Serum Lipase Activity in Young Dogs With Acute Enteritis or Gastroenteritis

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Blood serum lipase activity was determined in 48 young dogs with acute enteritis or gastroenteritis due to canine parvovirus (16 cases) and presumably to other infectious agents (32 cases). Elevated serum lipase activity (> 500 U/L) was found in 13 dogs (27.1%) with values ranging from 800 to 2,780 U/L. The hyperlipasemia of these cases may be attributed to acute pancreatitis secondary to acute gastroenteritis or to gastrointestinal upset.

Key Words: Young dogs, hyperlipasemia, acute enteritis, gastroenteritis

Introduction

In the dog, the main source of serum lipase activity is the pancreas, from which the enzyme is secreted in an active form. Lipase exerts its maximal activity in the alkaline environment of the duodenum, hydrolyzing triglycerides to fatty acids.

Hyperlipasemia is a common finding in canine acute or subacute pancreatitis, both clinical and experimental. The reliability of serum lipase analysis in the diagnosis of canine acute pancreatitis approaches 72%. Increased lipase activity, on the other hand, may be associated with nonpancreatic diseases such as gastroenteritis, renal failure, acute liver failure, malignancies, or with drug administration such as corticosteroids.

Hyperlipasemia in young dogs has been reported before. In the present work, an attempt has been made to find out the possible causes of hyperlipasemia in acute enteritis or gastroenteritis in young dogs.

Materials and Methods

A total of 48 young dogs, presenting a clinical picture compatible with acute enteritis or gastroenteritis, were studied. All dogs were admitted to the Clinic of Medicine, Veterinary Faculty, Aristotle University of Thessaloniki, Greece, between October 1990 and December 1992. The mean age of the dogs was 4.7 months, with a range of 1.5 to 10 months and a male to female ratio of 0.92 (23 males, 25 females). Thirty-seven were purebred dogs and the rest were mixed breeds. The dogs were unvaccinated or incompletely vaccinated against distemper, infectious hepatitis, leptospirosis, and canine parvovirus.

On admittance, a physical examination followed by hematology, biochemical screening, urinalysis, fecal examination for parasites, protozoa, and parvovirus were carried out in every case, before any kind of treatment was instituted.

The Wintrobe method was used for measuring hematocrit. White blood cell and platelet count was determined manually with a Neubauer hematocytometer (Unopette: Becton-Dickinson, Franklin Lakes, NJ). Serum alanine aminotransferase (ALT) and lipase activities, as well as serum urea nitrogen (SUN) concentration were measured using commercial kits (Boehringer Ingelheim, Indianapolis, IN). For lipase activity determination, in particular, the method used was turbidimetric, based on triolein degradation. Serum sodium (Na) and potassium (K) concentrations were measured by flame photometry. A direct fecal examination and fresh saline preparation were used for the detection of parasitic ova and intestinal protozoa, respectively. An ELISA test (CITE test: IDDEX Laboratories, Inc., Westbrook, ME) was also used for detecting parvoviral antigen in stool samples taken directly from the rectum. A dipstick method (Combur 8: Boehringer Mannheim) and the Heller test were used for qualitative urinalysis. The urine specific gravity and plasma total solids (TS) were measured with...
TABLE 1
Signalment and Clinical Findings in 13 Hyperlipasemic Dogs With Parvoviral Enteritis or With Acute Enteritis-Gastroenteritis Due to Other Causes

| Case Number | Breed         | Sex | Age (months) | Vomiting | Diarrhea | Abdominal Pain | Fever |
|-------------|---------------|-----|--------------|----------|----------|----------------|-------|
| 1           | German Shepherd | f   | 4            | +        | +        | -              | +     |
| 2           | German Shepherd | f   | 4            | +        | +        | -              | +     |
| 3           | German Shepherd | f   | 3            | +        | +        | -              | +     |
| 4           | mongrel        | m   | 1.5          | +        | +        | -              | -     |
| 5           | German Shepherd | f   | 4            | +        | +        | -              | +     |
| 6           | mongrel        | f   | 5            | +        | +        | +              | +     |
| 7           | GSH Pointer    | m   | 2.5          | +        | +        | -              | -     |
| 8           | GSH Pointer    | f   | 8            | +        | +        | -              | +     |
| 9           | mongrel        | f   | 5            | +        | +        | +              | -     |
| 10          | Greek Hunting  | m   | 6            | +        | +        | -              | +     |
| 11          | mongrel        | m   | 8            | +        | +        | +              | +     |
| 12          | German Shepherd | m   | 3            | +        | +        | -              | -     |
| 13          | German Shepherd | f   | 2            | +        | +        | -              | +     |

