VETERINARY DIAGNOSTIC LABORATORY

1800 DENISON AVENUE
MANHATTAN, KS 66506-5606
Phone #: 866-512-5650 Fax #: 785-532-4481

Owner: JOANN LIPS
503 BLUNT
CLAY CENTER, KS 67432

Accession Number:11-141203
Reference Number:081356
Case Coordinator: Gordon Andrews
Received:12/28/2011 **Finalized:** 01/10/2012
Sampled:

To: DR. THOMAS SCHERMERHORN
KSU VETERINARY TEACHING HOSPITAL
1800 DENISON AVE
MOSIER HALL
MANHATTAN, KS 66506

Phone # 785-532-4100
Fax # 785-532-4309

History: An 8-year-old, female intact white German Shepherd dog was submitted for necropsy on 12/28/11. The dog had history of hepatic disease which was diagnosed at KSU VMTH. The dog gotten progressively worse, anorexic, on and off for weeks. Owners elected euthanasia after multiple attempts of treatment at KSU and her relapsing.

Addended Report

ADMINISTRATION RESULTS

CREMATION

ANIMAL ID	KANDE
BREED	German Shepherd
AGE	8y
SEX	Female
COMMENTS	PICKED UP FOR CREMATION BY COMPANION ANIMAL PET CREMATORY

PATHOLOGY RESULTS

NECROPSY

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PATHOLOGY RESULTS

SPECIES	Dog
BREED	German Shepherd
SEX	Female
AGE	8y
ANIMAL ID	KANDE
SPECIMEN DESC	Dead Animal
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DIAGNOSIS

1. Liver: Chronic-active hepatitis characterized by bridging type (central to central, central to portal, and portal to portal) hepatocellular loss, lymphoplasmacytic and neutrophilic inflammation with mild fibrosis, single cell hepatocellular apoptosis/necrosis, micronodular regeneration, moderate periportal fibrosis, and cholestasis.
2. Small intestine: Enteritis, lymphoplasmacytic, diffuse with moderate mucosal fibrosis, lymphangectasia and crypt ectasia, and squamous metaplasia.
3. Large intestine: Colitis, lymphoplasmacytic, multifocal, mild with mild mucosal fibrosis, congestion, and crypt ectasia.

COMMENTS

The bridging pattern of hepatocellular necrosis, portal fibrosis, and nodular regeneration with lymphoplasmacytic inflammation is consistent with chronic-active hepatitis of dogs. The cause for the most cases of chronic hepatitis is unknown. Chronic insult to the hepatocytes by toxins, drugs, copper accumulation, infectious agents such as leptospira and adenovirus have been hypothesized to be the possible causes.

NECROPSY FINDINGS

Necropsy examination was performed on 12/28/11.
The dog was in poor body condition with a body condition score of 1/5. Externally the dog was thin with prominent ribs and sunken eyes. The skin, conjunctiva and oral mucosa were icteric.
The abdominal cavity contained 10 ml of amber colored clear fluid. The subcutaneous and abdominal fat were moderately icteric.
The liver was subjectively and moderately small and weighed 522 g. The liver was slightly yellow to bronze color with irregular rough surface and sharp edges. The cut surface of the liver revealed yellowish, friable parenchyma with numerous multifocal pale tiny white foci scattered throughout the parenchyma and a few roughly spherical well delineated expansile nodules ranging in size up to 1 cm diameter of similar color and texture to the surrounding parenchyma.
The mucosa of the small and large intestines were diffusely pale red and edematous. Most of the length of the intestinal lumina was devoid of food or fecal contents and contained brownish thick mucus.
The luminal surface of the aorta was markedly icteric.

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PATHOLOGY RESULTS

The skin on all the four limbs was removed and elbow, stifle, and hip joints were examined. There were no gross abnormalities in the joints.
There were no significant gross lesions in the thyroid glands, heart, lungs, stomach, spleen, pancreas, adrenal glands, kidneys, skeletal muscle, uterus, ovaries, urinary bladder, or brain.

