Ohio Department of Agriculture
ADDL
ANIMAL DISEASE DIAGNOSTIC LABORATORY

Animal Disease Diagnostic Laboratory
8995 East Main Street
Reynoldsburg OH 43068-3399
Phone: (614) 728-6220 Fax: (614) 728-6310

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Amended Report

Date Received: 4/13/2015
Collection Date: 4/13/2015

Case Coordinator: Dr. Jeffrey Hayes

Accession No: B1508573

MELISSA SIMMERMAN
8995 E. MAIN STREET
REYNOLDSBURG OH 43068
UNITED STATES

Phone: (614) 728-6279
Email: melissa.simmerman@agri.ohio.gov

Associated Parties

CC	Cindy Bodie	
Owner	Animal Disease Diagnostic Laboratory	
Veterinarian Submitter	Melissa Simmerman	Vet Code:8833
CC	Tony Forshey	

Animal Information

Taxonomy	Production Type	Sex	Age	Count
FELINE - Lion		Male Neutered	Adult	1

Diagnosis

- Rabies Neg, Human Exposure Unknown
- Intervertebral Disc Disease
- Degenerative Joint Disease
- Osteoarthritis
- Spondylosis
- Cholecystitis
- Cholestasis
- Cystadenoma, Biliary
- Cholangitis
- Hepatitis, Lymphoplasmacytic
- Cirrhosis
- Pneumonia, Interstitial
- Nephritis, Interstitial
- Periorchitis
- Urolithiasis

Comments

See page 2

Necropsy Report

Signalment: Intact male lion of unknown age
Animal Identification: "Leo," Accession # 15-1301-1
Body weight: 400 pounds
Nutritional status: Mild negative energy balance (BCS 2/5)
Hydration status: Mild dehydration
Tissue preservation status: Very good preservation

Clinical History:
The lion had chronic ataxia and was in lateral recumbency. It was anorexic with a history of watery diarrhea. It also was noted that there was a mass under the tongue.

Necropsy Examination:
Date of Necropsy 04/14/2015

Examined was one intact male lion of unknown age, weighing 400 pounds. Body fat stores were mildly present but appeared mildly diminished in the subcutis, perirenal, mesenteric, omental and epicardial regions (negative energy balance). Tissues were mildly dry and tacky, suggesting mild dehydration.

EXTERNAL EXAMINATION:
Several focal lesions were present on the skin at various locations, delineated as follows:
1. Left lateral hock, 42 x 32 mm, oval decubital ulcer with fresh blood;
2. Located 7.5 cm proximal to this lesion are 2 smaller skin ulcers, neither of which are bleeding: one is 23 x 13 mm, the other is11 x 18 mm.
3. Point of left hip, there is a focal 13 x 5 cm abrasion/excoriation,
4. Located 5cm proximal and ventral to this is a smaller focal excoriation which is 4 x 2 cm.
5. On the right medial second digit, on the lateral of foot pad, there was a focal oval abrasion, 11 x 8 mm.
6. On the same foot, the epithelium on the left side of the third digit, near the nail, and 18 x4 mm region was eroded and moist, with mild crust formation.
7. On the left lateral aspect of the face below the ear, there was an irregular 11 x 9mm area of hair loss and moist tissue, in the shape of a parallelogram / diamond.

All nails were worn, with no breakage or shredding seen.

The tail was 91 cm long, and the ventral and caudal aspects of were covered with dried brown fecal material (recent diarrhea).

There was a raised, oval firm 35 x 28 mm, mildly lobulated, smooth, tan to pink to gray, fleshy mass with a soft rubbery texture located on the ventral aspect of the tongue, extending to the right lateral lingual margin. The teeth were in good condition. No gross lesions were noted in the oral cavity.

INTERNAL EXAMINATION:
Yellow gelatinous fluid (subcutaneous edema) was present on the left side of the mandible, extending into the underlying fascia.

The heart had a mass of 1.1kilogram (kg), and was 17.4 cm (base to apex) x 14.6 x 9.8 cm. The right ventricular free wall was 4 mm, the left ventricular free wall was 23 mm, and the interventricular septum was 22 mm, There was 28 cc of mildly turbid serosanguineous fluid within the pericardial fluid (postmortem change, presumptive). The overall appearance was within normal limits although epicardial fat appeared mildly decreased.

The lungs were mildly congested, appearing within normal limits.
The trachea was dry and free of exudate.

The left thyroid gland was 63 x 39 x 7 mm and the right thyroid gland was 10.2 x 3.8 x 0.8 mm. Both appeared to be grossly within normal limits.

The liver had a mass of 4.37 kg. There were focal fibrous adhesions of the omentum to the gall bladder and bile duct. The bile duct was markedly elongated, tortuous, with thickened walls circumferentially, with the distal bile duct measuring 7 mm in diameter, and was wide as 12-14 mm where thickened. Incising the thickened bile duct revealed thick yellow to white turbid fluid in the lumen, which also contained multiple discrete dark green soft to very hard stones (choleliths) that together had a mass of 4 grams. Within the liver was a large round cystic structure, 33 x 33 mm, the wall of which was comprised of thick fibrous connective tissue. Within cavity of this cyst was filled a large amount of dark green soft choleliths (14 grams). There were numerous variably sized cysts throughout the parenchyma of the liver, several were approximately 10 x 5 mm, with the largest measuring 3-5 mm in diameter, with regions approximately 39 x 18 mm containing closely arranged cysts.

