Problem-Oriented Approach in Interpreting Clinical Pathology Laboratory Data

by E. D. Lassen, D.V.M., Ph.D.*
A. E. Ledet, D.V.M., Ph.D.†

We have found that a problem-oriented approach is a valuable method of evaluating laboratory data. Such an approach minimizes the amount of significant data which is overlooked and maximizes the amount of useful information which can be derived from such data. This method also lends itself to incorporation into the problem-oriented record-keeping system.

The following cases are examples of the evaluation of laboratory data by the problem-oriented approach. This data is derived from cases treated at the Veterinary Teaching Hospital at Iowa State University. The tests performed were those requested by the clinician in charge of the case. Where no results are given, it may be assumed that the test was not performed on that day.

It must be recognized that acceptable diagnostic procedures require a detailed history and a thorough physical examination. In addition, clinical laboratory tests, radiological studies, and microbiological procedures are often required. This article, however, emphasizes the use of the clinical laboratory as an aid in arriving at a diagnosis. For this reason, minimum information derived from other procedures is included.

Although interpretation of laboratory data is often speculative, the authors feel that explanations included with the following cases are those which are most likely in light of all of the information available. The main reference for the interpretation of the data in these cases is *Veterinary Laboratory Medicine—Clinical Pathology* by J. R. Duncan and K. W. Prasse.[1]

Case 1

Canine, German Shepherd, Male, 4 years old

*Dr. Lassen is an assistant professor in the Department of Veterinary Pathology, College of Veterinary Medicine, ISU.

†Dr. Ledet is a professor in the Department of Veterinary Pathology, College of Veterinary Medicine, ISU.

History and Physical Findings

Day 1—This farm dog was found in lateral recumbency with rigid hyperextention of the legs. No response to toe-pinch or loud noise could be elicited. Dyspnea was evident. Body temperature = 106.6°F (41.4°C).

Day 2—The dog could rise and walk but was depressed.

Day 3—The dog appeared to be normal and was sent home.

Day 4—The dog was returned to the hospital with vomiting, diarrhea, and ataxia. The animal was severely dehydrated. Body temperature = 100.6°F (38.1°C). Treatment—antibiotics, corticosteroids, and intravenous (IV) lactated Ringer's solution.

Day 5—Complete ataxia was evident. Radiographs were uninformative. Intravenous fluids were continued.

Day 6—The dog died.

Problems Identified by Laboratory Data (See p. 70)

1. Dehydration with relative polycythemia.
   The mild azotemia on days 1 and 2 is most likely pre-renal in origin.
2. Polychromasia and nucleated erythrocytes.
   The cause of this response was not determined. The dog was not anemic and the regenerative response had disappeared by day 5.
3. Systemic stress —increased levels of endogenous cortisol.
   This is most evident on day 1 when an absolute neutrophilia with a slight left shift and absolute lymphopenia are present. On subsequent days, there is an absolute neutrophilia and a relative lymphopenia but no left shift. Systemic stress is the best explanation for these findings in the dog. Monocytosis on day 5 is also compatible with systemic stress.

Table 1

Laboratory Data	Day 1	Day 2	Day 5
Hemoglobin g/dl (12-18)	21	22	20
Hematocrit % (37-55)	63	62	59
Total Leukocyte Count (6,000-17,000)	19,100	23,100	29,400
Band Neutrophils (0-300)	382	0	0
Segmented Neutrophils (3,000-11,400)	17,763	21,714	24,696
Lymphocytes (1,000-4,800)	764	1,386	2,940
Monocytes (150-1,350)	0	0	1,764
Eosinophils (100-750)	191	0	0
Erythrocyte Morphology	Polychromasia Anisocytosis	Polychromasia Anisocytosis	Normal
	2 nucleated RBC's / 100 WBC's		
Platelets	Adequate	Adequate	Adequate
Plasma protein g/dl (6.0-7.5)	6.8	6.8	6.8
Fibrinogen mg/dl (100-300)	100	500	800
Blood urea nitrogen mg/dl (5-28)	29	32	268
S.G.P.T. I.U. (4-66)	59	366	525
Sodium mEq/l (141-155)		156	143
Potassium mEq/l (3.6-5.6)		4.1	8.6
Blood pH* (7.31-7.42)		7.34	
Blood pCO_2* mmHg (29-42)		26.6	
Blood bicarbonate* mEq/l (17-24)		14.0	
Urinalysis:			
Color			Brown
Transparency			Cloudy
Specific gravity			1.020
Urobilinogen			1.0
Occult blood			1+
Conjugated bilirubin			Negative
Ketones			Negative
Glucose			1+
Protein			1+
pH			6.0
Leukocytes			Large numbers
Erythrocytes			Large numbers
Casts			Large numbers—graunlar

