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From the Departments of Medicine and Pathology, College of Veterinary Medicine, Helsinki, Finland.

SERUM PHOSPHOLIPASE A_2 IN CANINE ACUTE PANCREATITIS

By

E. Westermarck and *E. Rimaila-Pärnänen*

WESTERMARCK, E. and E. RIMAILA-PÄRNÄNEN: *Serum phospholipase A_2 in canine acute pancreatitis.* Acta vet. scand. 1983, 24, 477—487. — During 3 years 28 cases of acute pancreatitis were diagnosed in dogs. In 26 of these dogs, the disease was fatal. The most frequent symptoms were vomiting, anorexia and lethargy. Two thirds showed tenderness upon abdominal palpation. Ascites was found in 3 cases. Of the blood, parameters, serum amylase level was elevated in 86 % and lipase in 89 % of the cases. Sixteen dogs were uremic and half of the dogs were hyperglycemic. Two thirds of the dogs had leukocytosis. Using stepwise multiple regression the best blood parameters explaining acute pancreatitis were leukocytes together with lipase and glucose.

In an attempt to find a more specific serum test for dogs to diagnose acute pancreatitis serum phospholipase A_2 (PLA_2) activity was measured. In sixteen out of the 28 dogs with acute pancreatitis, serum PLA_2 activity was increased. The ascites fluids were rich in PLA_2. Serum PLA_2 is more often increased in the severe necrotizing pancreatitis (80 %) than in the milder forms of acute pancreatitis (44 %). All dogs with increased serum PLA_2 had also increased serum amylase and lipase activities. The dogs with an increased serum PLA_2 and dogs with ascites had fat necrosis in the vicinity of the pancreas. Experimental pancreatitis was induced in 4 dogs by injecting Na-taurocholate and trypsin into the pancreas. In these cases, very high PLA_2 activities in the serum and ascites fluids were detected, but none seemed to be present in the urine samples.

dog; acute pancreatitis; phospholipase A_2.

The clinical diagnosis of acute pancreatitis in dogs is extremely difficult. The symptoms of abdominal distress are variable and nonspecific. The diagnosis is usually based on the clinical symptoms and on supportive evidence from various blood parameters such as increased lipase and amylase levels, but no single method to confirm the clinical diagnosis is fully reliable (*Strombeck* 1979, *Hardy & Johnson* 1980, *Strombeck et al.* 1981, *Rogers* 1983).

Phospholipase A_2 (PLA_2) is a hydrolytic enzyme that splits one fatty acid off the phospholipid to form lysocompounds, which are well-known cytotoxins. The pancreas secretes large amounts of phospholipase into the bowel in the digestive process. In 1961 *Zieve & Vogel* described the serum PLA_2 increase in pancreatitis and recently the measurement of serum PLA_2 has proved to be a new detector for acute pancreatitis in man (*Hashihira* 1975, *Nevalainen* 1980, *Schröder et al.* 1980, *Tykkä et al.* 1984). Increased serum levels of PLA_2 are considered specific for pancreatitis and its quantitation has been used in differentiating mild forms of pancreatitis from hemorrhagic pancreatitis (*Schröder et al., Tykkä et al.*).

The content of pancreatic PLA_2 is smaller in dogs than in man (*Zieve et al.* 1963). Despite low pancreatic contents, PLA_2 in serum and ascitic fluids will rise in dogs as demonstrated by inducing experimental pancreatitis (*Zieve & Vogel* 1961, *Hatao* 1969).

The present study was carried out to study serum PLA_2 levels in canine acute pancreatitis. The study included clinical cases as well as experimentally induced pancreatitis.

The term acute pancreatitis is used to include different forms of pancreatitis, such as oedematous, hemorrhagic and necrotizing pancreatitis as well as the acute processes with fibrous tissue proliferation.

