Acta vet. scand. 1982, *23*, 400—406.

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# PANCREATIC DEGENERATIVE ATROPHY AND CHRONIC PANCREATITIS IN DOGS

## A COMPARATIVE STUDY OF 60 CASES

By

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RIMAILA-PÄRNÄNEN, EIJA and ELIAS WESTERMARCK: *Pancreatic degenerative atrophy and chronic pancreatitis in dogs — A comparative study of 60 cases.* Acta vet. scand. 1982, *23*, 400—406. — During the years 1977—1980 60 cases of non-neoplastic chronic exocrine pancreatic disease in dogs were investigated clinically and pathologically. The disorders were clinically divided into pancreatic degenerative atrophy (PDA) and chronic pancreatitis. Fifty dogs had PDA and 45 of them were German shepherd dogs. The PDA cases formed both clinically and pathologically a homogeneous group except for 1 case. All the dogs had maldigestion and protease activity was absent from the faeces. General inanition and highly atrophic pancreas were the most typical macroscopic findings. Histologically the exocrine pancreas contained atypical acinar tissue and mononuclear cell infiltrations. Five of the dogs died spontaneously, 4 of them had intestinal torsion and 1 had paralytic ileus.

There were 10 dogs with chronic pancreatitis. This group was rather heterogeneous both clinically and pathologically. The pancreas was slightly enlarged and the consistency was firm. The histologic picture was one of fibrous tissue proliferation and inflammatory cell infiltrations in the interstitium. The dogs nutritional state as well as faecal protease activity were normal.

dog; pancreatic degenerative atrophy; pancreatitis.

Many different classification schemes have been devised to categorize chronic pancreatic diseases of dogs (*Holroyd* 1968, *Jubb & Kennedy* 1970). Sometimes the diseases have been classified according to the clinical signs but more often according to pathological changes. The etiology of these diseases is not well understood and the terminology used is very heterogeneous.

In the present paper dogs with chronic non-neoplastic exocrine pancreatic disease have been divided into 2 groups. One

group includes dogs suffering from pancreatic degenerative atrophy (PDA). These animals have a deficiency of pancreatic hydrolases in the gut and faeces. On clinical grounds they form a heterogenous group of cases of chronic pancreatic disease (*Säteri* 1975). In this paper the postmortem picture of dogs with PDA is described with special reference to whether the pathological changes in the pancreas are as uniform as the clinical findings.

The postmortem findings of PDA cases are also compared with those of dogs with chronic pancreatitis. This is a second group of cases of chronic pancreatic disease which can be distinguished from PDA on clinical grounds.

## MATERIAL AND METHODS

Sixty dogs were diagnosed as suffering from chronic exocrine pancreatic disease on clinical grounds. They were seen during 1977 to 1980 at the small animal clinic of the College of Veterinary Medicine, Helsinki, Finland. Fifty of the cases had been diagnosed as pancreatic degenerative atrophy (PDA) and 10 as chronic pancreatitis.

The PDA cases showed signs of maldigestion. Typically the dogs were in poor condition, eating well, even voraciously, and passing bulky, pale, soft and foul smelling faeces. The diagnosis of PDA was confirmed by detecting completely negative faecal protease activity by the soybean stimulation test (*Westermarck & Sandholm* 1980). The PDA group included 45 German shepherds (25 males and 20 females), 3 female collies, 1 male dachshund and 1 male miniature pincher. The age of the dogs ranged from 1 to 5 years, with a mean of 3.2 years.

Ten cases had been diagnosed as chronic pancreatitis on the basis of the clinical signs which included vomiting, depression and anorexia and of blood tests, which often showed increased amylase, lipase, urea and glucose. These dogs had proteolytic activity in their faeces. The group included 3 German shepherd dogs, 2 poodles, 2 crossbreeds, 1 cocker spaniel, 1 Finnish Spitz and 1 Finnish Harrier. There were 6 females and 4 males. The dogs were 4 to 9 years old with a mean age of 6.1 years.

Five dogs with PDA and 4 with chronic pancreatitis died spontaneously and the others were euthanized. In each case the whole pancreas was removed immediately after death and

examined macroscopically. Half of each pancreas was used to measure the activity of pancreatic hydrolases; the results have been published by *Westermarck & Sandholm* (1980). The remaining part was fixed in 10 % formaldehyde and embedded in paraffin. Sections 4 µm thick were stained with haematoxylin-eosin (HE), van Gieson, periodic acid-Schiff and Sudan black. A thorough necropsy was performed on 13 of the PDA dogs and 9 of these suffering from chronic pancreatitis.