The left kidney was 14 x 9 x 7.4 mm, with a mass of 690 grams (gm). The right kidney was 14.5 x 9.2 x 8.6 mm, with a mass of 650 gm. Both organs appeared within normal limits grossly and upon cut sections.

The left adrenal gland was 60 x 40 x 10 mm, with a mass of 20.3 gm. The right adrenal gland was 56 x 53 x 9 mm, with a mass of 19.5 gm.

The spleen was irregularly U- shaped, was approximately 23 x 11-15 cm, and was 1.1-1.6 mm thick.

Three mL of clear yellow urine was aspirated by sterile syringe from the urinary bladder, There were two (2) discrete uroliths within the lumen of the urinary bladder. Each was spherical, translucent, off yellow, firm, with a sandy consistency. Each urolith was 4-5 mm in diameter. The bladder mucosa was grossly within normal limits (e.g., no hemorrhages, erosions, ulcers).

Stomach contained only a small amount of green mucoid fluid and one 11x3x3 cm trichobezoar weighing 43.9gm, consisting of firmly packed hair and plant material. The small and large intestines appeared within normal limits, with scant ingesta present in the lumen. Feces in the colon were firm (130 grams retained frozen).

The right testis was 48 x 38 x 29 mm, with a mass of 44.4 gm. The left testis was 52 x 38 x 34 mm, with a mass of 50.7g

MUSCULOSKELETAL SYSTEM
There was extensive degeneration and complete loss of intervertebral disc material involving multiple joints: between the fifth and sixth (C5-C6) and sixth and seventh (C6-C7) cervical vertebra, and between C7 and the first thoracic vertebra (T1), between T1-T2 and T2-T3. There were bony proliferations along the ventral aspects of the above listed vertebrae, resulting in fusion of the vertebral bodies to one another (spondylosis). Dissection of these joints revealed complete loss of disc material and articular cartilage, with exposure of subchondral bone which had been worn smooth (eburnation). Intervertebral discs between all other vertebrae appeared grossly normal.

There was no evidence of hip dysplasia or arthritis in either coxofemoral joint, The articular surfaces of the femoral heads and each acetabulum were within normal limits on gross inspection. The left femoral head was 35 x 42 mm, and the right femoral head was 44 x 41mm.

Necropsy Examination Diagnosis(ses):
General: Mild negative energy balance, mild dehydration
Tongue: Focal glossal neoplasm
Epidermis: Multifocal epidermal erosions and focal ulcerations
Left mandible: Mild focal subcutaneous edema
Liver / gall bladder: 1. Cholecystolithiasis, severe;
2. Bile duct hyperplasia and fibrosis, severe;
3. Cystic hepatopathy (hepatobiliary), moderate, multifocal;
4. Fibrous omental adhesions (chronic peritonitis), moderate, focal
Urinary bladder: Mild uroltihiasis (subclinical)

Stomach: Trichobezoar (subclinical)
Vertebral column (C5 through T3): Degenerative intervertebral joint disease and spinal osteoarthritis, with eburnation and spondylosis, multifocal, severe

Ancillary Test Selection:
Histopathology: In progress
Virology: Rabies testing of brain at Ohio Department of Health
Other: Tissues, choleliths, uroliths, urine, feces, hemolyzed serum retained frozen. Digital images of primary lesions described above are on file electronically.

Comments:
Necropsy of this animal revealed severe hepatobiliary disease and severe degenerative osteoarthritis involving five intervertebral joints (between fifth-sixth cervical vertebrae to second and third thoracic vertebrae). The latter condition was likely most involved with the reported clinical signs of chronic ataxia progressing to lateral recumbency and anorexia. In addition, severe cholecystolithiasis (gall bladder stones within intrahepatic and extrahepatic biliary tree) and subsequent degenerative cystic changes in the liver also were very likely also contributing factors to the deterioration of the animal's health. Other changes listed above were most likely secondary or incidental findings. Histopathology is in progress and results will be issued in an addended report.

Histopathology Report

Microscopic Description:
Slide 1:
Left mandibular lymph node (2 sections): Each section contains the following similar changes: 1) a focal loosely arranged aggregate of 20-30 macrophages within medullary sinuses that have coarse golden-brown granular cytoplasmic material (hemosiderin); irregular bands of fibrous connective tissue containing mature adipocytes that subdivide lymphoid tissue, effacing normal tissue architecture and resulting in atrophy and loss of lymphoid elements; 3) randomly scattered oval to round lakes of homogenous eosinophilic material (proteinaceous fluid) effacing both individual lymphoid follicles as well as medullary tissue; and, 4) moderate congestion of capillaries and blood vessels.

Slide 2:
Right mandibular lymph node (small section): This section contains all four changes listed for left mandibular lymph node.
Right cervical lymph node (large section): This section contains alterations 2,3 and 4 listed for left mandibular lymph node (no hemosiderosis noted).

Slide 3:
Heart (left ventricle): There is a focal region adjacent to a blood vessel in which fibrous connective tissue expands and separates individual cardiac myofibers (myocardial fibrosis), occasionally enveloping individual myofibers that are of diminished diameter in comparison to adjacent myofibers (myofiber atrophy). The epicardium contains abundant adipocytes that also are noted within the subjacent myocardium.
Heart (interventricular septum): No significant microscopic lesions.

