhamed H, Ibrahim I, Baumgartner W, Lawrence ML, Karsi A. Characterization of Histopathological and Ultrastructural Changes in Channel Catfish Experimentally Infected with Virulent *Aeromonas hydrophila*. Front Microbiol. 2017 Aug 15; 8:1519. doi: 10.3389/fmicb.2017.01519. eCollection 2017. PubMed PMID: 28861049; PubMed Central PMCID: PMC5559642.

Case #17 (19-15107-4) – William O'Neill and Christiane Loehr, Oregon State Veterinary Diagnostic Laboratory, Oregon State University.

Four approximately 5-week-old Ring-necked pheasants (*Phasianus colchicus)* were necropsied after the producer noticed a gradual increase in mortality rates. The submitter stated that the animals were raised in a single barn that had been having issues with rats, and that ducks were also raised in the area. Two of the four birds were thin and postmortem autolysis was mild to moderate. All four birds had dilated ceca and duodena containing green to dark brown, pasty material within the lumens.

Histologic findings: Sections include ileum and ceca. In most sections, there are signs of intraluminal hemorrhage (free erythrocytes, pigment products). In well-preserved sections, there are areas of lymphoplasmacytic inflammation within the lamina propria. There is mild vasculitis and the serosal surfaces are lined by scattered leukocytes and occasional fibrin conglomerates (coelomitis). There are frequent intracellular (endothelial) basophilic, gram-positive organisms, occasionally associated with fibrin thrombi.

Other findings:
Parasitology: Abundant *Capillaria* ova and moderate numbers of coccidial oocysts
Bacterial culture: 3+ Mixed Gram positive and negative aerobic bacteria and mixed anaerobes; Swab from conjunctiva of one bird yielded 2+ *Mycoplasma* sp. growth
Immunohistochemistry: *Listeria* markers on the slide in this set were negative

Comments:
This case and the other pheasant case from OSU (19-15236) were submitted with an intent to compare and contrast. The most common organisms present in these 5-week-old Ring-necked pheasants were small, thin, basophilic, gram-positive rods. The most common bacterial agents associated with outbreaks in pheasants, such as *Escherichia coli* and *Salmonella* spp., are gram-negative and thus ruled out; others that are gram-positive like *Clostridium* spp. have a different morphology and were also ruled out. *Listeria monocytogenes* has been reported as a rare enteric pathogen in poultry, including pheasants, and was high on our differential list, but immunohistochemistry suggested an alternative etiology.[1] *Sarcocystis* spp., a protozoal organism, can have a replicative phase that looks similar to what we saw here. However, in previously reported cases, morbidity/mortality rates were lower and the intracellular schizonts/meronts less pleomorphic. *Mycobacterium* spp. infection was considered as possibility but much less likely given the acute onset and young age of the affected birds. Additionally, no acid-fast bacteria were identified. In the bird with the suspected catarrhal rhinitis, we obtained growth of *Mycoplasma*, suggesting that this bacterium may also be impacting flock health via respiratory infection. We recommended increasing husbandry efforts to provide a clean, dust-free environment for the following weeks to help reduce transmission rates and complicating respiratory distress. Our bacteriology section reviewed culture growth, hoping to obtain a prominent colony. With the degree of autolysis and postmortem growth, commensal and environmental bacteria outcompeted potential pathogens, making exact identification impossible. Follow-up information was not available, but no further submissions were received from this producer.

References:
Rothrock MJ, Davis ML, Locatelli A, Bodie A, McIntosh TG, Donaldson JR, and Ricke SC. 2017. Listeria occurrence in poultry flocks: Detection and Potential Implications. *Frontiers in Veterinary Science* 4:125.

Case #18 (19-15236-4) - William O'Neill and Christiane Loehr, Oregon State Veterinary Diagnostic Laboratory, Oregon State University.

Fifteen five-day-old Ring-necked pheasant (*Phasianus colchicus*) chicks were necropsied after an increase in baseline mortality rates was noted. Total flock size was not noted, but 10-15 birds were dying per day. Gross findings included mild to severe autolysis, dark red lungs in one bird, and a segment of dark brown, dilated small intestine in another bird.

Histopathology:
Tissues present include ventriculus, proventriculus, liver, and multiple sections of intestine. Preservation is poor in some sections. There are occasional clusters of histiocytes within the liver. Ingesta present in some sections is coarse and poorly digested. There are occasional necrotic, hypereosinophilic enterocytes. There are multifocal areas of lymphohistiocytic and heterophilic inflammation within the lamina propria of the intestine. Rarely, basophilic, bacterial rods line up on the surface of enterocytes.

Other findings:
Aerobic culture: 4+ *Salmonella* group C1, 2+ Mixed Gram-positive organisms
Clostridium perfringens culture: 2+ C. perfringens
Salmonella culture: *Salmonella* sp. detected - Group C1
Salmonella serotyping (Oregon State Public Health Lab) – *Salmonella enterica* serovar infantis

Comment:
This case and the other pheasant case from OSU (19-15107) were submitted with an intent to compare and contrast.

The most common organisms present in this submission of 15 pheasant chicks were Gram-negative rod-shaped bacteria that, in some areas, lined up along the surface of enterocytes. While we were not able to pursue further work-up to look for specific virulence factors (pathogenicity islands) within the bacterial culture, we do believe that these bacteria are responsible for this mortality event and consistent with *Salmonella*. Mortality from pullorum disease caused by *S.* Pullorum is most common in birds less than 2-3 weeks of age, which is consistent with this case. In chickens and turkeys, those that survive the acute infection can become chronically infected carriers. Transmission can be horizontal or vertical, with carriers passing the bacterium on to successive generations. There is limited information on the infectivity of *S. enterica* serovar infantis, but it is presumed to be similar to other serotypes.

References:
Shivaprasad HL and Barrow PA. 2013. Chapter 16: Bacterial diseases: Pullorum disease and fowl typhoid. *Diseases of Poultry*, 13th edition. Ed: Swayne DE. Wiley-Blackwell: Ames, IA, USA. Pp. 677-736.