a Hemorrhagic

TABLE 2
Hematological and Biochemical Findings in 13 Hyperlipasemic Dogs With Parvoviral Enteritis or With Acute Enteritis-Gastroenteritis Due to Other Causes

| Case Number | PCV % | Platelets μl | WBC μl | SUN mg/100 mL | ALT U/L | Lip U/L | K mEq/L | Na mEq/L | TS (Plasma) | Parvovirus Test (Feces) |
|-------------|-------|--------------|--------|---------------|---------|---------|---------|----------|-------------|------------------------|
| 1           | 41    | 325,000      | 3,400  | 15            | 22      | 942     | 4.7     | 155      | 7           | +                      |
| 2           | 48    | 280,000      | 4,000  | 21            | 65      | 819     | 4.7     | 159      | 8.8         | +                      |
| 3           | 48    | 215,000      | 1,800  | 17            | 14      | 819     | 4.8     | 152      | 6.3         | +                      |
| 4           | 17    | 610,000      | 3,410  | 15            | 28      | 2,780   | 6.3     | 152      | 8.3         | -                      |
| 5           | 52    | 335,000      | 8,140  | 30            | 10      | 1,380   | nd      | nd       | 7.5         | +                      |
| 6           | 38    | 213,000      | 8,700  | 28            | 82      | 1,814   | 3.8     | 150      | 7           | -                      |
| 7           | 31    | 259,000      | 550    | 31            | 37      | 851     | 3.5     | 172      | 5.8         | -                      |
| 8           | 54    | 196,000      | 4,300  | 17            | 10      | 950     | 3.8     | 149      | 7.6         | -                      |
| 9           | 54    | 375,000      | 37,000 | 15            | 52      | 800     | 3.9     | 149      | 7           | +                      |
| 10          | 57    | 205,200      | 21,200 | 36            | 10      | 1,090   | 5.9     | 147      | 6           | -                      |
| 11          | 67    | 194,000      | 2,100  | 41            | 8       | 874     | 4.8     | 134      | 7.4         | -                      |
| 12          | 35    | 772,000      | 19,560 | 19            | 4       | 1,033   | 4.9     | 140      | 3.8         | -                      |
| 13          | 37    | 590,000      | 18,040 | 30            | 27      | 1,250   | 4.1     | 142      | 6.6         | -                      |

Normal values
33-55% 2 x 5 (x10^5) 6,000-17,000 7-36 6-90 26-500 3.5-5.9 134-159 6.8

a Jain NC: Schalm's Veterinary Hematology, ed 4. 1986.
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SUN = serum urea nitrogen
ALT = alanine aminotransferase
K = potassium
Na = sodium
T.S. = total solids
LIP = lipase

Results
Canine parvovirus antigens (CPV-2) were seen in 16 cases (33.3%) such that the cause of enteritis was considered to be parvoviral. The causative agent of enteritis or gastroenteritis in the remaining 32 dogs could not be determined. Neither gastrointestinal parasites nor protozoa were found in any of the dogs studied. Based on the history, concomitant clinical signs, hematological and biochemical findings, it was speculated that most of the parvovirus-negative dogs were infected by other viruses (distemper paramyxovirus, adenovirus-1, coronavirus), which usually cause acute gastrointestinal problems.
Serum Lipase Activity

![Table: Serum Lipase Activity](image)

- **Group**: Hyperlipasemic with pancreatitis
- **Number of cases**: 13
- **Range**: 800 - 9,783 U/L
- **Mean ± SD**: 1,185 ± 559 U/L
- **p-value**: < 0.001

- **Group**: Non-hyperlipasemic with pancreatitis
- **Number of cases**: 35
- **Range**: 27 - 504 U/L
- **Mean ± SD**: 164 ± 140 U/L
- **p-value**: < 0.001

- **Group**: Healthy (Controls)
- **Number of cases**: 41
- **Range**: 26 - 500 U/L
- **Mean ± SD**: 186 ± 136 U/L

**FIG. 1** — Serum lipase activity in 13 young dogs with hyperlipasemia, in 35 without hyperlipasemia, and in 41 clinically normal dogs of the same age group.

**Discussion**

The major site of lipase production is the pancreas, although a much smaller amount also derives from the gastric mucosa. The normal reference range of serum lipase activity is totally dependent upon the method used. The highest normal values reported in the dog have been 200 U/L, 600 U/L, and 9,000 U/L. In our laboratory, using the same turbidimetric method, the highest normal value of serum lipase detected in 41 healthy dogs (aged 2 months to 1 year) was 500 U/L.