Slide 4:
Aorta: No significant microscopic lesions.
Atrioventricular valve: The valve leaflet is moderately expanded by dense collagenous tissue (endocardiosis). No inflammation is associated with this change.

Slide 5:
Kidney (2 sections to include cortical surface to renal pelvis): There is mild and multifocal and irregular expansion of the cortical interstitium by pale eosinophilic fibrillar material that is not birefringent under polarized light (e.g., not collagen); amyloid deposition is suspected. This material appears to most often cause expansion of proximal tubular basement membranes, occasionally resulting in compression of tubular epithelium in individual tubules, with secondary atrophy and infrequent loss of tubules. This material occasionally partially envelops or expands Bowman's capsules of scattered glomeruli. There are a few scattered light infiltrates of lymphocytes and plasma cells in the deep cortical interstitium. The medullary tissue contains no significant microscopic lesions.

Slide 6:
Kidney (2 sections to include cortical surface to renal pelvis): Deposition of eosinophilic material in cortical interstitium and surrounding Bowman's capsules of scattered glomeruli is noted in the section of cortex. The section of medullary tissue is unremarkable.

Slide 7:
Spleen: The red pulp is moderately expanded by erythrocytes (congestion), resulting in apparent friability that causes fragmentation of the specimen (incidental change).
Pancreas: There are scattered areas within the exocrine pancreas in which there is extravasation of erythrocytes (acute hemorrhage). A source or cause of the hemorrhage is not evident in the section, which contains mildly autolytic changes evidenced by dissociation of cells from acini and mild loss of differential staining. Sheets of adipocytes with embedded fibrovascular tissue are noted in attached mesentery tissue.

Heart (right ventricle): No significant microscopic lesions.

Slide 8:
Adrenal gland (2 sections): Each section contains circumferential bands of fibrous connective tissue surrounding blood vessels in cortical regions (perivascular fibrosis, presumptive).

Slide 9:
Trachea: The well preserved section contains a complete layer of ciliated pseudostratified columnar epithelial cells with many goblet cells, with many submucosal glands noted also (within normal limits). No inflammation or other abnormal changes are noted.

Slide 10:
Thyroid gland (2 sections): Both sections contain sheets of variably sized follicles lined by low cuboidal epithelial cells surrounding lakes of homogeneous eosinophilic material (colloid). Variably sized sheets of adipose cells are attached to margins of the sections, occasionally being noted within the tissue, also.

Slide 11:
Thyroid gland (2 sections): As for slide 10.

Slide 12:
Tongue mass: There is a moderately circumscribed, non-encapsulated, mildly lobulated mass within the skeletal muscle of the tongue, which extends through ulcerated mucosal epithelium and is covered by mats of fibrin with enmeshed neutrophils, macrophages and erythrocytes. The mass is comprised of loosely arranged spindle cells as ribbons, interlacing bundles in a random pattern within a moderate eosinophilic fibrillar and vascular matrix. Spindle cells have fusiform to elongated eosinophilic cytoplasm, and in low numbers of such cells, cross striations are evident. Cell margins are often indistinct, blending into the fibrillar matrix. Round to oval vesicular nuclei vary in size (moderate to marked anisokaryosis) and contain one or two magenta nucleoli that also vary in size. Multinucleate cells resembling rhabdomyoblasts are noted (strap cells). Mitoses are low to moderate, ranging from 1 to 5 per 400x field (hpf), with a mean of 2.7 in ten hpf. No evidence of vacular or lymphatic invasion by neoplastic cells is observed. The mass appears to be completely removed as normal tissue is present between the mass and cut margins.

Slide 13:
Stomach (2 sections): There are no changes distinguishable from mild decomposition, shown by exfoliation and loss of superficial epithelium.
Small intestine (1 section): This well preserved section contains three dilated crypts, two of which contain loss amounts of cellular debris (cryptitis, an incidental finding).

Slide 14:
Stomach (fundus): As described for slide 13.
Stomach (pylorus): There is diffuse loss of superficial epithelium, attributed to postmortem change. No other alterations are noted.
Colon: No significant microscopic lesions.
Urinary bladder: No significant microscopic lesions.

Slide 15:
Small intestines (3 sections): There are no changes distinguishable from mild to moderate post mortem change. There is no evidence of inflammation, crypt epithelial damage or parasite structures (e.g, protozoa, nematodes, cestodes, trematodes).
Large intestines (2 sections): As described for small intestinal sections.

Slide 16:
Liver (1 of 5 sections): The section contains three large cysts that are approximately 5.4 x 1.0 mm, 680 x 620 microns and 480 x 430 microns in diameter, that are each bordered by a capsule so fibrous connective tissue (approximately 100-350 microns, 230 microns and 200 microns thick, respectively). Each capsule has an internal lining consisting of an epithelium of low cuboidal to flattened cells that have small amounts of eosinophilic cytoplasm and irregularly round to oval open faced nuclei that are compatible with attenuated biliary epithelial cells (biliary cysts). Portal triads in the section often contain 2-3, and up to ten, biliary ductule profiles (biliary hyperplasia).