*Arterial sample

Case 1 * * * * * *

4. Hyperfibrinogenemia.

   The plasma protein:fibrinogen ratio is significantly decreased on days 2 and 3. This indicates a significant absolute rise in fibrinogen. Inflammation and/or renal disease could cause such a rise. Renal disease is the best explanation in this case.

5. Primary renal disease.

   Severe azotemia on day 5 coupled with the inability of the kidneys to adequately concentrate urine (specific gravity should be above 1.025 in a dehydrated dog) point to primary renal disease. The large number of granular casts present in the urine are a localizing sign of renal disease. Glucosuria indicates a lack of tubular reabsorption which may be due to tubular injury.

6. Urogenital tract inflammation.

   Large numbers of erythrocytes (hematuria) and leukocytes (pyuria) in the urine suggest inflammation. The positive occult blood and proteinuria are also compatible with inflammatory disease. These findings do not localize the inflammation in any particular area of the urogenital tract.

7. Hepatocellular injury.

   The high S.G.P.T. (a liver specific, leakage-type enzyme in the dog) indicates hepatocellular injury but does not reveal the cause or extent of this injury.

8. Hyperkalemia.

   The severe hyperkalemia on day 5 is probably associated with a metabolic acidosis and severe renal tubular disease.

During metabolic acidosis, hydrogen ions enter and potassium ions leave cells, resulting in hyperkalemia. Unfortunately, blood gas analysis was not performed on day 5, and these theories cannot be confirmed.

9. Compensated metabolic acidosis.

   This is evident on day 2. Although bicarbonate is low, pCO_2 has also fallen, and the dog's blood pH has remained normal. This is probably an early stage of a metabolic acidosis which will become more severe as the dog's renal disease progresses.

Summary

This dog's primary problem appears to be a rapidly developing renal tubular disease accompanied by dehydration, systemic stress, and metabolic acidosis. Hepatocellular injury and urogenital tract inflammation are also evident, but their association with the primary etiology is not clear.

Final Diagnosis and Comments

The post mortem diagnosis on this dog was acute nephrosis probably due to ethylene glycol ingestion.

Ethylene glycol toxicosis may occur as 2 syndromes. The acute syndrome is characterized by depression, incoordination, ataxia, vomiting, and, possibly, coma and death. Animals with this syndrome are severely acidotic. A more chronic syndrome is seen in animals surviving for more than 24 hours after ingestion of the toxicant. This syndrome is characterized by vomiting, ataxia, and paresis, as well as a severe, progressive uremia.[2]

The dog in this case was admitted to the hospital in the acute phase and appeared to have recovered only to eventually die from severe renal tubular disease.

Case 2

Bovine, Limousin, Female, 3 years old

History and Physical Findings

This cow suddenly became anorexic and appeared to have severe abdominal pain. Rumen contractions were weak. No other significant abnormalities were found on physical examination. Body temperature = 102.0°F (38.9°C). A small amount of bloody feces was passed while the cow was in the clinic.

Problems Identified by Laboratory Data (See p. 72)

1. Dehydration.

   High normal to slightly elevated hemoglobin, hematocrit, and plasma protein concentrations on both days are most likely results of dehydration. Mild azotemia (B.U.N. = 63) with evidence of renal concentration of urine (specific gravity = 1.024; urine osmolality = >2 times plasma osmolality) is also compatible with dehydration.

