Clinical Pathology Review: Hepatic disease in a dog with Cholestasis and Normal Serum Enzyme Activity

By A. E. Ledet, DVM, PhD*

Serum glutamic pyruvic transaminase (SGPT) and serum alkaline phosphatase (SAP) activities are widely used to identify hepatic disease in the dog. To fully assess the type and degree of hepatic disease one must use a combination of liver function tests, biochemical tests, and liver biopsy.

The case presented here is unusual in that SGPT and SAP activity did not reflect the degenerative processes taking place in the liver.

A 2-year old spayed female dog was admitted to the Iowa State University Veterinary Hospital on August 13, 1980. The dog had a history of being sick for the past two weeks. Treatment by the referring veterinarian resulted in a brief positive response followed by a serious relapse. Presenting signs included icterus, ascites, weight loss, and severe lethargy.

LABORATORY DATA

Initial urinalysis—dark yellow color, clear, specific gravity 1.007, large amount of blood, strongly positive congjugated bilirubin, 1 plus proteinuria, pH 7, and a few granular casts.

Hematology (normals)	8-14	8-18
Hb (12-18 gm)	15.8	8.7
PCV (37-57%)	46	27
WBC (6-17 × 10^3)	13,400	16,300
Segs	11,256	14,996
Lymphs	1,206	1,304
Mono	670	...
Eosin	268	...
Plasma Protein (6.0-7.7 gm)	3.9	2.8
Fibrinogen (100-400 mg)	100	100
Reticulocytes (less than 1%)	ND	4.8

Blood Chemistries (normals)	8-14	8-18	8-19
BUN (10-30 mg/dl)	14	69	84
Glucose (60-115 mg/dl)	105	105	ND
Phosphorus (2.5-5.0 mg/dl)	5.4	9.1	ND
Calcium (9.4-12.2 mg/dl)	8.4	7.7	ND
Total Bilirubin (less than 1 mg/dl)	5.6	8.6	ND
Direct Bilirubin (less than 0.1 mg/dl)	4.5	6.6	ND
Albumin (3.0-4.3 gm/dl)	2.5	1.8	ND
SGPT (4-65 I.U.)	72	57	ND
SAP (10-80 I.U.)	97	60	ND
Sodium (141-155 mEq/L)	127	114	125
Potassium (3.7-5.8 mEq/L)	5.0	5.2	3.4

PROBLEMS IDENTIFIED BY LABORATORY DATA

1. Anemia—regenerative, most typical of blood loss. The rapidly developing anemia and concurrent fall in plasma protein suggest rapid blood loss. A reticulocyte count of 4.8 corrected for PCV to 2.9 indicates that the bone marrow has had time to respond to the anemia, therefore, the blood loss had begun at least four days earlier.
2. Hypoproteinemia characterized by loss of albumin and globulins. Most likely caused by several factors such as blood loss, urinary loss, inadequate dietary protein and possibly hepatic insufficiency.
3. Azotemia—pre-renal causes such as dehydration, hypovolemic shock and digestion of whole blood lost into gastrointestinal tract. Random urinalysis upon admission did not answer questions of renal involvement.
4. Hyperphosphatemia—decreased total GFR, pre-renal causes.
5. Hypocalcemia—lack of absorption from gut and related partially to hypoalbuminemia.

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6. Obstructive icterus—hyperbilirubinemia with 80% direct (conjugated) reading bilirubin caused by cholestasis.
7. Insignificant rise in SGPT and SAP. This is an unusual finding in an apparent cholestatic disease.
8. Hyponatremia—low total body sodium and lack of adequate dietary intake. Hyponatremia with normal potassium suggests causes other than adrenal insufficiency.

Paracentesis Abdominis August 14

Yellow, cloudy fluid with a pH of 7, less than 100 mg/dl protein, and low cell count. Interpretation—Transudate.

Liver Biopsy August 18

Diagnosis—Chronic Cholangiohepatitis.

Necropsy following euthanasia August 19

Diagnosis—Diffuse chronic active cholangiohepatitis; gastric and duodenal ulcers with intestinal hemorrhage.

Discussion

According to Strombeck, in most cases of chronic active hepatitis SGPT and SAP activities are elevated. However, the increase may not be marked.

Biopsy and necropsy findings in this case corroborated the clinical diagnosis of hepatitis and the laboratory diagnosis of obstructive icterus, blood loss anemia and pre-renal azotemia.

The unusual aspects of this case are the normal or near normal SGPT and SAP levels with a greatly increased serum bilirubin, the young age (2 years old) and relatively short duration (2 weeks) of clinical illness.

REFERENCE

Strombeck, DR: *Small animal gastroenterology*, Stonegate Publishing, Davis, California. 1979.

Brucella canis in Dogs

by Nancy Creek*

Brucella canis is a gram negative coccobacillus that was first isolated in 1966. It was first recognized in large breeding colonies of beagles, but subsequent independent investigators found many other breeds and mixed-breed dogs to be infected with the organism.[3]

Characteristics of the new *Brucella* species were determined by the usual biochemical tests and by analysis by gas chromatography of the metabolites excreted during growth. Cultural studies, agglutination tests, and pathogenicity tests were also done. The new *Brucella* species was found to be similar to *B. suis* morphologically and biochemically but similar to *B. ovis* antigenically.[4]

B. canis grows well in tryptose broth at 37° C but even better on Brucella broth (Albimi). Initially it is a uniform growth; after 48 to 72 hours of incubation a viscous, ropey sediment occurs. No pellicle is formed. Growth is inhibited by ten percent CO_2 and no growth occurs under strict anaerobic conditions.[3,13] Table I lists the biochemical characteristics of *B. canis*.[4,8]

TABLE I: Biochemical Characterisics of *Brucella canis*

Hemolysis	–	Dextrose	–
Motility	–	MacConkey	–
Citrate	–	MR-VP	–
Urease	+	Catalase	+
H_2S	+slow	Litmus Milk	alk
Indol	–	Gelatin	–
Gas	–	Oxidase	–
Lactose	–	Nitrate	+

The surface antigens of *B. canis* are very similar to *B. ovis*. It also cross-reacts significantly with other bacteria such as *B. suis, Actinobacillus equuli,* and a *Moraxella*-like bacterium. *B. canis* antisera react very slightly with *Bordetella bronchiseptica, B. abortus,* and *B. melitensis.*

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