Slide 17:
Liver (2 of 5 sections): This section contains at least ten cysts as described in slide 16 that are of varying shape and size, large cysts ranging up to approximately 2600 x 1650 microns (2.6 x 1.65 mm) to as small as 650x280 microns. These are often adjacent to one another and elongated / flattened, suggesting compression of adjacent individual cysts or tortuosity of a dilated ductal structure. In addition to biliary hyperplasia describe in slide 16, some portal triads are noted to contain abundant fibrous connective tissue that occasionally envelops individual hepatocytes (portal fibrosis). One margin of the section has a thick band of vascularized fibrous connective tissue that has a superficial lining of flattened marked attenuated cells (interpreted as mesothelial cells), indicating hepatic capsular fibrosis.

Slide 18:
Liver (3 of 5 sections): Features as described for slides 16 and 17 are present on this slide that includes at least 16 variably sized cystic structures that are in close proximity to none another, near the hepatic capsular surface, resulting in compression of low numbers of hepatocytes near the capsule, and entrapment of islands and aggregates of other hepatocytes by fibrous connective tissue.

Slide 19:

Liver (4 of 5 sections): This section contains one large elongated distorted cystic structure as described above, that is approximately 1.5 x 1.0 cm and that contains numerous small biliary ductular profiles within the fibrous stroma of its capsule, which also multifocally envelops aggregates of hepatocytes which appear small and atrophic. At least six other smaller diameter cysts are present in the section. Mild biliary stasis is evidenced by accumulations of eosinophilic fibrillar to homogenous material in the lumens of some biliary ductules, and by presence of golden brown homogenous material in the lumen of others and in the cytoplasm of portal macrophages.

Slide 20:
Liver (5 of 5 sections): The primary changes in this section are the presence of one large cystic structure as previously described, and mild portal fibrosis and biliary hyperplasia of portal triads throughout the section.

Slide 21:
Lung (2 sections): Alterations vary in distribution among these two and the next four sections of lung. Salient features include – 1) low numbers of lymphocytes, histiocytes, plasma cells in perivascular and peribronchial connective tissue (interstitial pneumonia);
2) low numbers of macrophages containing punctate black cytoplasmic granules (carbon particles,) in peribronchial regions (pneumoconiosis);
3) irregular and patch filling of alveolar spaces by flocculent to homogenous eosinophilic material (proteinaceous fluid representing pulmonary edema);
4) scattered aggregates of macrophages within alveolar spaces (alveolar histiocytosis); and
5) pulmonary vessels and alveolar septal capillaries filled with erythrocytes (pulmonary congestion).

Slide 22:
Lung (2 sections): As for slide 21.

Slide 23:
Lung (2 sections): As for slides 21 and 22.

Slide 24:
Liver / gall bladder: The wall of the gall bladder ranges from approximately 500 microns to more than 1000 microns (1 cm) in thickness, comprised of densely arranged collagenous connective tissue, especially in deep layers adjacent to hepatocytes (with occasional enveloping and entrapment of hepatocytes). More superficial aspects of the fibrous wall of the gall bladder consists of densely arranged layers of spindle cells with scant elongated to fusiform cytoplasm and oval compressed hyperchromatic nuclei (fibroblasts and fibrocytes, some of the former noted undergoing mitosis). Moderate numbers of lymphocytes, plasma cells – several of which contain large eosinophilic cytoplasmic granules (Russell bodies) – and histiocytes infiltrate the fibrous stroma (cholecystitis). Low numbers of macrophages contain golden homogenous cytoplasmic pigment (bile) and others contain coarse golden brown cytoplasmic granules (hemosiderin). Moderate biliary hyperplasia and portal fibrosis are noted in portal triads in the subjacent hepatic parenchyma.

Slide 25:
Bile duct: Seven variably sized cross sections of this structure are presented. Large numbers of lymphocytes, plasma cells, macrophages and fewer neutrophils expand the lamina propria of all sections, and infiltrate the peripheral fibrous stroma, often in a perivascular pattern in the latter region. Low numbers of macrophages contain golden homogeneous cytoplasmic pigment (bile). One section contains large numbers of neutrophils ad mixed with mononuclear cells and exfoliated columnar epithelial cells in the lumen. Two sections contain a variably sized coagulum of homogenous dark golden brown material (inspissated bile / choleliths), the larger of which is colonized by small basophilic coccoid-like structures less than 3-4 microns (bacteria, presumptive). The walls of each section are markedly expanded by fibrocytes and collagen bundles as noted for gall bladder (fibrosis). In addition, the numbers and volume of smooth muscle fibers in each section appears to be markedly expanded (smooth muscle hypertrophy and hyperplasia).

Slide 26:
Pancreas / mesenteric lymph node: There are no changes distinguishable from moderate autolysis in the section of pancreas. Three sections of adjacent mesenteric lymph nodes contain moderate numbers of macrophages in medullary sinuses and cords that have homogenous to finely granular golden cytoplasmic pigment (bile). Aggregates of large bacilli in the lumen of blood vessels and in subcapsular sinuses of lymph nodes are interpreted as postmortem bacterial overgrowth (no associated inflammation present). Extravasation of erythrocytes in adjacent mesenteric tissue appears as an artifact of sample collection. The mesentery contains sheets of adipocytes within a fibrovascular stroma.

Slide 27:
Brainstem (medulla oblongata): No significant microscopic lesions.
Pituitary gland: No significant microscopic lesions.

Slide 28:
Cerebellum: No significant microscopic lesions.