2. Inflammatory leukocyte response.

   Although total leukocyte and segmented neutrophil counts are normal, absolute increases in band neutrophil counts on both days indicate a tissue demand for neutrophils. Borderline lymphopenia on day 2 suggests systemic stress.

3. Hyperfibrinogenemia.

   Plasma protein:fibrinogen ratios are decreased on both days, indicating an absolute hyperfibrinogenemia. The hyperfibrinogenemia in this case is probably due to inflammation. It should be noted that the hyperfibrinogenemia is contributing to the hyperproteinemia.

4. Glucosuria.

   This could be due to renal tubular damage with insufficient glucose reabsorption or due to blood glucose concentrations exceeding the renal threshhold (usually as a result of IV glucose therapy in the bovine). The cause is not obvious in this case.

5. Hyperphosphatemia.

   This is probably associated with a reduced glomerular filtration rate (GFR) due to dehydration. Since phosphates are excreted passively via the glomerulus, a decrease in GFR can result in hyperphosphatemia.

6. Hypocalcemia.

   There may be many causes contributing to hypocalcemia in this case. Anorexia might be a major cause. Also, hyperphosphatemia tends to induce a com-

Table 2

Laboratory Data	Day 1	Day 2
Hemoglobin g/dl (8-15)	14.5	15.5
Hematocrit % (24-46)	42	45
Total Leukocyte		
Count (4,000-12,000)	7,200	5,400
Band Neutrophils (0-120)	360	324
Segmented Neutrophils (600-4,000)	2,808	2,430
Lymphocytes (2,500-7,500)	3,672	2,646
Monocytes (25-850)	144	0
Eosinophils (200-2,400)	216	0
Erythrocyte Morphology	Normal	Normal
Platelets	Adequate	Adequate
Plasma protein g/dl (6.0-8.0)	7.9	8.2
Fibrinogen mg/dl (100-500)	800	900
Blood urea nitrogen mg/dl (10-20)	63	
S.G.O.T. I.U. (0-150)	109	
Alkaline phosphatase I.U. (10-80)	30	
Sodium mEq/l (132-152)	134	
Potassium mEq/l (3.9-5.8)	2.9	
Calcium mg/dl (9.4-12.2)	8.7	
Phosphorus mg/dl (2.3-7.4)	10.4	
Magnesium mg/dl (1.2-3.5)	2.7	
Chloride mEq/l (97-111)	72	
Blood pH* (7.35-7.50)	7.58	7.54
Blood pCO_2* mmHg (35-44)	55.7	50.0
Blood bicarbonate* mEq/l (20-30)	52.2	42.7
Urinalysis:		
Color		Yellow
Transparency		Cloudy
Specific Gravity		1.024
Urobilinogen		0
Occult blood		Negative
Conjugated bilirubin		Negative
Ketones		Negative
Glucose		1+
Protein		Negative
pH		5.0
Leukocytes		Normal
Erythrocytes		Normal
Casts		Negative
Plasma Osmolality mOsm/Kg (270-300)		306
Urine Osmolality mOsm/Kg		654

*Arterial sample

Case 2 * * * * * *

pensatory hypocalcemia.

7. Metabolic alkalosis with partial compensation, hypochloremia, and hypokalemia.

This alteration is most commonly associated with obstructive gastrointestinal (G-I) disease in ruminants. Hydrochloric acid (HCl) is secreted into the abomasum by the following reaction:

$$H_2CO_3 + NaCl \rightleftharpoons HCl + NaHCO_3$$

Carbonic Acid — Sodium chloride — Sodium bicarbonate

As hydrochloric acid enters the abomasum, sodium bicarbonate is returned to the blood. Under normal circumstances, the hydrochloric acid passes into the small intestine and is reabsorbed. If outflow from the abomasum is blocked or if high intestinal obstruction is present, such absorption cannot occur. This results in a net gain of bicarbonate and a net loss of chloride from the extracellular fluid. Metabolic alkalosis and hypochloremia (which are present here), therefore, result. In this case, pCO_2 has risen to partially compensate for the metabolic alkalosis.

