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.[1-3] Lipase exerts its maximal activity in the alkaline environment of the duodenum, hydrolyzing triglycerides to fatty acids.[1]

Hyperlipasemia is a common finding in canine acute or subacute pancreatitis, both clinical and experimental.[4,5] The reliability of serum lipase analysis in the diagnosis of canine acute pancreatitis approaches 72%.[6] 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.[1,3,6-8]

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

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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)	Clinical Findings: Vomiting	Diarrhea	Abdominal Pain	Fever
1	German Shepherd	f	4	+	+[a]	-	+
2	German Shepherd	f	4	+	+[a]	-	-
3	German Shepherd	f	3	+	+[a]	-	+
4	mongrel	m	1.5	+	+	-	-
5	German Shepherd	f	4	+	+[a]		+
6	mongrel	f	5	+	+	+	-
7	GSH Pointer	m	2.5	+	+	-	-
8	GSH Pointer	f	8	+	+	-	+
9	mongrel	f	5	+	+[a]	+	-
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	333,000	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[a]	2 x 5 (x10^5)[a]	6,000-17,000[a]	7-36[b]	6-90[b]	26-500[b]	3.5-5.9[b]	134-159[b]	6.8[b]	

[a]Jain NC: Schalm's Veterinary Hematology, ed 4. 1986.
[b]Clinical Chemistry Laboratory, Faculty of Veterinary Medicine, Thessaloniki, Greece
SUN = serum urea nitrogen
ALT = alanine aminotransferase
K = potassium
Na = sodium
T.S. = total solids
LIP = lipase

a clinical refractometer. For normal lipase activity range, blood from 41 young (< 12 months old) healthy dogs was collected and analyzed.

For statistical method, one-way analysis of variance and square transformation in order to normalize the homogeneity of variances was used. For determination of differences between three groups the Duncan Procedure was used.

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 in

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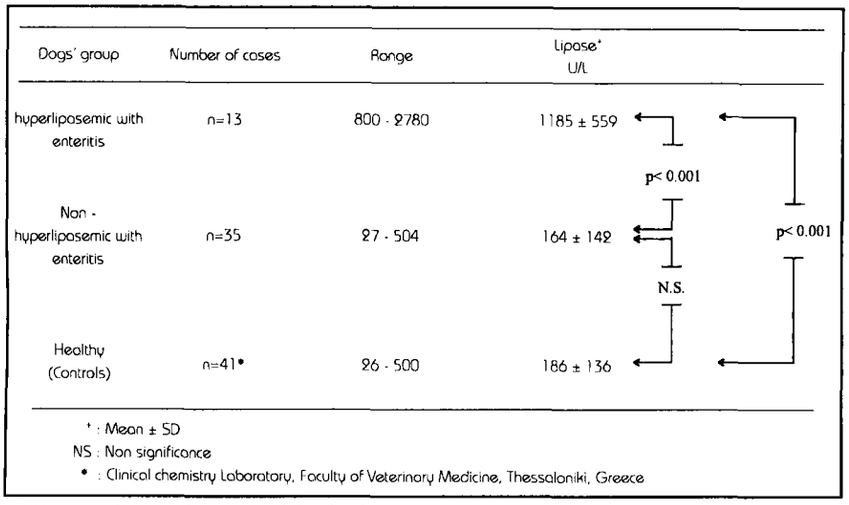

FIG. 1 — Serum lipase activity in 13 young dogs with hyperlipasemia, in 35 without hyperlipasemia, and in 41 clinically normal dogs of the same age group.

young dogs. Additionally, none of the dogs studied had dexamethasone or any other kind of corticosteroids administered before admittance to the clinic.

Thirteen out of 48 dogs (27.1%) had hyperlipasemia (lipase activity > 500 U/L). Signalment, clinical, hematological and biochemical findings from hyperlipasemic dogs are seen in Tables 1 and 2. The 13 hyperlipasemic dogs were presenting depression, anorexia, vomiting, and diarrhea, which was hemorrhagic in five of them. Abdominal pain upon palpation and fever were detected in five out of 13 cases (38.5%). Polycythemia and anemia were seen in two out of 13 cases (15.5%). Leucocytosis was present in four out of 13 dogs (31%) and leucopenia in seven dogs (54%). In Figure 1 the activity of lipase in hyperlipasemic, non-hyperlipasemic, and healthy (control) dogs as well as their significance are presented.

Discussion

The major site of lipase production is the pancreas, although a much smaller amount also derives from the gastric mucosa.[1-3,6,9] The normal reference range of serum lipase activity is totally dependent upon the method used.[3] The highest normal values reported in the dog have been 200 U/L,[10] 600 U/L,[11] and U/L.[12] 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.[2,5,7,8] Marked elevation of lipase was reported to correlate well with morpho-logic evidence of pancreatitis.[5] In a clinical study on spontaneous acute pancreatitis in dogs, hyper-lipasemia was detected in 85% of cases;[13] however, a good correlation between the magnitude of lipase elevation and the clinical severity or prognosis of the disease could not be found.[8,13]

Extrapancreatic causes of hyperlipasemia in the dog include gastric, renal, hepatic, or neoplastic disease, and dexamethasone administration.[5,14-16] 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.[17] Furthermore, hyperlipasemia has not been seen in dogs with chronic renal disease.[6] Consequently, the hyperlipasemia noticed in the azotemic dog may have originated from the pancreas or gastric mucosa. Historical evaluation revealed that in 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[3]. 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.[18]

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.[19] 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.[20] 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,[21] the role of intestinal bacteria or their toxins[22] 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[23] and the high pressure of pancreatic juice into the pancreatic duct.[6] This

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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.[25] It has also been suggested that intraduodenal fluid pressure can be very high in dogs with intestinal obstruction, although the latter is rarely associated with acute pancreatitis.[6] In case of duodenal reflux, enteropeptidase, activated pancreatic enzymes, bacteria, and bile present in the duodenal chyme may all contribute to the development of pancreatitis.[24]

Intestinal bacteria or their toxins that gain entry into the pancreatic duct directly from the duodenum may induce pancreatitis.[22] Moreover, the lymphogenous spread of bacteria from the extrahepatic biliary tract to pancreas could pose another mechanism for initiating pancreatitis.[26] The hyperlipasemia observed may also be attributed to gastrointestinal upset.

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

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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.

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