Slide 29:
Mesencephalon: No significant microscopic lesions.

Slide 30:
Cerebrum (2 sections): No significant microscopic lesions.

Slide 31:
Spinal cord (4 of 18 sections): No significant microscopic lesions in four sections examined.

Slide 32:
Spinal cord (2 of 18 sections): No significant microscopic lesions in two sections examined.

Slide 33:
Spinal cord (4 of 18 sections): No significant microscopic lesions in four sections examined.

Slide 34:
Spinal cord (4 of 18 sections): No significant microscopic lesions in four sections examined.

Slide 35:
Spinal cord (4 of 18 sections): No significant microscopic lesions in four sections examined.

Slide 36:
Right coxofemoral joint synovium: No significant microscopic lesions.

Slide 37:
Left coxofemoral joint synovium): No significant microscopic lesions.

Slide 38:
Sciatic nerve (transverse and longitudinal): No significant microscopic lesions in either section examined.

Slide 39:
Testis: No active spermatogenesis is observed and no spermatozoa are present in the lumen of seminiferous tubules (atrophy).

Slide 40:
Testis: Within the tunica albuginea, there is a dense band of mixed mononuclear leukocytes including macrophages, lymphocytes, fewer plasma cells and numerous hemosiderin-laden macrophages (siderophages). There is a split in this band of inflammatory cells of this capsular structure, with two focal aggregates of purulent exudate in the lumen of this cystic space, that consist of neutrophils, macrophages, low numbers of erythrocytes and streaming nuclear debris. Very small pale eosinophilic granular structures, possibly bacterial cocci, are within this material. No active spermatogenesis is observed and no spermatozoa are present in the lumen of seminiferous tubules (atrophy).

Slide 41:
Intrahepatic biliary cyst: This intact structure presented on a slide has a capsular diameter of 37 mm and a luminal diameter of 25 mm. No lining epithelium is present along the innermost aspect of this large cyst within the liver (attributed to postmortem changes). Multiple variably sized mats of fibrin admixed with entrapped macrophages and viable and degenerate neutrophils adhere to the inner surfaces of exposed collagen bundles, in which moderate numbers of lymphocytes, plasma cells and macrophages are noted. Fibrous connective tissue emanates from the large cyst and dissects into and irregularly effaces normal architecture of the liver parenchyma (cirrhosis), entrapping small islands and aggregates of hepatocytes as well as obviously resulting in loss of parenchymal mass. Large numbers of mixed mononuclear cells infiltrate this fibrous tissue and infiltrate the parenchyma, also (hepatitis).

Slide 42-44:
Vertebral body (3): Two of three sections contain fibrocartilage on the articular surface that is eroded and fibrillated on the surface , accompanied by very fine basophilic granular material (mineralization) of frayed regions of this cartilage. No normal hyaline cartilage is present in any section. Subchondral bone often has many lacunae that are void of osteocytes (osteonecrosis). Changes in a third section are inconclusive.

Morphologic Diagnosis:
Left and right mandibular lymph nodes: 1. Mild focal hemosiderosis; 2. Moderate lymphoid atrophy and loss with fibroadipose tissue replacement and multifocal cystic degeneration. 3. Moderate congestion
Right cervical lymph node: 1. Moderate lymphoid atrophy and loss with fibroadipose tissue replacement and multifocal cystic degeneration. 2. Moderate congestion

Heart (left ventricle): Mild myocardial fibrosis and mild cardiac myofiber atrophy

Atrioventricular valve: Moderate atrioventricular valvular endocardiosis

Kidneys (left and right): 1. Mild multifocal cortical nephropathy (amyloidosis, presumptive) – both kidneys; 2.Mild multifocal lymphoplasmacytic interstitial nephritis – one kidney

Adrenal glands: Mild perivascular fibrosis

Tongue: Rhabdomyosarcoma, presumptive

Liver: 1. Marked multifocal and coalescing biliary cysts (5 of 5 sections); 2. Moderate multifocal portal fibrosis and biliary hyperplasia (5 of 5 sections); 3. Mild biliary stasis (1 of 5 sections); 4. Focally extensive hepatic capsular fibrosis (1 of 5 sections)

Lung: 1. Mild multifocal subacute to chronic lymphohistiocytic interstitial pneumonia with pneumoconiosis
2. Mild pulmonary congestion and edema with mild alveolar histiocytosis

Gall bladder/liver: 1. Marked multifocal and coalescing chronic lymphoplasmacytic and histiocytic cholecystitis with severe fibrosis and mild bile stasis and hepatic hemosiderosis; 2. Moderate portal fibrosis and biliary hyperplasia

Bile duct: Severe diffuse chronic lymphoplasmacytic to chronic suppurative cholangitis with marked intramural fibrosis, smooth muscle hypertrophy and hyperplasia, mild bile stasis and intralesional bacterial cocci (presumptive)

Testes: 1. Testicular atrophy, bilateral; 2. Focally extensive chronic purulent periorchitis with hemosiderosis and presumptive intralesional bacterial cocci

Intrahepatic biliary cyst: 1. Marked focal biliary cyst with severe multifocal chronic fibrinopurulent cholangitis;
2. Marked multifocal and coalescing chronic lymphoplasmacytic hepatitis with hepatic fibrosis, hepatocellular atrophy and loss (hepatic cirrhosis)

Vertebral bodies: Degenerative joint disease, moderate to marked – 2 of 3 sections

Comments:
These comments are listed in the same order as the histologic slides were presented and examined, NOT by degree of clinical significance.