Case #19 (EEHV-ht-om) - William O'Neill and Christiane Loehr, Oregon State Veterinary Diagnostic Laboratory, Oregon State University.

A female, juvenile Asian elephant (*Elaphas maximus*) within an AZA-accredited facility tested positive for endotheliotropic herpesvirus (EEHV) activity during routine monitoring through the Simthsonian's National Elephant Herpesvirus Laboratory. Shortly thereafter, the animal became lethargic and anorexic. Despite aggressive treatment with fluids, anti-virals, and transfusion therapy, the elephant passed away. At necropsy, there were multifocal areas of hemorrhage throughout most tissues, including the oral cavity and intestinal tract. The lesions were most prevalent in the heart and highly vascular organs.

Histopathology:
Multifocal areas of hemorrhage and edema were present throughout all tissues examined including the oral cavity and tongue submitted to the conference. In addition, there was mild lymphoid depletion in primary and secondary lymphoid organs. Rare, eosinophilic, intranuclear inclusion bodies were identified within endothelial cells within the heart.

Comment:
The primary target of elephant endotheliotropic herpesvirus (EEHV) is endothelial cells, throughout the body. Thus, hemorrhage and edema are not limited to the gastrointestinal tract but rather widespread, most notably in the endocardium. In this case, there were prominent, multifocal areas of petechial hemorrhage within the oral mucosa and serosal surfaces of the intestines. Due to the extensive workup performed by the zoo and the Smithsonian, there was minimal additional testing done at the OVDL. Asian elephants are susceptible to infections with (EEHV1A and EEHV1B) (Long 2016). In contrast, African elephants have their own host-adapted herpesviruses, EEHV2, 3, 6, and 7. Asian elephant calves are highly susceptible and prone to developing clinically fulminant, fatal infections. Currently, EEHV-associated disease is understood as a sporadic event rather than highly contagious, as is the case with many other herpes viruses. What, if any role latency and recrudescence play in the epidemiology of EEHV has not been determined.

References:
Long SY, Latimer EM, and Hayward GS. 2016. Review of Elephant Endotheliotropic Herpesviruses and Acute Hemorrhagic Diseases. *ILAR J*. 56: 283-296.

Case #20 (18-19638) – Ivanna Kozii and Enrique Aburto, WCVM.

Porcine proliferative enteropathy/salmonellosis

History and gross findings: Porcine farm, 320 sow F-F, PRRS/Mycoplasma hyopneumoniae positive. Wasting grower pigs. One of the affected pigs had enlarged thickened ileum on necropsy. Fixed tissue samples and swabs were submitted for histopathology and culture/sensitivity, respectively

Histopathology: The mucosa of the ileum is partially thickened with elongation, dilation, and branching of intestinal glands. The glands are lined by hyperplastic/dysplastic pseudostratified columnar epithelial cells with basophilic cytoplasm and frequent mitotic figures; goblet cells are markedly reduced in number within the affected areas. The hyperplastic glands sometimes protrude into the underlying submucosal lymphoid tissue. There is villus atrophy and crypt necrosis. The hyperplastic glandular epithelium contains numerous curved rod-shaped bacteria within the apical cytoplasm; these bacteria stain positive with Warthin-Faulkner special stain.

Morphological diagnosis: Proliferative enteritis, diffuse, severe with villus atrophy and numerous intraepithelial rod-shaped bacteria.

Etiology: *Lawsonia intracellularis* and *Salmonella* ser. Derbi

Comments: *Lawsonia intracellularis* elicits proliferative lesions of cryptal intestinal epithelium in a number of species. This obligate intracellular bacterium is an S-shaped rod that is gram negative and is microaerophilic. The curved rods can be highlighted in the apical cytoplasm of glandular epithelium with silver-stained tissue sections. The pathogenesis of porcine proliferative enteropathy (PPE) is related to active uptake of the bacteria by epithelial cells. Once the bacteria are engulfed by the host cell, they persists and replicate freely within the apical cytoplasm, causing hyperplasia of crypt epithelium. Cell division is required for bacterial replication; bacteria are transferred to daughter epithelial cells and exit via extrusion from the cytoplasm of enterocytes on villi or between crypt openings. The bacteria disrupts the differentiation of infected epithelial cells which appears to be a central event in the pathogenesis of the disease. Infected intestinal cells become a population of mitotically active and poorly differentiated cells; therefore, proliferation of the crypt epithelium is considered a primary lesion, and not an atypical regenerative response secondary to increased epithelial exfoliation. This explains the villus atrophy commonly seen in PPE. The gross changes are prominent in the intestinal serosa as cerebriform projections and depressions. When the infection is accompanied by intestinal hemorrhage it is recognized as a distinct syndrome called proliferative hemorrhagic enteropathy. Co-infections of *Lawsonia intracellularis* and Salmonella species are common. The diagnosis of *L. intracellularis* is based on typical gross and histomorphologic appearance. Confirmatory tests include immunohistochemistry and PCR. *Lawsonia intracellularis* infection remains to be a prevalent disease and should be included on the differential list of enteric diseases.

References:

- Uzal, FA et al: "Alimentary System", pages 177-180. In: Jubb Kennedy and Palmer's Pathology of Domestic Animals, 6th edition, volume 2. Maxie, GM, editor. Elsevier, 2016.

- Kroll JJ, et al. Proliferative enteropathy: a global enteric disease of pigs caused by *Lawsonia intracellularis*. *Anim Health Res Rev* 2005; 6:173-197.

Case #21 (19-19219) – Maodong Zhang and Ryan Dickinson, WCVM.

Equine Angiomatosis in Small Intestine

Multiple red, raised nodules distributed along the serosal surface of the normal and nonviable jejunum and were identified in a 24-year-old neutered male horse for removing the strangulating lipoma around the jejunum. Histologically, these nodules consisted of numerous significantly and variably dilated, blood-filled vascular channels lined by single layers of flattened, morphologically-unremarkable endothelial cells with occasional thrombi within a mildly thickened fibrous stroma. A diagnosis of intestinal angiomatosis was proposed. To the best of the authors' knowledge, this is the second report of small intestinal angiomatosis in a horse.