MATERIAL AND METHODS

During 1980—1983, 28 cases of acute pancreatitis were diagnosed in dogs at The Small Animal Clinic of the College of Veterinary Medicine, Helsinki. In 26 of these dogs, the disease was fatal or the animals had to be euthanized because of an unfavourable prognosis.

The clinical symptoms, urine and blood parameters had been suggestive of pancreatitis, but in most cases the diagnosis was supported visually via exploratory laparatomy. The diagnosis was confirmed by histology of pancreatic samples dissected and fixed immediately after death. The specimens were fixed in 10 % formaldehyde and embedded in paraffin. Sections 4 µm thick were stained with haematoxylin-eosin and van Gieson.

Experimental pancreatitis was induced in 4 anesthetized dogs (Halothane anestesia) by infusing a mixture of 1500 IU trypsin/ml (Trypure®, Novo industries, Denmark) and 15 % Na- tauro-

cholate in a volume of 1 ml/kg into the pancreas during laparatomy. In 2 of these dogs the agents were injected into the pancreatic duct and in the two others directly into the pancreatic tissue. The dogs were maintained under inhalation anestesia throughout the 4 h experiment. At the end of the experiment, the dogs were euthanized. Urine and blood samples were collected before and during the experiment at 2 and 4 h. Ascitic fluid was collected as well.

Clinico-chemical analysis

The PLA_2 content of the samples were measured by the gel-diffusion method, which has been described previously (*Westermarck et al.* 1984). Blood samples collected from 30 healthy dogs served as controls for normal PLA_2 activity. In this method, the serum samples are allowed to diffuse out of wells on agar plates containing phospholipid membranes which dissolve and the plates become clear around the wells if PLA_2 activity is present. The level of amylase in the serum was determined by the amyloclastic method according to *Street & Close* (1956) using Merchotest — Amylase kit. The serum lipase activity was determined by turbidimetric method (*Verduin et al.* 1973) using the Lipase monotest kit (Boehringer Mannheim GmbH, West Germany). Serum ALAT and AP were analyzed according to the standard methods of the *Committee on Enzymes of the Scandinavian Society for Clinical Chemistry and Clinical Physiology* (1974) using a Gilford System 3500 analyzer. Serum glucose and urea were determinated by the glucose oxidase and urease methods as modified for the same analyzer. The total leukocyte count of EDTA whole blood was measured in Burker-counting chambers (*Schalm et al.* 1975).

Statistical methods

In order to clarify relationships in the data both correlation and stepwise multiple regression analysis have been used (*Wonnacott & Wonnacott* 1977).

RESULTS

The clinical symptoms, blood parameters and pathology of the dogs with acute pancreatitis are tabulated in Table 1. No breed disposition was evident. Sixteen of the dogs were females

and 12 males. The age varied, half of them were more than 8 years old, but even a 4 month old puppy was affected. The dogs died an average of 4.4 days after the symptoms had been noticed by the owner. The most frequent symptoms included vomiting, anorexia and lethargy. Vomiting was generally severe and only 5 dogs out of 28 had no history of vomiting. All of the dogs were without appetite. Most dogs did not even touch their food, but a few nibbled at snacks. Almost all dogs were lethargic and several could stand only when assisted. Two thirds showed tenderness upon abdominal palpation. The spectrum of palpatory discomfort ranged from mild to a loud yelp when touched. One half of the dogs exhibited polydipsia for some time prior to the onset of pancreatitis. Six dogs had a recent history of symptoms probably associated with pancreatitis. Besides acute changes in the pancreas 3 of these dogs exhibited fibrous tissue proliferation upon histological evolution. Ascites was found in 3 cases, 2 of which involved necrotizing pancreatitis. Nineteen dogs were radiographed, half of which exhibited changes suggestive of pancreatitis; an increased radiographic density in the cranial right quadrant of the abdomen or the doudenal wall appeared irregular.