Hemosiderosis noted in the left and right mandibular lymph nodes suggests resolution of either drainage of a site of hemorrhage (e.g., oral cavity), or of prior focal hemorrhage within those structures (no active hemorrhage observed at the time of specimen collection). Fibroadipose tissue infiltration, lymphoid atrophy and loss and cystic degeneration in all lymph node sections are consistent with aging changes (senescence). There was no evidence of lymphadenitis in any section, or of intra-nodal or metastatic neoplasia in any section.

Mild myocardial fibrosis and mild cardiac myofiber atrophy observed in the section of left ventricle is regarded as an incidental aging change, and likely was not of clinical significance at the time of death. However, this change may have increased in distribution and severity with time to the point of becoming a clinically significant problem. No evidence of cardiomyopathy was observed in the two sections of heart examined.

Moderate atrioventricular valvular endocardiosis is commonly seen in older animals as a benign aging change.

Pathologic alterations in the sections of liver, gall bladder and bile duct confirm the macroscopic observations of severe disease of the liver and biliary tree including hepatitis, early changes of cirrhosis, extensive cystic biliary disease, cholangitis and cholecystitis, in addition to macroscopic findings of severe cholelithiasis (gallstone formation) involving an intrahepatic cyst, the gall bladder and bile duct. Without extreme intervention efforts, it is considered very likely that the sum total of lesions observed were irreversible and irreparable.

Alterations noted in the kidney sections were mild and multifocal, and were of doubtful clinical significance. Mild mononuclear cell infiltrates noted in one of the sections is regarded as an incidental, non-specific finding.

Mild perivascular fibrosis in adrenal gland sections is of uncertain significance without sections of an age-matched control. This alteration is regarded as an incidental aging change.

The presence of strap cells and cross striations in neoplastic cells warrants consideration of this mesenchymal neoplasm to be of striated muscle origin. Examination of the mass with a panel of immunohistochemistry assays at a referral laboratory (e.g., vimentin, actin, fast myosin, smooth muscle actin, and myoglobin) would be needed for confirmation. Features of atypia (anisokaryosis, double nucleoli of varying size, mitotic index) are compatible with a malignancy.

Periorchitis noted in the tunica albuginea connective tissue capsule surrounding testicular tissue of one testis is of uncertain origin. The presence of linear bands of siderophages is compatible with resolved hemorrhage surrounding a focus of purulent exudate (pus), which suggests a bacterial component. It is uncertain but considered doubtful that this was a clinically significant event, and appeared to be at least several weeks' in duration.

Microscopic findings of three affected vertebra sections are compatible with gross observations of degenerative disc disease and osteoarthritis involving multiple vertebral bodies in the distal cervical and proximal thoracic regions of the spinal column.

Executive Summary

EXECUTIVE SUMMARY
Necropsy and histopathologic investigation of this animal revealed extensive chronic conditions of the vertebral column involving vertebrae C5, C6, C7, T1 and T2, and of the liver, gall bladder and bile duct, that are regarded as likely contributing directly with the clinical signs reported on the submission form (chronic ataxia, lateral recumbency and anorexia). The conditions of hepatitis and

cholangitis (cholangiohepatitis), cholecystitis, cholelithiasis, biliary cystic disease and cirrhosis as well as vertebral lesions of degenerative joint disease, osteoarthritis and spondylosis affecting five vertebrae in the cervical and thoracic regions of the spinal column appeared to be irreversible changes. Whether these changes were reparable or irreparable would likely be controversial among veterinary surgeons, but it is my opinion that these were most likely irreparable. Furthermore, the presence of concurrent hepatitis, interstitial pneumonia and interstitial nephritis suggest that the animal was becoming septicemic (systemic bacterial infection), most likely secondary to lesions of cholangitis.

The neoplasm present on the tongue had multiple pleomorphic features of a mesenchymal malignant neoplasm, and specifically had features compatible with a rhabdomyosarcoma. Further characterization of the affected cell type can be investigated by a panel of immunohistochemical assays at a referral diagnostic laboratory. This can be arranged with authorization at another AAVLD accredited laboratory such as the Diagnostic Center for Population and Animal Health at Michigan State University, the Indiana Animal Disease Diagnostic Laboratory at Purdue University or the California Animal Health and Food Safety Laboratory at the University of California at Davis, California, all laboratories with which the Ohio ADDL has working relationships. Examination of several regional lymph nodes did not identify that metastasis had occurred at the time of the animal's death.

Other alterations noted including uroltihiasis, myocardial fibrosis, and unilateral periorchitis were likely mild and incidental findings at the time of the animal's death. Some or all of these conditions may (or may not) have had the potential of progressing and becoming clinically significant with time. Lastly, other changes noted (1 - atrioventricular endocardiosis, 2 - adrenal perivascular fibrosis, 3 - degenerative cystic changes and fibroadipose tissue replacement and lymphoid depletion in various lymph nodes) were likely changes of senescence (aging changes) that were of no clinical significance.

Addendum regarding hepatobiliary lesions:

A variety of different biliary lesions have been previously reported in the livers of adult lions, including peribiliary cysts, biliary cystadenomas, polycystic liver and kidney disease, and gallbladder adenocarcinomas (see 13 references cited below).