References:

1. Khan S, Pujani M, Jetley S, Neogi S. Angiomatosis: A Rare Vascular Proliferation of Head and Neck Region. *J Cutan Aesthet Surg.* 2015;8(2):108-110.

2. Lamm CG, Njaa BL. Ovarian and intestinal angiomatosis in a horse. *Vet Pathol.* 2007;44(3):386-388.

3. Shetty SR, Prabhu S. Angiomatosis in the head and neck--3 case reports. *Head Neck Pathol.* 2009;3(1):54-58.

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Case #22 (19-6963 8-4) – Chrissy Eckstrand, Washington State University.

Necrosuppurative enterotyphlocolitis in a dairy calf

Clinical history: 10 day old female Jersey heifer calf found dead. On-farm necropsy revealed an animal in good post-mortem condition and poor body condition. In the mid-body of the cecum was a focal 0.5 cm irregular area of serosal discoloration red/brown with fibrin strands at the edges. The mucosa was diffusely dark red.

Histopathology: In all sections of the cecum, and colon, the mucosa is multifocally to diffusely covered by a thick layer of neutrophils with admixed fibrin, hemorrhage and large numbers of superficial bacteria. Neutrophils transmurally expand the cecal wall. Large fibrin thrombi are multifocally present within the submucosa and there is multifocal complete devitalization/necrosis of the cecal wall. The lamina propria is expanded by neutrophils, lymphocytes, plasma cells and in areas of ulceration there are proliferating reactive fibroblasts. Crypts are multifocally regenerative. Diffusely, mucosal vessels are congested. Multifocally within the affected sections of colon, the submucosa is expanded by multiple variably sized clear spaces that are occasionally lined by thin endothelium (emphysema). These clear spaces extend all the way out to the serosa and mesentery. Tissue Gram-stains (Brown and Hopps) reveal large numbers of mixed bacteria on the affected colonic surfaces including Gram-positive short rods, Gram-positive large bacilli, and Gram-negative rods.

Morphologic diagnosis: Cecum, colon: Severe subacute diffuse fibrinosuppurative and necrotizing typhlocolitis with superficial bacteria and glandular regeneration

Ancillary tests: Rotavirus and Coronavirus detected by PCR; Cryptosporidium not detected by PCR, *Salmonella spp.* not detected by culture, *Clostridium difficile* culture and toxin testing negative, *Clostridium perfringens* type A identified by genotyping.

Comments: There are a myriad of infectious agents including bacteria, viruses and protozoa that can cause or contribute to the severity of diarrhea in neonatal calves. In this case the main clinical sign was diarrhea and histopathologic investigation confirmed severe intestinal disease. In a calf of this age (10 days old) the etiologic differential diagnosis list is long and includes (but is not limited to) Rotavirus, Coronavirus, *Clostridium perfringens* type C, Cryptosporidium, Coccidia, *Salmonella spp*. and K99 *E. coli*. Both Rotavirus and Coronavirus were detected in this calf, and while there is villous blunting, the fibrinonecrotic membrane seen in this case may be considered unusual for these viruses. *Salmonella spp.* was considered likely given the histopathologic findings; however bacterial enrichment and culture did not reveal its presence. An infectious etiologic cause for severe intestinal disease is undetermined in this case, which may be due to poor test sensitivity, the inability to identify/test for the definitive agent, financial constraints, or other environmental factors.

Case #23 (PC-5336-19) – Brenda Bryan and Vasyl Shpyrka, Manitoba Agriculture, Veterinary Diagnostic Services Laboratory.

Rectal Stricture in Swine

CLINICAL HISTORY:
A load of 26 hogs was delivered for slaughter. At ante mortem inspection 3 out of 26 hogs appeared to have distended abdomen and fair body condition. All three hogs were marked as suspected and segregated in a separate pen. 1 was killed on the day of delivery and condemned on a post mortem inspection for enteritis by the inspector in charge. 2 others were humanely euthanized morning of the following day and delivered promptly to the provincial diagnostic laboratory for necropsy and further investigation.

GROSS EXAMINATION:
Received dead pig previously euthanized by captive bolt. Female weighing 65kg. Body condition score was 3 of 5 (Body condition score handbook, saskatchewan.ca/livestock, Government of Saskatchewan) where firm pressure with the palm is needed to feel the hip bones and back bone, pelvic bones, ribs can be felt but with pressure and spine was covered and rounded. The face was slightly thin. Abdomen was moderately distended. There was pasty tan stool at the anus. Lungs were diffusely dark pink, soft, rubbery with caudal lobe rib impression. The heart was unremarkable. In the abdomen, the spiral colon was dilated approximately 2-4 fold and was filled with large volume of creamy tan feces proximally and pasty tan feces distally. The descending colon was dilated approximately 5 fold and filled with creamy tan feces. The descending colon formed a funnel shape approximately 5-6cm long and colonic wall of 5-6mm thick. At the level of the pelvic inlet, the descending colon and proximal rectum for a distance of approximately 4.5cm was strictured with a diameter of approximately 1.5cm. The distal rectum and anal opening diameter was approximately 7mm. On longitudinal cut section of the affected colon and rectum, the mucosa was proximally tan and shiny. There was a sharp ridge of rounded to scalloped demarcation between the dilated smooth grey to tan distal colonic mucosa and the creamy shiny smooth colonic mucosa at the pelvic inlet which merged into the strictured smooth mucosa of the pelvic colon and proximal rectum. The distal rectum and anus had red mucosa with rugae. Longitudinally split femur bone marrow was pink and greasy.