Serum lipase activity is a more reliable indicator than amylase for diagnosing acute pancreatitis in dogs. Marked elevation of lipase was reported to correlate well with morphologic evidence of pancreatitis. In a clinical study on spontaneous acute pancreatitis in dogs, hyper-lipasemia was detected in 85% of cases; however, a good correlation between the magnitude of lipase elevation and the clinical severity or prognosis of the disease could not be found.

Extrapancreatic causes of hyper-lipasemia in the dog include gastric, renal, hepatic, or neoplastic disease, and dexamethasone administration. Only one of the 13 hyperlipasemic dogs (Case 11) had increased SUN concentration. The azotemia was prerenal, as urine specific gravity supported dehydration. Although lipase is being cleared by kidneys, nephrectomy in experimental dogs did not produce an increase of basal plasma lipase activity. Furthermore, hyper-lipasemia has not been seen in dogs with chronic renal disease. Consequently, the hyper-lipasemia noticed in the azotemic dog may have originated from the pancreas or gastric mucosa. Historical evaluation revealed that none of the dogs studied dexamethasone or any other kind of corticosteroids had been administered before admittance to the clinic.

The turbidimetric method we used for determining lipase activity may give falsely elevated values when there is severe hemolysis and/or hyperbilirubinemia. No hemolysis was visible in the sera of any of the 48 dogs. In addition, the mucosae and skin of the same animals were not icteric, an otherwise expected clinical finding with serum bilirubin values as high as 20 mg/100 mL.

The elevated lipase activity seen in the 13 dogs might be the result of acute pancreatitis or acute gastroenteritis. Massive pancreatic necrosis can occur in dogs with canine parvovirus infection. It is unknown whether the parvovirus is directly cytotoxic to pancreatic tissue or whether pancreatitis develops secondary to profound paralytic ileus and reflux of enterokinase into the pancreatic ducts. In our young dogs, this "biochemical" diagnosis of acute pancreatitis was not confirmed upon necropsy and/or histopathology of the pancreas. The speculated pathogenic mechanisms underlying the induction of pancreatitis in these cases include the reflux of duodenal juice into pancreatic ducts, the role of intestinal bacteria or their toxins or a combination thereof. Under normal circumstances, the reflux of duodenal juice into the pancreatic ducts is unlikely to occur in dogs because of the presence of an independent sphincter muscle located at the duct opening and the high pressure of pancreatic juice into the pancreatic duct.
anti-reflux mechanism sometimes fails in the case of abnormally high duodenal pressure, as may occur in vomiting.24 It should be emphasized that all 13 dogs had experienced forceful and frequent vomiting; however, experimental evidence indicates that reflux of small quantities of duodenal fluid occurs in 38% of normal dogs within 2 hours of feeding.

In conclusion, some young dogs with acute enteritis or gastroenteritis have increased serum lipase activity.