## RESULTS

### *Macroscopic findings*

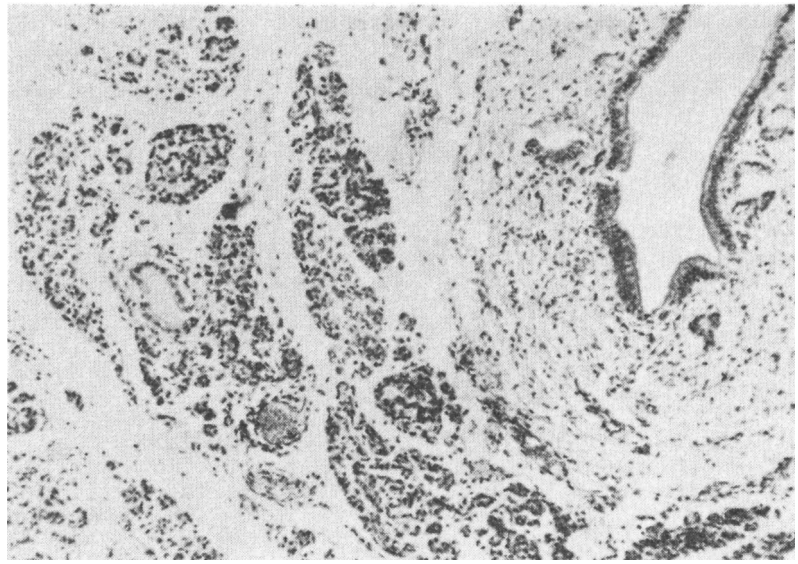
The most common findings in the PDA dogs were general inanition and dilated intestines with loose yellow ingesta and gas. In 49 of the 50 cases clinically diagnosed as PDA the pancreas showed significant atrophy. The length was normal but the thickness was much reduced. The pancreatic ducts were distinctly visible. The organ seemed to consist of connective tissue rather than glandular tissue. One clinical case of PDA was exceptional because the pancreas was macroscopically normal in size and structure. The dog itself was cachectic.

Of the 5 dogs with PDA which died naturally 4 had intestinal torsion and 1 had paralytic ileus. The intestines were very dilated. Massive hyperaemia was present in the gut wall. In the cases of intestinal torsion the jejunum and ileum were involved in the torsion but the stomach was in its normal position.

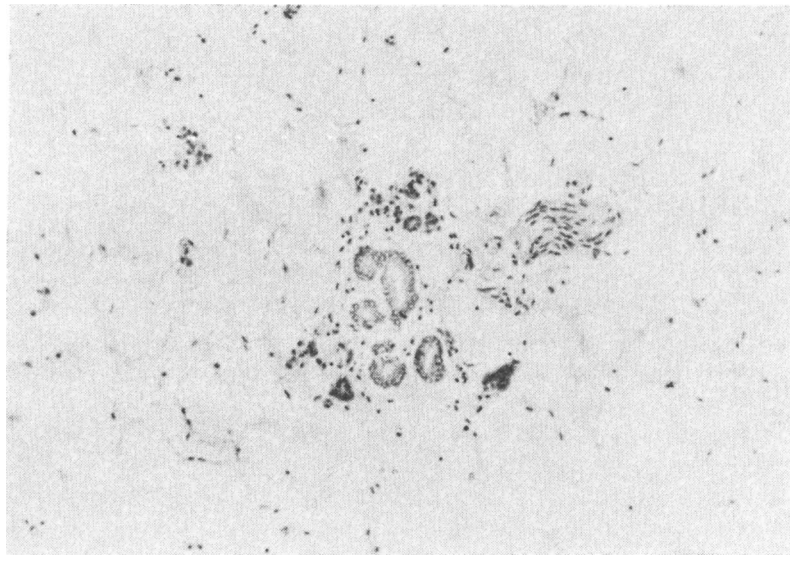
The 10 dogs with chronic pancreatitis were in normal body condition. The pancreas was heterogenously enlarged and the consistency varied from focal to general firmness. The proliferative changes were mostly located in the corpus pancreatis region. In 2 cases fat necrosis was seen in the surrounding tissue. Four of the dogs with chronic pancreatitis had chronic duodenitis and ulcers in the pyloric region.

### *Microscopic findings*

The parenchyma of the pancreas of PDA cases was dominated by atypical acinar tissue. The acini consisted of very small round cells with a central nucleus and a light acidophilic, often slightly granular cytoplasm. Within the atypical acinar tissue some isolated areas of normal acinar tissue were present. In all cases mononuclear infiltrations mainly of lymphocytes and plasma



F i g u r e 1. Atypical acini in pancreatic degenerative atrophy. Acinar cells are small, round with almost no zymogen granules and arranged in nets or in glandular structure. The ducts are clearly visible and there are mononuclear cell infiltrations. H E × 160.