Serum amylase level was elevated in 86 % and lipase in 89 % of the 28 dogs with acute pancreatitis. The correlation between the levels of these 2 enzymes was 0.83 in the material consisting of 26 dogs with acute pancreatitis (2 dogs with acute pancreatitis were rejected due to their incomplete data) and 30 healthy control dogs (Table 2). Sixteen of the dogs with acute pancreatitis were uremic, some of them severely. Half of the dogs were hyperglycemic and about one third both hyperglycemic and uremic. Serum ALAT was usually normal or only slightly elevated, but AP was elevated as a rule. Leukocytosis was found in two thirds (18) of the dogs and 7 had a lipemic serum. The urine was analysed in 20 cases, half of which were hematuric. As a criterium for a deviation from the "normal limit" the normal range $X \pm 2\ s$ was used. Values outside this were classified abnormal. An analysis of the correlation matrix describing the association between acute pancreatitis (as diagnosed by autopsy and histological examination and coded as either absent or present: 0 and 1, respectively) and the different blood parameters showed that the correlations between acute pancreatitis and total leukocyte count, amylase and lipase are high and that the correlations between acute pancreatitis and glucose, urea and PLA_2 are quite high

Table 1. List of dogs with acute pancreatitis and their clinical symptoms, blood parameters and pathology.

Dog no.	Breed	Sex	Age years	Duration of symptoms/days	Anorexia	Lethargy	Abdominal pain	Vomiting	Diarrhoea	Serum amylase units	Serum lipase units	Serum PLA_2 units	Blood urea mmol/l	Blood glucose mmol/l	Blood leukocyte (thousands)/mm_3	Ascites PLA_2 units	Peri pancreal fat necrosis	Path. anat. diagnosis of pancreatitis (p)
1	Dachshund	F	4	2	++	++	+	++	—	5577	2002	0.30					—	P. ac.
2	Beagle	F	5	5	++	++	—	++	—	9470	3475	6.1	26.6	68.5	12.0		++	P. ac.
3	Griffon	M	2	8	++	++	—	+	—	1627	360	0.50	37.0	6.0	13.4		+	P. ac.
4	German shepherd	F	1	2	++	++	++	++	++	581	235	0.35	20.0	30.8	18.0		—	P. ac.
5	German shepherd	M	6	8	++	++	++	++	—	1917	151	0.45	5.1		10.0		—	P. ac.
6	Afghan hound	F	4	2	++	++	—	++	++	6158	2506	0.65	23.5	8.5	10.0		++	P. ac.
7	Cocker spaniel	F	4	7	+	+	++	—	+	5512	965	0.45	2.7	20.1	15.0		+	P. ac. necrot.
8	German shepherd	M	3	3	+	+	+	+	—	3895	1901	0.45	16.6	5.1	20.5		—	P. ac.
9	Welsh terrier	M	2	7*	++	++	++	—	—	2034	590	0.35	2.9	4.2	23.7		—	P. ac.
10	Afghan hound	F	8	3	++	++	—	++	—	7204	1656	1.1	2.7	19.7	7.0		+	P. ac.
11	Somoyed	F	6	4	++	++	—	++	—	5436	1121	1.7	27.4	50.0	15.0		++	P. ac.
12	Cocker spaniel	F	9	2	++	++	—	++	—	9100	3470	4.5	4.7	5.6	31.2		+	P. ac. necrot.
13	Schnautzer	F	4	3	++	++	++	++	—	3718	2190	8.2	17.2	6.8	19.5	8.5	+	P. ac. necrot.
14	Poodle	M	5	4	++	++	++	+	—	6583	1237	1.1	24.4	45.3	19.1	7.5	++	P. ac. necrot.
15	Maltese dog	F	11	3	++	++	++	++	—	9301	1125	3.8	49.5	76.4	29.0		+	P. ac.
16	Mongrel	M	9	7	++	++	++	++	—	2963	547	0.45	17.0	6.5	12.0		—	P. ac.
17	Cocker spaniel	M	10	4	++	++	++	++	—	7785	3370	1.2	19.7	9.6	23.0		+	P. ac.
18	Dalmatian	F	8	2	++	++	—	++	—	8598	2431	4.0	40.0	6.5	33.7	2.0	+	P. ac. hemorh. necrot.
19	Welsh terrier	F	9	12	++	++	—	—	—	4299	994	7.5	3.0	7.2	34.4		++	P. ac.
20	Fox terrier	F	½	10	++	++	++	++	—	5229	72	0.45	0.6	79.0	18.0		++	P. ac. hemorh. necrot.
21	Dachshund	M	8	4	++	++	—	++	—	11329	2635	1.5	37.3	54.7	28.0		++	P. ac. hemorh. necrot.
22	Mongrel	F	9	2	++	++	+	++	—	4200	937	7.3	17.6	31.4	24.5		+	P. ac.
23	Maltese dog	F	10	4	++	++	—	++	—	7117	882	0.55	60.3	15.1	28.0		+	P. ac.
24	Schnautzer	M	10	2	++	++	++	++	+	11198	3124	16.1	7.9	7.9	23.4		++	P. ac. hemorh. necrot.
25	Samoyed	M	2	5*	++	++	++	++	—	3079	2887	0.55	1.9	5.5	19.8		+	P. ac.
26	Mongrel	M	12	2	++	+	++	—	—	3776	806	0.50	7.0	18.5	15.9		+	P. ac.
27	Mongrel	M	10	4	++	++	++	++	—	4293	1482	5.8	47.6	56.8	30.2		++	P. ac. necrot.
28	Schnautzer	F	12	2	++	++	++	—	—	4504	1450	1.3	2.0	4.8	20.0		++	P. ac. hemorh. necrot.