Peribiliary cysts and biliary cystadenomas have been reported occasionally in lions. Based on previous microscopic descriptions of these conditions, the intra-hepatic lesions in this case had tissue distribution features of biliary cystadenoma and histologic features of peribiliary cysts. As both conditions are those of benign tumors, this distinction may not be clinically significant, and the mere presence of cysts within the liver parenchyma were likely not clinical problems. In fact, it has usually been suggested that these lesions are congenital in origin, but their presentation has normally been noted in adult animals (aged13–22 years), and liver disease itself was not always present in previously reported cases. In one section of the present case, however, intra-hepatic inflammation appeared to be directly associated with inflammation of an intra-hepatic cystic lesion, which also contained abnormal multinucleated epithelial cells, suggesting the possibility of pre-malignant changes.

However, these cysts were not the only biliary tree-associated lesion observed in this case, and these other changes are considered to be potentially clinically significant. Concurrent extra-hepatic biliary anatomical abnormalities observed in this case in the main bile duct may have contributed to obstructive biliary disease (cholelithiasis) that was noted in the bile duct, as well as in the gall bladder and a large intra-hepatic cyst.

The combination of anatomical changes and choleliths and bladder stones may have predisposed this animal to the microscopic lesions of cholangiohepatitis observed (see reference 3, page 925). This ranged from chronic and active fibrinopurulent cholangitis, with intralesional coccoid-shaped bacteria in the thickened and apparently elongated and serpentine-like bile duct, to nonsuppurative cholangitis and hepatitis observed in at least one microscopic liver section. The additional presence of concurrent nonsuppurative interstitial pneumonia and interstitial nephritis may be considered as compatible with the development of septicemia. Bacteria that are usually isolated from cholangiohepatitis lesions include *Actinomyces, Bacteroides, Clostridia*, *E. coli,* and alpha-hemolytic *Streptococcus*.

1. Bosje, J. T., T. S. Van den Ingh, and J. S. van der Linde-Sipman. 1998. Polycystic kidney and liver disease in cats. 20: 136–139.
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13. Yu, C. H., K. T. Kim, D. N. Hwang, J. Y. Yhee, C. T. Moon, T. Y. Hur, and J. H. Sur. 2007. Peribiliary cysts associated with severe liver disease: a previously unrecognized tumor in a lion (Panthera leo). J. Vet. Diagn. Invest. 19: 709–712.

Lab Findings

All Testing done at ADDL unless otherwise indicated

Pathology

Specimen	Test Name
Lion - FELINE - Lion - Male Neutered - Adult	
Dead Animal - 1	Necropsy - Regulatory
	This service was provided by Jeff Hayes DVM MS

Tested by: Jeffrey Hayes, Test Date: 7/17/2015

Outsource

Specimen	Test Name	Result
Lion - FELINE - Lion - Male Neutered - Adult		
Brain - 3	Rabies Examination	Negative
	(Test Performed at ODH)	
	Brain tissue submitted to the Ohio Department of Health has been determined to be negative for rabies. Per telephone call from Karin Catrell of the ODH laboratory on 4/15/2015 4:01 pm - JRH	

Tested by: Jeffrey Hayes, Test Date: 4/15/2015

Pending Tests

No Pending Tests

Client Report History

Report Type	Delivery Method	Sent To	Date Sent
Final	Email	melissa.simmerman@agri.ohio.gov	7/17/2015 6:48 PM
Amended	Email	melissa.simmerman@agri.ohio.gov	7/31/2015 4:01 PM
Amended	Email	melissa.simmerman@agri.ohio.gov	7/31/2015 4:02 PM

Bulletin(s)

Preliminary and Interim reports are NOT official results. Results are official when denoted as Final Report.

ADDL results are available by email and by secure web portal access. Call 1-614-728-6220 to inquire about registering to receive web portal results. Access to the fillable ADDL SUBMISSION FORM can be found on the ADDL website:
http://www.agri.ohio.gov/divs/ai/addl/forms/anim-addlsample.pdf

THANK YOU FOR YOUR BUSINESS!

Attachment Table Of Contents

File Name	Attached To
B1508573.pdf	Accession
ADDL_B1508573_ODH_2015003125-Rabies Report.pdf	Accession
B1508573_ADDL.pdf	Accession

Ohio Department of Agriculture

ADDL
ANIMAL DISEASE DIAGNOSTIC LABORATORY

Animal Disease Diagnostic Laboratory
8995 East Main Street
Reynoldsburg, Ohio 43068
Phone: 614-728-6220
Fax: 614-728-6310

	DIVISION
FORM #:	ADDL/C
REVISION:	Decem
# PAGES:	Two (2
ACCOUNT #:	

Acc:B1508573
-A-HNS

ADDL SAMPLE SUBMISSION FORM

Date collected 4/13/15 **Date shipped** 4/13/15
Submitter's Name Melissa Simmerman
Clinic Name ODA
Address 8995 E main st
City Reynoldsburg State OH Zip 43068
Phone 614-728-6279 FAX
Vet License # 8833 ADDL Client #

Owner's Name ODA
Farm Name
Address
City State Zip
County
Phone FAX
Bill will be sent to the clinic.

