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REPORT OF LABORATORY EXAMINATION

Client:
White Shepherd Genetics- (295483)
Project - Attn: Judy Huston
PO Box 404
Howell, MI 48844

Owner:
Anderson, Pam
12885 Pine Lake
Cedar Springs MI 49319

Rcvd Date:	5/18/2012 12:01:00 PM	Animal:	GYPSY	MRN:	
Admitted By:	Not, Provided	Species:	Canine	Breed:	German Shepherd
Ordered By:	N/A	Age:	11 years	Gender:	Female
Encounter:	01366766	Tag/Reg ID:			
CR#:	AP	Other ID:			

Necropsy Preliminary Report

Accession Number:	Received Date/Time:	Verified Date/Time:	Pathologist:
NC-12-0000507	5/18/2012 12:03:00 PM	5/19/2012 12:53:14 PM	Patterson, Jon S.

History

An 11-year-old, intact female white German shepherd was euthanized on 5/18/12. A brief history indicated that the dog became weak the night before. Abnormal laboratory findings on blood taken the day of euthanasia included elevated blood urea nitrogen (BUN), phosphorus, and total bilirubin concentrations; elevated alanine aminotransferase (ALT), alkaline phosphatase (ALP), gamma glutamyl transferase (GGT), amylase, and lipase activities; low sodium concentration; and severe panleukopenia (WBC=1,150/uL).
Previous medical history indicated bouts of hematuria in 2008 and 2009 (treated with antibiotics), and mammary tumor removal in 2010 and 2011. Biopsy diagnosis of the tumor removed in 2011 was cystic papillary carcinoma of mammary gland origin (noninvasive, noninfiltrative).

Gross Description

A 36-kg dog in good nutritonal condition and good post mortem condition was presented dead for necropsy. The skin was shaved in a rectangular, 10x3-cm area on the dorsal aspect of the left antebrachium, and in a rectangular, 4x3-cm area on the dorsal aspect of the right antebrachium. The abdominal cavity contained approximately 800 mL of dark red, unclotted and clotted blood.

The liver contained hundreds of spherical to irregularly shaped, 2 mm to 5 cm diameter, soft to firm, pink to dark red nodules, many of which caused elevation of the hepatic capsular surface. Nodules were present in all liver lobes, though over half were located in the cranial half of the liver, and more were present in the left lateral and medial lobes than in the right lobes. Overall, approximately 40% of the liver volume was consumed by neoplastic masses. Some nodules were dark gray red and friable to fluid in consistency, and 2 nodules with this consistency near the capsular surface had ragged surfaces, suggesting that these nodules had ruptured. Several of the lighter colored nodules on the capsular surface had umbilicated centers.

The spleen had an irregular capsular surface because of the presence of numerous coalescing, irregularly shaped, raised, dark red purple areas involving approximately 25% of the parenchyma. These rather nodular areas were somewhat softer

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than the consistency of the rest of the spleen, which was lighter purple in color. The irregular nodules ranged in diameter from 6 mm to 3 cm.

The capsular surfaces of the kidneys were somewhat mottled gray red and light brown, but the surfaces were smooth. The urinary bladder was contracted, with an external diameter of approximately 3.5 cm, and the wall was approximately 1.5 cm thick, firm, and tan. The bladder mucosa was grossly normal.

The gastrointestinal tract, pancreas, adrenal glands, ovaries, and uterus were grossly normal.

The mitral valve was mildly thickened and nodular, but the heart was otherwise normal. The lungs, thyroid gland, trachea, and esophagus were grossly normal.

The brain was grossly normal. The entire spinal cord was removed, and was grossly normal. Intervertebral discs at T5-T6, T6-T7, T7-T8, T8-T9, and the lumbosacral junction (L7-S) showed evidence of degeneration. The discs at T5-T6, T6-T7, and T8-T9 were opaque white to tan white, and somewhat dry and flaky. Minimal disc material was present at T7-T8, and what was present was opaque white and flaky. The L7-S disc material was opaque white and fibrous, and there was slight (1 mm) protrusion of disc material into the vertebral canal at this site. Bridging spondylosis was mild (1-2 mm) at T5-T6, T6-T7, L5-L6, and L6-L7. Bridging was moderate (4 mm) at L7-S.

Gross Diagnosis(es)

liver: disseminated cancer, believed to be of hepatic origin
abdomen: severe hemoabdomen
vertebral column: multifocal intervertebral disc degeneration (T5-T6, T6-T7, T7-T8, T8-T9, L7-S); bridging spondylosis (T5-T6, T6-T7, L5-L6, L6-L7, L7-S)

Comment:
Rupture of one or more cancerous nodules in the liver probably led to hemoabdomen, and the dog probably became acutely weak because of blood loss. Histopathologic examination of all tissues, including complete assessment of spinal cord, is in progress.

Jon S. Patterson, DVM, PhD, Dipl ACVP
Anatomic Pathologist

Jon S. Patterson, DVM, PhD, DACVP

(Electronically signed by) JSP

Verified: 05.19.2012 12:53

JSP /JSP

Necropsy Final Report

Accession Number:	Received Date/Time:	Verified Date/Time:	Pathologist:
NC-12-0000507	5/18/2012 12:03:00 PM	5/29/2012 5:08:14 PM	Patterson, Jon S.

History

Please see previous report.

Gross Description

Please see previous report.

Gross Diagnosis(es)

Please see previous report.

Laboratory Findings

NA

Microscopic Description

Sections of liver, spleen, heart, kidney, lung, adrenal gland, small intestine, pancreas, urinary bladder, uterus, and lymph node, 8 sections of brain, and 20 sections of spinal cord (from cervical through sacral segments, plus cauda equina) were examined.