* Dogs which survived

Symptoms: + greater than usual; ++ much greater than usual; — no signs

Normal blood values: amylase< 2430 units; lipase < 235 units; PLA_2 < 0.55 Sigma units; urea 1.8—7.1 mmol/l; glucose 3.3—8.5 mmol/l; leukocytes (thousands) 6.0—17.0/mm^3

Table 2. The correlation matrix explaining the association between acute pancreatitis and the examined blood parameters. The inter-correlations among the various blood parameters are also shown. The information is based on 26 dogs with acute pancreatitis and 30 healthy control dogs.

	Amylase	Lipase	Urea	Glucose	Total leukocyte count	PLA_2
Acute pancreatitis	0.75	0.73	0.50	0.52	0.77	0.45
Amylase		0.83	0.52	0.56	0.69	0.57
Lipase			0.36	0.29	0.63	0.57
Urea				0.51	0.55	0.21
Glucose					0.40	0.23
Leukocyte						0.54

(Table 2). The data was further analysed by a stepwise multiple regression which resulted in a final regression equation ($y = 0.03$ leukocyte $+ 0.00019$ lipase $+ 0.0059$ glucose $- 0.15$) were y is a dummy variable (0 or 1) explaining acute pancreatitis. Healthy controls were given the value 0 and those suffering from acute pancreatitis were given the value 1 in entering the data into the regression analysis. This equation showed that the best blood parameters in predicting acute pancreatitis (dependent variable) were leukocytes together with lipase and glucose. The three blood parameters explained more efficiently acute pancreatitis than these three parameters supplemented with amylase.

Normal dogs exhibited a low activity for serum PLA_2. Their serum produced only a small clear circle around the well in the gel-diffusion test. The normal values were less than 0.55 Sigma units. The serum PLA_2 activity of 12 dogs with pancreatitis was normal and in 7 dogs slightly elevated (0.65—1.7 units). Nine dogs exhibited a remarkably elevated activity ranging from 3.8 to 16.0 Sigma units. All dogs with increased serum PLA_2 had also increased serum amylase and lipase activities. Of the 10 dogs with necrotizing pancreatitis, 8 had increased serum PLA_2 activity. All dogs with an increased serum PLA_2 had fat necrosis in the vicinity of the pancreas, usually in the omentum. The fat necrosis seems to be connected with free PLA_2. When ascitic fluid was encountered, it contained large amounts of PLA_2 and widespread fat necrosis was found as well. All the patients with ascites exhibited an increased serum PLA_2 activity.