REFERENCES

1. Brobst DF: Pancreatic Function. In: Clinical Biochemistry of Domestic Animals. Ed by JJ Kaneko. San Diego, CA, Academic Press, 1989, pp 398-416.
2. Bush BM: Interpretation of Laboratory Results for Small Animal Clinicians. Oxford, Blackwell Scientific Publication, 1991, pp 324-327.
3. Cornelius LM: Laboratory Diagnosis of Acute Pancreatitis and Pancreatic Adenocarcinoma. Vet Clin North Am 1:671-678, 1976.
4. Attix E, Strombeck DR, Wheeldon EB, et al: Effects of an Anticholinergic and a Corticosteroid on Acute Pancreatitis in Experimental Dogs. Am J Vet Res 42:1688-1704, 1981.
5. Strombeck DR, Parver T, Kaneko JJ: Serum Amylase and Lipase Activities in the Diagnosis of Pancreatitis in Dogs. Am J Vet Res 42:1688-1704, 1981.
6. Strombeck DR, Guilford WG: Small Animal Gastroenterology. London, Wolfe Publishing, 1991, pp 429-458.
7. Schaer M: Acute Pancreatitis in Dogs. Comp Cont Ed 13:1769-1780, 1991.
8. Dillon R: Inflammatory and Neoplastic Diseases of the Pancreas. In: Veterinary Gastroenterology. Ed by N Anderson. Philadelphia, Lea and Febiger, 1992, pp 579-594.
9. Blum AL: Linscheer WG: Lipase in Canine Gastric Juice. Proc Soc Exp Biol Med 135:565-568, 1970.
10. Kaneko JJ: Appendixes. In: Clinical Biochemistry of Domestic Animals. San Diego, CA, Academic Press, 1989, 877-901.
11. Plumb DC: Veterinary Drug Handbook, Pharma Veterinary Publishing, 1991, p 565.
12. Jacobs RL, Lumsden HS, Varnes W: Canine and Feline Reference Values. In: Current Veterinary Therapy XI Small Animal Practice. Ed by RW Kirk, JD Bonagura. Philadelphia, WB Saunders Co, 1990, pp 1250-1277.
13. Schaer M: A Clinical Pathological Survey of Acute Pancreatitis in 30 Dogs and Five Cats. JAAHA 15:681-687, 1979.
14. Hall JA, Macy GW: Acute Canine Pancreatitis. Comp Cont Ed 10:403-415, 1988.
15. Schaepri K, Britt LG, Blackwell CF, et al: Acute hemorrhagic Pancreatitis in the Dog. Arch Surg 107:609-612, 1973.
16. Hardy MR: Inflammatory Pancreatic Disease. In: Veterinary Gastroenterology. Ed by N Anderson. Philadelphia, Lea and Febiger, 1992, pp 215-294.
17. Hall JA, Macy GW: Acute Canine Pancreatitis. Comp Cont Ed 10:404-415, 1988.
18. Center AS: Pathophysiology and Laboratory Diagnosis of Liver Disease. In: Textbook of Veterinary Internal Medicine. Ed by SJ Ettinger. Philadelphia, WB Saunders Co, 1989, pp 1421-1479.
19. Drazner RH: Diseases of the Canine and Feline Pancreas. Proc 52nd Annual Meeting AHA. 1985, p 292-300.
20. Hall JA, Macy GW: Acute Canine Pancreatitis. Comp Cont Ed 10:403-415, 1988.
21. Schapiro K, Britt LG, Blackwell CF, et al: Acute hemorrhagic Pancreatitis in the Dog. Arch Surg 107:609-612, 1973.
22. Hardy MR: Inflammatory Pancreatic Disease. In: Veterinary Gastroenterology. Ed by N Anderson. Philadelphia, Lea and Febiger, 1992, pp 215-294.
23. Keene FL, Doppola RF, Go VL: et al: Interdigestive Canine Pancreatic Juice Composition and Pancreatic Reflux and Pancreatic Sphincter Anatomy. Dig Dis Sci 26:677-684, 1981.
24. Adler G, Kern HF, Schecke GA: Experimental Models and Concepts in Acute Pancreatitis. In: The Exocrine Pancreas: Biology, Pathology, and Diseases. Ed by VL Go. Philadelphia, WB Saunders Co, 1992, pp 1250-1277.
25. Hendricks JC, Di Magno EP, Go VL: et al: Reflux of Duodenal Contents Into the Pancreatic Duct of Dogs. J Lab Clin Med 96:912-921, 1980.
26. Weiner S, Gramatica L, Voegle LD: Role of the Lymphatic Biliary Tract and Pancreas. Am J Surg 119:55-61, 1970.

Rebar Named Dean at Purdue

Veterinary Practice Publishing Company extends its congratulations to Dr. Alan H. Rebar on his appointment as Dean of Purdue University’s School of Veterinary Medicine, effective July 1, 1996. Dr. Rebar served for many years as the editor-in-chief of Veterinary Clinical Pathology.

The Publisher

Dr. Alan H. Rebar was recently appointed the next dean of Purdue University’s School of Veterinary Medicine. He has been head of the Department of Veterinary Pathobiology since July 1995 and served as associate dean of research since 1989 at Purdue. He will succeed Dr. Hugh B. Lewis, dean since 1986. In 1995, Dr. Rebar was elected to a 6-year term on the American Veterinary Medical Association Council on Research.

A professor of veterinary clinical pathology, Dr. Rebar has held other administrative positions in the Purdue Veterinary School, including director of continuing education, director of research programs development, and director of the Veterinary Cytology Resource Center. He joined the Purdue faculty in 1976 and left in 1977 to spend 2 years working as an experimental pathologist at the Lovelace Inhalation Toxicology Research Institute in Albuquerque, New Mexico. In 1979, he returned to Purdue. He received the Award for Excellence in Undergraduate Teaching for Best Teacher in Veterinary Medicine at Purdue.

Dr. Rebar received his DVM degree in 1973 and a doctor of philosophy degree in 1975, both from Purdue. He is a diplomate of the American College of Veterinary Pathologists.