The liver contained numerous small to large, round to irregularly shaped nodules of neoplastic epithelial cells arranged in solid lobules or nests, cords, and subtle acinar or ductular structures. The neoplastic cells were markedly pleomorphic, but were generally cuboidal to polygonal, with large, round, hypochromatic to vesicular nuclei and a moderate to large quantity of pale eosinophilic to pale basophilic cytoplasm. Nuclei contained 1 to 3 prominent nucleoli, and there were 5-10 mitotic figures (including several atypical ones) per 10 HPF. Neoplastic nodules contained small to large areas of necrosis, and some contained small to large lakes of erythrocytes, with or without fibrin. Some nodules were completely necrotic and surrounded by a zone of degenerating neutrophils. Thin bands of fibrovascular tissue dissected through the larger neoplastic nodules, and some nodules were partially surrounded by a fibrous capsule. Many entrapped hepatocytes, or hepatocytes between neoplastic nodules contained yellow brown, fine granular, intracytoplasmic pigment (hemosiderin, bile pigments, or lipofuscin). Small numbers of remaining hepatocytes contained discrete, intracytoplasmic vacuoles.

One section of lymph node was examined, and in it, the architecture was completely effaced because of infiltration by neoplastic cells similar to those described in the liver. The neoplastic cells were arranged primarily in solid sheets, but in some areas formed acini and cords as described above.

In sections of brain, there was mild diffuse neuronal lipofuscinosis. Moderate numbers of neurons were surrounded by 2-3 glial cells (satellitosis). In the cerebrum, there was mild multifocal leptomeningeal fibrosis.

There was mild multfocal lipofuscinosis in spinal motor neurons. The white matter of the spinal cord was essentially normal diffusely, except for minimal, isolated changes in a few sections. One ventral funiculus at the level of T3-T4 contained a swollen axon (spheroid) and one distended myelin sheath with an associated foamy macrophage. Single clusters of ellipsoids were present in the white matter of longitudinal sections of L1 and L2 segments. A longitudinal segment of one cauda equina nerve root was essentially devoid of myelinated axons, with replacement by proliferating Schwann cells, numerous foamy macrophages, macrophages with gray brown, granular pigment, and foci of cholesterol clefts. Similar changes were noted in portions of 3 other nerve roots, in cross sections.

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The spleen was irregularly congested, with areas where erythrocytes obscured the architecture of both the red pulp and white pulp, and other areas where the parenchyma was devoid of erythrocytes, with increased density of fibromuscular trabeculae due to contraction. Aggregates of hemosiderin-laden macrophages were scattered throughout the spleen, especially near fibromuscular trabeculae.
Renal corpuscles were slightly enlarged, as the glomerular tufts filled Bowman's spaces. The enlargement appeared to be due to increased mesangial matrix in the glomerulus.
Occasional hypertrophic myocytes were present in the myocardium, and scattered myocytes contained noticeable lipofuscin pigment.
In a section of lung, there was mild anthracosis.
The zona reticularis of the adrenal cortex contained multiple small aggregates of large cells with pale eosinophilic, granular to finely vacuolated cytoplasm. In addition, a few irregular nodules of cortical cells were noted at the corticomedullary junction.
The serosa of the urinary bladder was moderately thickened by dense granulation tissue, with foci of hemorrhage. In a few areas, lining mesothelial cells were hypertrophic and hyperplastic.
The pancreas contained a nodule of hyperplastic acinar epithelium.
Small to large numbers of myofibers within lumbar epaxial muscle bundles were smaller than normal, with hypereosinophilic sarcoplasm, suggesting atrophy. There also was multifocal replacement of myofibers with adipocytes.

Morphologic Diagnosis(es)

liver: hepatocellular carcinoma, with intrahepatic and regional lymph node metastases
cauda equina: multifocal nerve fiber atrophy, loss, and Schwann cell proliferation
brain, spinal cord: mild neuronal lipofuscinosis
spleen: multifocal congestion and hemosiderosis
kidneys: mild diffuse membranous glomerulopathy
adrenal gland: multinodular adrenocortical hyperplasia
lumbar epaxial muscles: multifocal myofiber atrophy and loss, with adipose replacement

Final Diagnosis(es)

liver: hepatocellular carcinoma, with focal or multifocal rupture of a neoplastic mass, leading to hemoabdomen

Comment:
As explained in the preliminary gross necropsy report, rupture of one or more of the cancerous nodules in the liver apparently led to blood loss into the abdomen and caused this dog to become acutely weak. Histopathology confirmed that the cancer was of liver (hepatocellular) origin.

There was no histopathologic evidence of degenerative myelopathy. The major finding upon examination of nervous tissues was in the cauda equina--i.e., the nerve roots which extend past the end of the spinal cord. It is possible that the described changes resulted from nerve root compression near the lumbosacral junction. It is difficult to say with certainty, however, whether this dog had lumbosacral syndrome (lumbosacral stenosis) since accurate diagnosis of this condition requires high-quality radiographs ante mortem, or close inspection of the lumbosacral spine post mortem after all muscle tissue has been removed by means beyond simple dissection. I will say that in most cases of lumbosacral syndrome, there is severe bridging spondylosis at the L7-sacral joint, and bridging was not severe in this case.

Essentially all of the other lesions described above were incidental (i.e., not clinically significant) and age-related.

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Anatomic Pathologist

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(Electronically signed by) JSP

Verified: 05.29.2012 17:08

JSP /JSP

Special Requests

Collected Date/Time (If Provided)	05/18/2012 12:11:00
Procedure	
Notification *	"See Below"

5/18/2012 12:11:00 PM Notification:
This report informs you of laboratory results associated with an Anatomic Pathology case. Laboratory results should be interpreted in conjunction with pathologic findings. In some instances, laboratory results may be received prior to the pathology report. In all instances, a cumulative report will be issued.