The morphologic changes in the pancreas and its vicinity varied markedly. Sometimes only mild hyperaemia and petechiae were seen. In most cases, however, the pancreas was noticeably altered. Mostly the corpus was enlarged, firm consistency, hemorrhages and white grey dull necrotic areas were found. Fat necrosis and petechiae were seen in the surrounding omentum. Adherences between the pancreas and adjacent organs were found occasionally. The severity of the clinical signs and the macroscopic changes in the pancreas were not always in agreement.

Upon microscopic examination of the histologic slides, oedema and hyperemia of varying degree, as well as leukocytic infiltrations were found in all cases. In the more severe cases there were large necrotic areas in the parenchyma of the pancreas having massive leukocytic infiltrations. Proliferation of connective tissue was encountered in six cases. In experimental pancreatitis, the serum PLA_2 activity correlated to the method of induction of pancreatitis (Table 3). In those cases, where the trypsin and Na-

Table 3. The PLA_2 activities in serum and ascites fluids in 4 dogs with experimental pancreatitis induced with Trypsin and Na-taurocholate.

Dog No	Site where the agents were injected	Serum PLA_2 units			Ascites PLA_2 units		Path. anat. diagnosis of pancreatitis (p)
		0	2 h	4 h	2 h	4 h	
1	Pancreatic duct	0.35	14.0	18.5	120.0	135.0	P. ac. hemorh. necrot.
2	Pancreatic duct	0.40	13.8	10.8	125.0	98.0	P. ac. hemorh. necrot.
3	Pancreatic tissue	0.45	2.6	2.8	107.0	130.0	P. ac. hemorh. necrot.
4	Pancreatic tissue	0.40	2.5	3.1	105.0	160.0	P. ac. hemorh. necrot.

taurocholate were injected into ductus pancreaticus, the PLA_2 activity increased much more than in those cases, where the injection was made into the corpus of the pancreas. Huge amounts, from 1—3 l of ascitic fluid were produced in all induced cases. The PLA_2 activity of ascitic fluid was several times higher than the corresponding serum activity. PLA_2 activity was never encountered in the urine. The pathology of all experimental cases showed hemorrhagic, necrotizing pancreatitis.

DISCUSSION

The greater part of the dogs in our study succumbed to their disease within a few days, despite intensive treatment. This illustrates how fatal acute pancreatitis can be for dogs.

One of the most typical symptoms in pancreatitis is pain upon abdominal palpation: in the present study one out of three patients exhibited pain upon palpation. Absence of this sign does not seem to rule out pancreatitis.

Hematuria and polydipsia, which were exhibited in almost half of the dogs, have usually not been connected with pancreatitis. The hematuria may stem from coagulation disorders present in connection with pancreatitis (*Feldman et al.* 1981). The etiology of the polydipsia is obscure. None of the dogs was known to have any polydipsia-percipitating disease prior to the onset of pancreatitis.