HISTOPATHOLOGY:
Colon/rectum: mild diffuse autolysis
Slide A Dilated distal colon: moderate lymphoplasmacytic lamina propria infiltrates, multifocal to coalescing crypt drop out. Mucosa muscularis was thickened approximately 3x. There were multifocal vessels with perivascular lymphoplasmacytic infiltrates. Serosa was thickened approximately 3-4x by edema and few diffuse random neutrophils.
Slide B Demarcation line: colonic mucosa ends abruptly, replaced by ulceration and granulation tissue infiltrated by many lymphocytes and plasma cells and rare neutrophils with scant surface fibrin and hemorrhage. Tunica muscularis round and longitudinal were thickened by fibroblast infiltrates and rarely there were vascular fibrin thrombi. Multifocal fibrovascular tags infiltrated randomly with few neutrophils was apparent on the dilated colonic serosa.
Slide C pelvic rectal stricture: colonic ulceration characterized by luminal surface pavemented with thin diffuse layer of nuclear debris and scant multifocal hemorrhage, and diffuse variable numbers of small rod bacteria and fibroblasts that are horizontally and front to back oriented. This surface rests on a 0.5mm layer of lymphocytes, plasma cells, small vessels and few fibroblasts all oriented horizontal and front to back. The tunica muscularis layers were not apparent and replaced by fibrous tissue

approximately 8mm thick with few multifocal hemorrhages and rare thick walled medium caliber vessels and few scant to mild lymphoplasmacytic cuffs. Multifocal fibrovascular tags were apparent on the dilated colonic serosa.
Slide D rectal stricture and anus: colonic mucosa and normal colonic muscularis layers abruptly begin and merges with the proximal anal mucosa.
Lung: alveolar high protein edema with multifocal variable hemorrhage (agonal).
Spleen: multifocal PALS had few collaring neutrophils (incidental).
Inguinal, submandibular, bronchial, mesenteric lymph node: unremarkable architecture.
NSF: liver, normal colon, small intestine, stomach.

SPECIAL HISTOCHEMICAL STAINS:
In development

VIROLOGY: RT PCR
PCV2 lung: weak positive Ct 30
PRRS lung: negative

BACTERIOLOGY:
-heart, spleen: no growth
-liver: Streptococcus pseudoporcinus 3+
-colon: no pathogens
-tonsil: Streptococcus pseudoporcinus 3+, Pasturella multocida 2+
- Lymph node mesenteric: E coli 3+, Streptococcus pseudoporcinus 3+
-Lymph node submandibular: Streptococcus equisimilis 2+

BONE MARROW ESTIMATED FAT %:
Split femur: 64%, greasy pink

FINAL DIAGNOSIS: Ulcerative colitis annular fibrosis, focally extensive, chronic (Rectal stricture).

FINAL COMMENT: Main findings was rectal stricture with dilation of spiral colon and descending colon. At the stricture there was abrupt loss of colonic mucosa (ulceration) and replacement by thick granulation tissue and fibrous tissue. Rectal stricture is thought to be as a result of ischemic proctitis (inflammation of the rectal mucosa), most likely related to infection with *Salmonella Typhimurium*. In a pig model of proctitis, there was mucosal damage with crypt depletion, macrophage infiltration and fibrosis, and expression of inflammatory cytokines. In humans, infections with Salmonella, Shigella and Campylobacter cause proctitis as well as chronic ischemic colitis secondary to arteriosclerosis or cardiac failure. Articles listed below describe the lesion and pathogenesis in pigs.
Lymph node and tonsillar bacteria reflect resident populations. In this pig, the estimated percentage of bone marrow fat was 64%. Bone marrow dehydration is a crude method to assess bone marrow fat percentage. In pigs with adequate nutrition, the percentage fat of the bone marrow is greater than 80%. Pigs with less than 20% bone marrow fat are known to have severe protein and caloric malnutrition. Given the bone marrow fat estimate of 64%, history, gross and microscopic findings the pig was experiencing chronic energy and protein deprivation malnutrition secondary to the rectal stricture.

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5. Pathologic basis of veterinary disease/ [edited by] James F. Zachary, sixth edition, 2017; pp: 368-369; 378

Case #24 (18-11157) – Ursula Perdrizet and Enrique Aburto, WCVM.

Rectal papillary adenoma in a dog

History and gross findings: A 10-year-old, male, Labrador retriever with episodes of fresh blood on normal consistency feces. A rectal polyp was found on clinical exam. The polyp was excised and submitted for histological assessment. The mass measured 1.3 x 0.8 x 1.1 cm.

Histopathology: The rectal mucosa is markedly expanded and effaced due to the proliferation of branching crypts, and short papillary projections lined by neoplastic columnar to cuboidal epithelial cells supported by a fibrovascular core. The neoplastic cells contain abundant basophilic cytoplasm and poorly defined cell margins. Their nuclei are round to ovoid, with finely stippled chromatin and 1-3 small magenta nucleoli. There is mild anisocytosis and moderate anisokaryosis. A total of 18 mitotic figures are counted in 10 randomly selected 400x fields. Loss of nuclear polarity and prominent nucleoli are seen in some areas (cell atypia). Small numbers of goblet cells are noted within the neoplastic crypts. Many crypts/glands are dilated (or ruptured) and filled with homogeneous eosinophilic proteinaceous material. Moderate numbers of lymphocytes, plasma cells, and neutrophils admixed with a few eosinophils infiltrate the supporting stroma. The neoplastic population appears to infiltrate the underlying smooth muscle and connective tissue.

Morphological diagnosis: Rectal papillary adenoma with marked epithelial dysplasia.

Comments: The microscopic findings in the submitted rectal mass are consistent with the diagnosis of rectal papillary adenoma (also called adenomatous hyperplasia, colorectal polyp, and polypoid carcinoma in situ). There is limited information on the biological behavior of these tumors. In dogs, complete surgical excision is usually curative. Recurrence may occur in polyps that are >1 cm in diameter, lesions containing cells with higher degree of epithelial dysplasia, sessile polyps with diffuse colonic involvement, or when lesions are multiple. In this case, the presence of neoplastic crypts within the underlying muscle at the rectoanal junction suggest either evidence of focal invasion or a plane of section artifact; regardless of this, the frequent cellular atypia in this rectal mass did not allow us to completely rule out the possibility of a well-differentiated adenocarcinoma.