HISTOPATHOLOGIC DESCRIPTION

Liver: Multiple sections from the different liver lobes are examined and all have similar microscopic lesions. Normal hepatic lobular architecture is altered by bands or tracts of parenchyma in which there is loss of hepatocytes leaving behind stroma and sinusoids which are dilated and congested. The tracts extend or bridge between central vein to central vein, sometimes from central vein to portal triads, and rarely from portal triad to portal triad. This sometimes results in a "reversal of lobular architecture" with a portal triad in the center of a lobule. Some of these nodules are expansile and represent regenerative nodular hyperplasia. There is mild reticulin type fibrosis in these tracts and infiltrates of very small numbers of lymphocytes, plasma cells and rare neutrophils. There are small to moderate numbers of hemosiderin laden macrophages and Kupffer cells. At the interface of these tracts and adjacent more normal parenchyma there are small numbers of individual necrotic or apoptotic hepatocytes, Copper stains show very small amounts of copper pigment in a few hepatocytes along this interface area. There is a mild degree of cannicular bile plugging and intrahepatocellular bile stasis. There is mild to moderate dense fibrosis around many portal triads. A few larger foci of nodular regenerative hyperplasia are also present corresponding to the hepatic nodules described grossly.

Small intestine: The lamina propria contain moderate to large numbers of lymphocytes and plasma cell mixed with few epitheliod macrophages which expands the villi. There is multifocal dilation of the lacteals which are surrounded by moderately increased fibroblasts and collagen fibers (fibrosis). Multifocally, crypts are distended with pale eosinophilic mucin to eosinophilic secretory material often mixed with necrotic cellular debri and the crypt epithelial cells are partially or completely replaced by squamous epithelial cells. The capillaries in the lamina propria are congested.
Large intestine: There are multifocal patchy aggregates of plasma cells in the lamina propria. Occasionally the crypts are distended with mucin which rarely contains small numbers of neutrophils.
Pancreas: The peripancreatic adipose tissue is multifocally infiltrated by foamy macrophages and neutrophils, with fat necrosis and mild fibrosis

Kidney: Multifocally, the tubular epithelial cells and tubular lumina contain aggregates of bile pigment (bile casts)
There are no significant microscopic lesions in thyroid glands, trachea, heart, lungs, stomach, spleen, lymph nodes, adrenal glands, kidneys, skeletal muscle, uterus, ovaries, urinary bladder, or brain.

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PATHOLOGY RESULTS

PATHOLOGIST	GORDON ANDREWS, DVM, PHD, DIPLOMATE ACVP
RESIDENT	MADHUSUDAN GORAVANAHALLY, DVM, PHD

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Client's Reference # 081356

Animal ID: KANDE

Species: DOG

Breed: GERMAN SHEPHERD

Multiple Specimens

Summary of Estimated Charges

The charges displayed may be split among two or more client accounts.

Description	Item #	Qty	Unit Price	Tax	Total
CREMATION	800-1095	1	$0.00	$0.00	$0.00
HISTOPATH NECROPSY 5+ TISSUES	805-10	1	$68.50	$0.00	$68.50
MASSON TRICHROME STAIN	805-1016	1	$0.00	$0.00	$0.00
PERIODIC ACID SCHIFF STAIN	805-1022	1	$0.00	$0.00	$0.00
PERLS PRUSSIAN BLUE STAIN	805-1023	1	$0.00	$0.00	$0.00
RUBEANIC ACID COPPER STAIN	805-1027	1	$0.00	$0.00	$0.00
NECROPSY	810-1005	1	$0.00	$0.00	$0.00
NECROPSY, LEVEL 6 VMTH CHARGE PER TEST	810-1035	1	$0.00	$0.00	$0.00
			Total:	**$0.00**	**$68.50**
				Grand Total:	**$68.50**

This is not an invoice; it is a summary of charges for this accession as of this date and time.

A monthly invoice will be sent the first week of the month from the Kansas State University Business Office. Monthly invoice will display all current charges and the balance due. For all charges that are not paid within 30 days of the monthly invoice, there will be a finance charge computed from the invoice date. Please direct any billing questions to the KSU Business Office, 785-532-3294.