☐ FAX results ☐ Diagnostic sample ☐ Export sample Country : ☐ Scrapie
Program sample: ☐ PRV ☐ Brucella ☐ NPIP ☐ Johne's ☐ EIA (EIA requests must be accompanied by form 0251) ☐ CWD

☐ Cattle ☐ Cat
☐ Horse ☐ Turkey
☐ Swine ☐ Chicken
☐ Sheep ☐ Psittacine
☐ Goat ☐ Ratite
☐ Dog ☒ Other: lion

Herd/Flock ID Grower House # Layer/Finisher #
Epidemiologic Info: # in herd/flock # in group # sick # dead
Date died 4/13/15 Euth? ☒ Yes ☐ No Abortion: trimester Age of Dam

History (clinical signs, nutrition, housing, vaccination, treatments, production level, related accessions, etc.):
Chronic ataxia
Lateral recumbency
Anorexia
Watery diarrhea
mass under tongue

(Continue on back if necessary)

Disease(s) or condition(s) suspected: ☐ Request antimicrobial susceptibility on bacterial pathogens.

Sample Data:

NO.	ANIMAL ID	BREED	SEX	AGE	QTY	SPECIMEN	TEST(S) REQUESTED
1	lion	lion	ml	adult	1	whole body	Regulatory panel

5/99

I certify that I have collected these samples and officially identified the animals indicated.

Melissa Simmerman
Signature of Licensed Veterinarian

☒ Accredited

History (continued on Page 2)

Ohio Department of Health
Bureau of Public Health Laboratories
8995 E. Main St, Bldg 22
Reynoldsburg, OH 43068
Phone: 1-888-634-5227
CLIA ID #:36D0655844

John R. Kasich/Governor
Richard Hodges, MPA Director of Health
Tammy L. Bannerman, Ph.D. D(ABMM)
Fax: (614) 387-1505
Email: odhlabs@odh.ohio.gov

Laboratory Report

Submitter ID: 0000090433
OHIO DEPARTMENT OF AGRICULTURE RABIES
8995 E MAIN STREET BLDG 6
REYNOLDSBURG, OH 43068

Phone:
County: LICKING

Location of Animal: OHIO ADDL 8995 E MAIN ST Reynoldsburg, OH 43068
Longitude: **Latitude:**

ODHL Specimen ID: 2015003125
Date Collected: 4/14/2015
Date Received: 4/15/2015

Species: Lion
Specimen:
- [x] Brain Stem
- [x] Cerebellum
- [ ] Hippocampus

Test	Result	Reference Range	Reported
Rabies*	Negative	Negative	4/16/2015

Comment:
■ 04/15/2015 KMC called spoke to Dr. Jeff Hayes with result #B1508573

RECEIVED
APR 17 2015
The Ohio Department of Agriculture
Division of Animal Health

FINAL REPORT

Methodology:
* Direct Fluorescent Antibody

Rabies_final.rpt

ODHL Specimen ID: 2015003125

4/16/2015 3:47:33PM

Ohio Department of Agriculture
ADDL
ANIMAL DISEASE DIAGNOSTIC LABORATORY

Animal Disease Diagnostic Labo
8995 East Main Street
Reynoldsburg, Ohio 43068
Phone: 614-728-6220
Fax: 614-728-6310

B1508573
V: Simmerman, M O: ODA/DWA
Feline A:Lion Age:Adult
MC 4/13/2015 03:39 PM CC: DN

Acc:B1508573
-A-HNS

ADDL SAMPLE SUBMISSION FORM

Date collected 4/13/15 Date shipped 4/13/15
Submitter's Name Melissa Simmerman
Clinic Name ODA
Address 8995 E main st
City Reynoldsburg State OH Zip 43068
Phone 614-728-6279 FAX
Vet License # 8833 ADDL Client #

Owner's Name ODA
Farm Name
Address
City State Zip
County
Phone FAX
Bill will be sent to the clinic.

☐ FAX results ☐ Diagnostic sample ☐ Export sample Country :
Program sample: ☐ PRV ☐ Brucella ☐ NPIP ☐ Johne's ☐ EIA (EIA requests must be accompanied by form 0251) ☐ CWD ☐ Scrapie

☐ Cattle ☐ Cat
☐ Horse ☐ Turkey
☐ Swine ☐ Chicken
☐ Sheep ☐ Psittacine
☐ Goat ☐ Ratite
☐ Dog ☒ Other: lion

Herd/Flock ID Grower House # Layer/Finisher #
Epidemiologic Info: # in herd/flock # in group # sick # dead
Date died 4/13/15 Euth? ☒Yes ☐No Abortion: trimester Age of Dam

History (clinical signs, nutrition, housing, vaccination, treatments, production level, related accessions, etc.):
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Lateral recumbency
Anorexia
Watery diarrhea
mass under tongue

(Continue on back if necessary)

Disease(s) or condition(s) suspected: ☐ Request antimicrobial susceptibility on bacterial pathogens.

Sample Data:

NO.	ANIMAL ID	BREED	SEX	AGE	QTY	SPECIMEN	TEST(S) REQUESTED
1	Lion	lion	ml	adult	1	whole body	Regulatory panel
	"neo" Accession # 15-1301-1		UMA 7/31/15				

5/99

I certify that I have collected these samples and officially identified the animals indicated.

Melissa Simmerman
Signature of Licensed Veterinarian

☒ Accredited

History (continued on Page 2)