In humans, intestinal polyps are most common in the colo-rectal region and are classified as inflammatory, hamartomatous, hyperplastic, and neoplastic. The most common neoplastic polyps are colonic adenomas, which are precursors to the majority of colorectal adenocarcinomas. It is know that regular surveillance colonoscopy and polyp removal reduces the incidence of colorectal adenocarcinoma in people; however, it is important to mention that the majority of adenomas do not progress to become adenocarcinomas. There are no tools presently available to determine which adenomas will undergo malignant transformation. Size is probably the most important characteristic that correlates with risk of malignancy. High-grade dysplasia is also a risk factor for cancer in an individual lesion.

References:

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Case #25 (19-17281) - Colby Klein, Igor Medici de Mattos, Juliana Sartori Lunardi, Ivanna Kozii, Sarah Wood, Elemir Simko, WCVM.

Clinical History: European honeybee hives were unwrapped and inspected in early May. One hive displayed marked fecal staining at the entrances and was notably weaker. Returning forager workers were collected and fixed in formalin. Several bees were dissected and had their abdominal contents examined histologically.

Gross Findings: Dissections of the fixed bee abdominal organs did not reveal abnormalities when inspected under the dissecting microscope.

Histopathology: Several bees have thickened ventricular walls caused by increased size and number of epithelial cells which contain abundant 4-5µm by 2-3µm ovoid organisms. The apical aspect of the epithelium is disrupted with loss of microvilli and peritrophic membrane. The lumen of the ventriculus, intestine and rectum contain sloughed infected ventricular epithelial cells and individual organisms from lysed cells. The underlying basal cells are less affected, with no organisms present beyond the basal lamina. The microsporidian organisms stain strongly positive (light blue to magenta) with acid fast stain and smaller numbers stain variably gram positive (dark blue).

Comments: Nosemosis is caused by the microsporidian *Nosema apis,* and the emerging pathogen *Nosema ceranae.* It is a common disease characterized by dysentery and fecal staining of the hive entrance during the spring season. Diagnosis is based on correlating clinical signs with a spore count of the macerated contents of 60 bees in solution via hemocytometer. Great than 10^6spores/bee is considered clinically significant. Definitive identification of the Nosema species requires PCR analysis. Sub-gross inspection of heavily infected bees may show ventricular thickening, loss of annular rings and increased opacity on fresh wet mounts but these changes were not observed in this case.

Case #26 (Bee larvae) – Sarah Wood, Elemir Simko, WCVM.

Clinical history and gross findings:
A commercial beekeeper has noticed numerous weak colonies this spring shortly after the first honey flow. Affected colonies have a 'shotgun' brood pattern with numerous dead, discolored larvae. The beekeeper discontinued oxytetracycline metaphylaxis two years ago.

Histopathology:
The larval midgut lumen is expanded by a mass of gram positive cocci.

Diagnosis:
Midgut. Acute diffuse bacterial overgrowth

Disease: European foulbrood

Ancillary test:
Gram stain, culture, PCR and multilocus sequencing

Etiology:
Melissococcus plutonius

Comments:
European foulbrood is a bacterial disease of honey bee larvae. Honey bee colonies experiencing nutritional or environmental stress are predisposed to this disease. Overgrowth of the bacterium in the larval midgut results in starvation and death of the larva.

References:
Takamatsu *et al*. 2016. J. Vet. Med. Sci. 78(1):29-34.

Case #27 (19-11243-1/2/8/9) – Shelagh Copeland, Prairie Diagnostic Services Inc., and Ashish Gupta, WCVM.

Mycobacterium avium ssp. paratuberculosis

Case Introduction: Fifteen-year-old female bison did poorly most of the winter and had very loose stools. It was on pasture with the rest of herd, fed hay and bison mineral block. Other younger animals have had diarrhea with no confirmed cause. Animal euthanized that morning and submitted for a post mortem. On post mortem, the animal was thin with pale mucus membranes and moderate accumulation of perineal dried fecal material. The small amounts of fat were yellow tinged. The esophagus had multifocal shallow linear erosions to possible ulcers. The liver had sharp borders. The rumen and abomasum were moderately full of most green forage; pH of abomasal fluid was 6 at ~ 3 hours after euthanasia. The mucosa of the lower abomasum was moderately thickened with a slight cobblestone appearance and marked ulceration of torus. Mesenteric lymph nodes had mild to moderately thickened irregular pale tan cortices, which were most marked in the ileocolic. The small intestinal mucosa from ~mid length posteriorly appeared slightly thickened, which was most marked ~ 30 cm from the junction with colon/cecum. The anterior colon also appeared mildly thickened. The digesta remained fluid in the colon with no formed fecal material.

Histopathology: The small and large intestine have moderate to marked increase of proprial plasma cells with small numbers of lymphocytes and a few to many giant cells and epithelioid macrophages sometimes in clusters. Villi are markedly shortened. The crypts and glands are occasionally dilated and lined by attenuated epithelium with mild accumulation of cellular debris and macrophages and infrequent necrotic cells. Affected glands and crypts are often rimmed by light fibrosis. Within the submucosa are multifocal light to moderate aggregates of lymphoid cells that often contain clusters of epithelioid and multinucleated macrophages. Epithelioid cells in some clusters contain light grey brown material and small amounts of refractile material**.** Intestinal lymph nodes have small to moderate numbers of epithelioid and multinucleated macrophages usually in clusters within paracortex. Perinodal tissue has occasional small clusters of lymphocytes and similar macrophages. The esophagus has multifocal extensive accumulation of neutrophils within corneum with numerous bacteria, often moderately long thin bacilli, on surface and infiltrating superficial layers. Affected corneum has multifocal erosion to ulceration with transmigration of neutrophils. Propria has mild infiltration of lymphocytes, plasma cells and neutrophils with a few thrombi and mild fibrinous exudation in areas of ulceration.