At the onset of pancreatic symptoms it is impossible to distinguish between hemorrhagic pancreatitis and the milder form of acute pancreatitis and at the present time it is difficult clinically to be sure of the diagnosis of acute pancreatitis. It is therefore important to look for new parameters which would be specific for pancreatitis. No single blood parameter was completely indicative of acute pancreatitis. Serum amylase and lipase, which are considered the most important parameters in the diagnosis of pancreatitis, were abnormal in almost all cases. They are, therefore, of indisputable value in the diagnosis of pancreatitis. According to *Strombeck et al.* (1981) serum lipase is more specific than amylase and a low serum lipase almost excludes the possibility of pancreatitis. One has to remember, however, that both enzymes may become abnormal in other conditions as well, for example in liver and renal disorders. Serum glucose, BUN and the total leukocyte count are important parameters when a rapid diagnosis of pancreatitis is strived for, especially during conditions were serum lipase and amylase measurements are not available. In the present study the lipase and amylase activities correlated so well with each other that it seems unnecessary to evaluate both the enzymes. Further diagnostic accuracy can be achieved by combining the serum lipase analysis with the analysis of serum glucose and the total leukocyte count. In human medicine a lot of attention has been given to measuring serum PLA_2 in pancreatitis and recently it has been suggested, that

serum PLA_2 will increase in most cases of hemorrhagic pancreatitis (*Schröder et al.* 1980, *Tykkä et al.* 1984).

It was found in the present study that all dogs with induced hemorrhagic necrotizing pancreatitis had an increased serum PLA_2 activity. Serum PLA_2 of the clinical cases was increased in 16 dogs. There seemed to be evidence that serum PLA_2 is more often increased in the severe necrotizing pancreatitis (80 %) than in the milder forms of acute pancreatitis (44 %). According to this material the measurement of serum PLA_2 does not solve the problem of diagnosing acute pancreatitis in the dog. This, however, can be of great help as PLA_2 is considered very specific for acute pancreatitis. The gel-diffusion method for measuring serum PLA_2 seemed to function equally well on canine and human samples (*Westermarck et al.* 1984). Normal human and canine serum PLA_2-activities are very low and of equal magnitude in both species.

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SAMMANFATTNING

Fosfolipase A_2-halten i serum vid akut inflammation i bukspottskörteln hos hund.

Under en tre års period diagnostiserades 28 hundar med akut inflammation i bukspottskörteln. Tjugosex av hundarna dog av sjukdomen. De mest typiska symtomen var kränkningar, aptitlöshet och apati. Två tredjedelar visade ömhet vid bukpalpation. Tre hundar hade vätska i bukhålan. Av de 28 hundarna hade 86 % onormalt hög amylashalt och 89 % hög lipashalt i serum. Sexton hundar var uremiska och hälften av hundarna var hyperglykemiska. Två tredjedelar av hundarna hade leukocytos. Vid användning av stepwise multiple regression visade det sig att leukosythalten tillsammans med lipas- och glucoshalten var de bästa blodvärden som förklarade den akuta inflammationen i bukspottskörteln.

För att finna ett mera spesifikt serum test för hundar att diagnostisera inflammation i bukspottskörteln, mättes fosfolipas A_2 (PLA_2) halten i serum. Hos sexton av de 28 hundarna med akut bukspottskörtels inflammation var PLA_2-halten i serum onormalt hög. Vätskan i bukhålan innehöll rikligt med PLA_2.

Resultaten utvisar en tendens till oftare förekommande onormalt höga serum PLA_2-halter vid de allvarliga nekrotiserande inflammationerna i bukspottskörteln (80 %) jämfört med de lindrigare formerna av akuta inflammationer i bukspottskörteln (44 %). Alla de hundar

som hade onormalt hög PLA_2 aktivitet i serum hade också onormalt hög lipas- och amylashalt i serum.

Hundar med onormalt hög PLA_2-halt i serum och hundar med vätska i bukhålan hade fettvävsnekroser i närheten av bukspottskörteln. På fyra hundar framkallades en experimentell inflammation i bukspottskörteln genom att injisera Na-taurocholat och trypsin i bukspottskörteln. Hos dessa djur mättes mycket höga PLA_2-halter både i serum och i vätskan i bukhålan, men urinproverna visade ingen PLA_2-aktivitet.

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Reprints may be requested from: E. Westermarck, the Department of Medicine, College of Veterinary Medicine, P. O. Box 6, 00551 Helsinki, Finland.