In response to the alkalosis, hydrogen ions (H^+) leave cells in an attempt to neutralize bicarbonate ions (HCO_3^-). At the same time, potassium (K^+) ions enter cells in order to balance charges. A

hypokalemia results.

8. Paradoxic aciduria.

   Normally, alkalotic animals are expected to have alkaline urine, but ruminants with G-I obstruction often have acid urine.

   In a state of dehydration, there is a renal retention of sodium ions (Na^+) in an attempt to maintain extracellular fluid volume. In order to absorb sodium ions, the kidney must concurrently absorb chloride ions (Cl^-) or excrete potassium ions (K^+) or hydrogen ions (H^+). With the acid-base and electrolyte imbalances caused by G-I obstruction in the ruminant, however, the animal is already hypochloremic and hypokalemic. Ruminants will maintain potassium concentrations at the expense of hydrogen concentrations; therefore, the only alternative left to the kidney is excretion of hydrogen ions in exchange for absorption of sodium ions. An acid urine results.

9. Hyperosmolality.

   This may be related to increased phosphates, urea, bicarbonate, and sulfates in the plasma, but hypokalemia and hypochloremia have partially counteracted the hyperosmolar effects of these substances.

Summary

The metabolic alkalosis and hypochloremia point toward a high G-I obstruction (abomasal or high intestinal). Dehydration, inflammation, and other electrolyte imbalances are probably related to such obstruction.

Final Diagnosis

Post mortem examination revealed an intussusception at about the middle of the jejunum. The intestine cranial to the lesions was necrotic. The kidneys appeared to be normal.

BIBLIOGRAPHY

1. Duncan, J. R. and Prasse, K. W. *Veterinary Laboratory Medicine—Clinical Pathology.* Iowa State University Press, Ames, Iowa. 1977.
2. Oehme, F. W. Antifreeze (Ethylene Glycol) Poisoning. In *Current Veterinary Therapy VI.* Edited by R. W. Kirk. W. B. Saunders Company, Philadelphia, Pennsylvania. p. 135-137, 1977.

THE LAMENT OF THE INSEMINATED COW

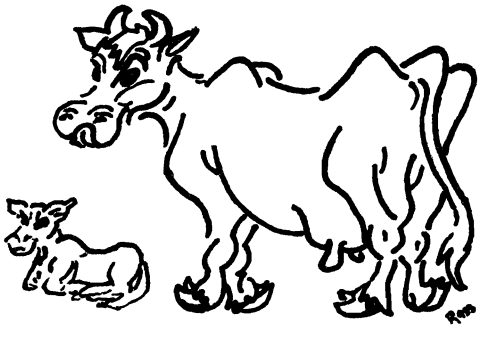
I have just given birth to a calf, sir,
And with motherly pride I am full.
But please don't chaff and please don't laugh
When I tell you I've not seen a bull.

The joy has gone out of the meadow
And the summer is no longer gay
Since the one bit of fun in the years dreary run
Has by science been taken away.

No bull has embraced me with passion
I've not had the ghost of a binge
But only been rubbed and ruthlessly shoved
By a vet with a cold glass syringe.

Oh! please listen to my lament, sir,
Though the're some things a cow shouldn't say
Those vets, though scientific, are fairly prolific
And still do it the old-fashioned way.

Author Unknown
contributed by Dr. Mike Shires



drawn by Dave Rassmussen

CORRECTION

In the preceeding issue (*Iowa State Veterinarian,* Vol. 40, Issue I, 1978) in our article "Gastric Dilatation and Volvulus in the Dog" (p. 16-24) we used a non-existent word "vomition". More correct terms are eructation or vomiting. We regret the error and thank Dr. H. N. Engel for calling it to our attention and furthering our education.

G. F. Grauer
D. W. DeYoung