Ancillary tests: Numerous acid-fact bacilli were seen within epithelioid and multinucleated macrophages on Fite's. Ileum and intestinal lymph nodes were positive on real-time PCR (Ct 25.87) for *Mycobacterium avium ssp. paratuberculosis* (Tetracore Mptb kit, primers and probe target the hspX gene, which contains M. paratuberculosis-specific DNA sequences).

Morphologic diagnosis: Mild to Moderate Granulomatous Enteritis and Colitis; Severe Granulomatous Mesenteric Lymphadenitis; Moderate Esophagitis with Linear Erosion to Ulceration, Chronic

Etiology: *Mycobacterium avium ssp. paratuberculosis* (MAP)

Comments: Bison with Johne's may have weight loss without diarrhea. Typical intestinal corrugated lesions are often not present and thickening can be subtle on necropsy. Other causes may initially be suspected such as Ostertagiasis or as a component of weight loss as suspected in this case. PCR can be

negative for MAP as had occurred with two other submissions from this herd with histologically numerous acid-fast bacilli (ileum and colon). No attempts to culture the MAP from this case was done but should be considered for epidemiological studies and genotyping. Bison infections have been associated with a distinct "bison-type" strain of MAP related to the cattle type. "Bison-type" strains have also been found in dairy cattle in Canada. The esophageal lesion was included as in the submitter's experience it is not uncommon to find in ruminants with emaciation from many causes. It is not described in the literature but submitter wonders if secondary to inadequate protein, vitamins, and/or minerals causing increased fragility, decreased ability to repair or increased susceptibility to secondary infections.

References:

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Case #28 (19-24803-9+10) – Shelagh Copeland, Prairie Diagnostic Services Inc., and Javier Barrera, WCVM.

Jejunal Hemorrhage Syndrome

Case Introduction: Three-year-old purebred Charolais bull was put out onto pasture July 11 and found down the next morning bleeding from the rectum. Bull was able to rise, be loaded and transported to the veterinary clinic. On examination, the bull had tachycardia, distended abdomen with no pings, and a large rectal blood clot. On blood analysis, bull was alkalotic, hypochloremic, anemic and hyperlactatemic. Intestinal structures were thickened on ultrasound. The animal was euthanized due to grave prognosis and submitted immediately for a post mortem. On post mortem, the bull was in very good body condition with a severely distended rumen and abomasum by green fluid and digesta. The anterior jejunum had an area of marked reddening and thickening ~ 90 cm in length with fairly distinct borders and moderate accumulation of fibrin on serosa. The anterior edge was lightly telescoped into the adjacent intestine, which was easily reduced. The affected section contained large lightly adhered blood clots overlying a much-reddened mucosal surface. Anterior to the affected segment the small intestine had a small amount of blood and ~ four ulcers lightly rimmed by friable tissue with largest ~ 3.5 x 3 cm in diameter. Posteriorly the intestines appeared relatively normal but blood filled along its length with tarry changes in colon.

Histopathology: Small intestine with luminal blood clot has complete loss of underlying mucosa. The leading edge of the blood clot extends between the mucosa and muscularis mucosa lifting up the mucosa. Superficial layers of blood clot contain a mixed proliferation of bacteria, many of which are medium to long thin bacilli. The submucosa has moderate edema, mild fibrinous exudation and hemorrhage, and moderate numbers of neutrophils. Vessels have plump endothelium and those adjacent to ulceration have segmental mural necrosis. The adjacent intact mucosa has moderate dilation of lacteals with a few containing erythrocytes. Propria in this area has mild increase in neutrophils and moderate numbers of brown-green pigmented macrophages near mucosal surface. In the other two intestinal sections taken at the leading anterior edge of luminal clots and at a separate orad ulcerated area have loss of mucosa down to the muscularis mucosa and occasionally also into the submucosa. The underlying area has mild edema, moderate infiltration of neutrophils, mild to marked proliferation of fibroblasts, light hemorrhage and occasional macrophages with moderate amounts of brown pigment. Luminal surface has a membrane of fibrin, variable numbers of leukocytes, erythrocytes and cellular debris with mixed bacteria, imbedded plant material and a few fungal hyphae segments. In the most affected segment, there is neovascularization with a few submucosal arteries having moderate intimal and medial hypertrophy with one having also focal fibrinoid necrosis. In this segment, serosa has moderate accumulation of fibrin and degenerate leukocytes. Intact mucosa in both of these sections is similar to that already described but with minimal lacteal dilation. A few thin walled mucosal vessels are also thrombosed.

Morphologic diagnosis:
1. Subacute Severe Segmental Necro-Hemorrhagic Ulcerative Enteritis (Jejunal Hemorrhage Syndrome) and Anterior Gastrointestinal Obstruction due to Blood Clots.
2. Chronic Ulcerative Enteritis and Focal Peritonitis with Submucosal Vasculopathy.

Etiology: Unknown

Comments: The cause of jejunal hemorrhagic syndrome is unknown but is thought to be multifactorial. Risk factors proposed include feeding of silage or total mixed ration, feeding finely ground corn (or other finely ground starchy material), early stage of lactation, high level of production, free-choice feeding, improper mixing of ration, and abrasive material in the feed such as gravel. It has been proposed stress, excess gastrointestinal starch, and subclinical rumen acidosis may allow overgrowth of *C perfringens* type A bacteria in the gastrointestinal tract with production of α-toxin and possibly beta2 toxin. *Aspergillus fumigatus* has also been proposed as it has been isolated from affected animals but not from normal animals. *A fumigatus* also seems to cause a similar disease in humans. This is the second young beef bull the submitter has seen with this syndrome.

References:

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Case #29 (19-25601-1) – Shelagh Copeland, Prairie Diagnostic Services Inc., and Meghan Baker, WCVM.

Clostridium chauvoei

History/Necropsy Findings: Sudden death of bovine calves out on pasture in July with one very ill. One calf had pericarditis with black tissue/muscle and foul smell. The other calf had black area in muscle with gas. Fixed and fresh tissue submitted.

Histopathology: Ileum is mildly autolyzed and severely congested with a focally extensive area of transmural red tinged edema and hemorrhage with a few to moderate numbers of neutrophils. Lacteals in affected area are severely dilated by red tinged fluid. Erythrocytes are often lysed within stroma and in vessels. Small numbers of thin walled vessels in affected area and one submucosal artery have segmental loss of mural detail with small amounts of nuclear debris. Vessels otherwise have often plump endothelium. A few submucosal arteries have transmigrating neutrophils. Within the stromal hemorrhage and edema are scattered small to occasionally moderate numbers of small to medium sized Gram-positive bacilli with often a thin clear zone around them. Mucosal surface has small numbers of medium to long bacilli. Serosal surface has plump mesothelium and occasionally small amounts of fine fibrin and degenerate leukocytes. Villi have a few developing coccidia in enterocytes and luminal debris has a few oocysts. A few crypts contain eosinophils and/or neutrophils.

Morphologic diagnosis: Severe Acute Hemorrhagic Transmural Enteritis and Mild Peritonitis

Ancillary Tests: Intestinal Culture: *Clostridium chauvoei* 3+, *Bibersteinia trehalosi* 1+, Clostridium species 2+ (*C. bifermentans*). The fluorescent antibody test (FAT) on skeletal muscle with typical gross lesions of "black leg" submitted from another calf was positive for *C. chauvoei* and negative for C*. novyi, septicum* and *sordelli*.

Etiology: *Clostridium chauvoei*

Comments: We often do not think of *C. chauvoei* being associated with enteric lesions but there has been at least one reported outbreak[1]. The enteritis in this case may be secondary to circulating toxins released by *C. chauvoei* from an affected muscle site. However, the number of organisms cultured from the intestine and histological presence of bacteria compatible with *C. chauvoei* suggests it may have been more direct. It is thought that *C. chauvoei* spores initially enter through the intestinal lymphoid tissue. Perhaps if the animal ingests large numbers it may cause intestinal lesions. The coccidia found are thought to be an incidental finding versus causing sufficient damage to initiate the lesion. Bibersteinia trehalosi has been isolated from both healthy and diseased cattle. The *B. trehalosi* isolated from the intestines is also thought to be an incidental finding. The organism can be carried on nasal, conjunctival, and nasopharyngeal mucosa in healthy cattle. In the 1990's at the WCVM, the submitter had a case in feedlot cattle that died from a necrotizing colitis in which *C. chauvoei* bacteria, detected on FAT, were thought to be the cause.

References:

1. Harwood DJ, Higgins R J, Aggett DJ. Outbreak of intestinal and lingual *Clostridium chauvoei* infection in two-year-old Friesian heifers. Vet Rec 2007; 161: 307-308.

2. Abreu CC, Edwards EE, Edwards JF, Gibbons PM, de Araújo JL, Rech RR, Uzal FA. Blackleg in cattle: A case report of fetal infection and a literature review. J Vet Diagn Invest 2017; 29: 612–621.

Case #30 (19-25601-1) – Gwen Roy and Trent Bollinger, WCVM.

Red-tailed hawk Sarcocystosis and Avian Tuberculosis

A weak, emaciated, adult, female red-tailed hawk was submitted for rehabilitation at the WCVM in December 2018. On presentation it had no musculoskeletal reason evident for emaciation. The bird was euthanized approximately one month after admission due to increased respiratory effort of several days duration. The bird was frozen and submitted for necropsy. On gross necropsy there were multiple large granulomas within multiple tissues (lung, spleen, bone etc.). Histologic examination confirmed avian tuberculosis and aspergillosis and identified multiple parasitic organisms with the most fulminant parasitic infection being sarcocystosis within the small intestine. PCR confirmed *Mycobacterium avium* subsp. *avium* infection.

Histology description:
1. Within the lamina propria of the small intestine there are numerous protozoal organisms (sporulated oocysts) measuring approximately 15 um in length.

2. Within the splenic parenchyma there are multiple expansile areas of caseous necrosis and accompanying granulomatous inflammation. There are recognizable macrophages within this material and basophilic stippling. Surrounding the areas of caseous necrosis are multinucleated giant cells with multiple optically vacant vacuoles within the cytoplasm. Special stains showed as follows: Modified Fite's stain for acid-fast organisms stained large numbers of rod-shaped bacteria red, both within macrophages and extracellularly. Gram's stain stained both gram-positive (basophilic) and gram-negative (eosinophilic) rod shaped structures within macrophages. Giemsa staining demonstrated large numbers of blue, rod-shaped organisms within macrophages and extracellularly. No specific structures were identified using PAS stain (in this section).

Morph diagnosis:
1. Small intestine: Intestinal parasitism.
2. Spleen (lung, liver, bone etc.): Severe, multifocal to coalescing, granulomatous inflammation and caseous necrosis.

Significance of disease:
1. Sarcocystis sp. is a common incidental finding within the intestine of raptorial species.
2. Avian tuberculosis is an uncommon finding in both wild and domestic birds, but recently there have been concerns with avian tuberculosis in immunocompromised individuals.

References:

1. Fayer, R. Sarcocystis spp. in Human Infections. *Clin. Microbiol. Rev.* **17**, 894–902 (2004).

2. Schmidt, R., Reavill, D. & Phalen, D. *Pathology of Pet and Aviary Birds: Second Edition*. (2015).

Case #31 (19B-2865-92) – Donal O'Toole, Wyoming State Veterinary Laboratory, University of Wyoming.

A neonatal Red Angus-Simmental bull calf was born full term in 2019 with essentially no hair coat and without erupted teeth. A series of such calves was born on the property since 2013. This calf was kept indoors with his dam in a heated barn. He was bright and alert. Unlike previous affected calves, which were exposed to the elements and developed clinical pneumonia, this fellow had no signs of respiratory disease. He was euthanized and examined to establish whether this was hypotrichosis and anodontia defect (HAD), or a similar syndrome. Grossly, there were hairs in the pinnae, distal aspects of limbs, around the muzzle, and on the tail. No teeth were visible. Cheek teeth were palpable in the gums, and were visible radiographically.

Histopathology: This is a section of a cheek tooth about to erupt. I had looked forward to finding lesions of profound dysplasia in one or more cellular components: ameloblasts, cementoblasts, odontoblasts or mesenchymal components. While lesions are indeed present (mineralized concretions in papillary layer of ameloblasts), for the most part dental components were developing normally. The challenge for me was to recognize normal histological features, as well as artifacts caused by layers that slid or were mechanically disrupted during cutting in. Little has been published on the histology of developing bovine teeth, or on the histological hallmarks of dental dysplasia.

Disease: Hypotrichosis oligodontia; often referred to in mice and people as X-linked hypohidrotic ectodermal dysplasia; XLHED or XHED.

Etiology: Large deletion mutation spanning parts of *EDA* and all of *AWAT2*

Discussion: It is not unusual for diagnosticians to receive calls about bald neonatal calves, sometimes accompanied by the offer of photographs, with the question: what causes this? The point of the presented case is to highlight a relatively common form of hypotrichosis: mutations in *EDA*. *EDA* mutations leading to hypotrichosis-oligodontia of cheek teeth and anodontia of incisors are reported in cattle, dogs, mice (tabby strain), and children. Commonly associated changes are hypotrichosis and failure of development of ectodermal and endodermal glands, such as in nasal passages, trachea, and bronchi. These probably predispose to pneumonia. Sweat glands were present in the calf, but were abnormally dilated. The mutation described here, which was identified by Dr. Tosso Leeb and his group in Switzerland, is novel. Useful questions to ask about calves with hypotrichosis include: are bull calves alone affected (ADA mutations are X-linked); are teeth present or absent; do dams have abnormalities, particularly in hair coat and oral cavity. My assumption for this disease is that absence of EDA results largely in delayed eruption rather than profound dysplasia. The shape of teeth may however be abnormal.

References:

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2. Drögemüller C., Distl O. & Leeb T. (2001) Partial deletion of the bovine ED1 gene causes anhidrotic ectodermal dysplasia in cattle. Genome Research 11, 1699–1705.
3. Drögemüller C., Peters M., Pohlenz J., Distl O. & Leeb T. (2002) A single point mutation within the ED1 gene disrupts correct splicing at two different splice sites and leads to anhidrotic ectodermal dysplasia in cattle. Journal of Molecular Medicine 80, 319–323.

4. OMIM # 305100: Ectodermal dysplasia 1, hypohidrotic, X-linked; XHED. https://www.omim.org/entry/305100

Case #32 (No histology slide) - Delaney Schofer, WCVM, and Chelsea Himsworth Animal Health Centre, BC Ministry of Agriculture.

Small colon stricture secondary to rectal prolapse in a warmblood mare

Abstract
A warmblood mare (15 years old) produced a stillborn foal in the spring of 2018, resulting in dystocia and a severe (type 4) rectal prolapse, which was treated medically. Subsequently the mare had multiple episodes of small colon impaction which lead to euthanasia in June 2019. On gross examination the mare was in excellent body condition with good muscling and fat stores. There was a ~ 6' long impaction of the small colon which originated from an area of stricture that was ~ 2' proximal to the anal orifice. At the area of stricture, the colonic lumen was narrowed to < 1/4 of its normal diameter. There was a prominent, red, fibrous band in the mucosa of the stricture site that was oriented perpendicularly to the long axis of the colon and extended circumferentially around the affected area. The corresponding serosa and mesocolon were fibrotic and deformed. There were no other significant findings. On histopathology, at the stricture site there was full-thickness loss of the normal colonic architecture and replacement by fibrosis and neovascularization. Type 4 rectal prolapse in the horse is characterized by prolapse of the peritoneal rectum and a variable length of the small colon forming an intussusception through the anus. This type of rectal prolapse is most often associated with dystocia and can result in infarction of the distal small colon secondary to pressure/tension on the mesocolon and colonic vasculature. Type 4 prolapses have a low success rate for medical or surgical treatment, and most horses are euthanized shortly after the prolapse event. However, for horses that do survive, there are occasional reports of subsequent small colon stricture and impaction.

References:
Patrick T. Colahan, Alfred M. Merritt, James N. Moore, I. G. Mayhew. Equine Medicine and Surgery, Mosby 1998.

Edwards GB. A review of 38 cases of small colon obstruction in the horse. Equine Vet J Suppl 13 1992: 42-50.

Case #33 (No histology slide) – Carolyn Legge, UCVM.

Gastrointestinal pseudo-obstruction.

A 7-year-old, male-castrated, Border collie presented for a prolonged (> 12 day) history of weight loss with periodic vomiting and diarrhea. Clinical exam findings included 10% dehydration, generalized muscle atrophy, and a mild systolic murmur. Diagnostic imaging revealed a gas distended small intestine with lack of motility. Exploratory laparotomy (EL) revealed an absence of borborygmi and severe ileus in the small and large. Surgical biopsy samples of liver, stomach and jejunum were collected during the EL, but were not submitted for histopathology. Five days following EL, the patient decompensated and died. Postmortem examination revealed evidence of a diffusely dilated and flaccid gastrointestinal tract with fibrinous adhesions noted around the gastrotomy and enterotomy sites. Histopathological examination of the surgical biopsies and postmortem gastrointestinal sections revealed neutrophilic to lymphoplasmacytic inflammation centered on the inner circular and outer longitudinal smooth muscle layers of the gastrointestinal tract. Within the postmortem samples, the lesions were most dramatic in the small intestine and were milder changes present in the stomach and large intestine. Foci of vascular thrombosis noted in the lung and liver were suggestive septicemia, which was the most likely cause of death. The clinical findings and histopathological features were most consistent with a diagnosis of canine intestinal pseud-obstruction. Intestinal pseudo-obstruction (IPO) is a rare intestinal motility disorder reported in humans and animals. It is presumed to be the result of immune-mediated destruction of the intestinal smooth muscle. The prognosis for chronic IPO is generally considered poor as the intestinal smooth muscle loss is permanent. There has been only a single reported case of successful medical management of canine IPO following early clinical and histopathological diagnosis.