F i g u r e 2. The exceptional case of pancreatic degenerative atrophy. Abundant adipose tissue with very small atypical acinar tissue foci embedded in it. The ducts are prominent. H E × 160.

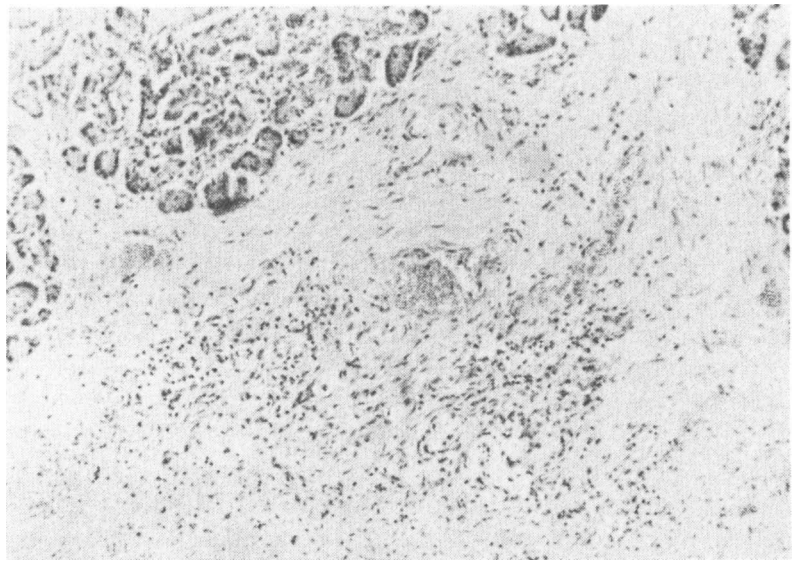


Figure 3. Chronic pancreatitis. Massive inflammatory cell infiltration and fibrous tissue proliferation in the interstitium. H E × 160.

cells were present. The infiltrations varied from mild to moderate and the location from focal to diffuse. The ducts were prominent and fat tissue was almost absent. The vessels and the nerves showed no changes.

The one clinical case of PDA with a pancreas of normal size had a gland containing abundant fat tissue with very small scattered foci of atypical acinar tissue and no mononuclear cells.

The histological findings in the pancreas of dogs with chronic pancreatitis varied from case to case. There were both mild and marked fibrous tissue proliferation and inflammatory cell infiltrations in the interstitium.

## DISCUSSION

The macroscopic appearance of the pancreas from all the clinically diagnosed cases of PDA, except 1 was very uniform. Histologically the glands showed almost identical changes. There were some differences in the amount of atrophic atypical acinar tissue and in the number of mononuclear cells, but it was considered unnecessary to subdivide the material as suggested by *Säteri* (1975). Differences in the histological picture presumably depend on the stage of progression of the disease.

The pancreas of the exceptional case with clinical PDA resembled the cases reported by *Prentice et al.* (1980). There the pancreas consisted of small scattered atypical acinar tissue surrounded by abundant adipose tissue.

In earlier reports dealing with exocrine pancreatic insufficiency one frequent pathological diagnosis was pancreatic fibrosis. This is considered by *Hardy & Stevens* (1975) to be the end stage of chronic pancreatitis. In the present material no such cases were seen.

In the present 3 years survey the 5 diagnosed cases of intestinal torsion and paralytic ileus were all associated with PDA. This finding suggests that abnormal gas fermentation in the intestine, which is typical of dogs with PDA predisposes to intestinal torsion and paralytic ileus.

The etiology of PDA is still unclear, although its hereditary nature has been established (*von Weber & Freudiger* 1977, *Westermarck* 1980). The histological picture of PDA does not exclude the possibility of autoimmune disease or virus infection suggested by the lymphocyte and plasma cell infiltrations. In the PDA cases described in the present paper there were no fibrous tissue proliferations to support the theory of a chronic pancreatitis followed by atrophy. An additional possibility is that the pancreas atrophies due to some disturbance in the mechanism which stimulates activity of the exocrine pancreas.

The clinical and pathological findings in the cases of chronic pancreatitis were rather confusing. The clinical appearance of some cases resembled that of acute pancreatitis but the fibrous tissue proliferation seen at histological examination supported the diagnosis of chronic pancreatitis. The possibility of residival pancreatitis cannot be excluded although the owners of the dogs had not noticed earlier attacks.

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## SAMMANFATTNING

*Bukspottkörtelns degenerativ atrofi och kronisk inflamation i bukspottkörteln hos hundar — En studie omfattande 60 fall.*

Under åren 1977—1980 utfördes en klinisk och patoanatomisk undersökning på 60 hundar med en kronisk sjukdom i bukspottskörtelns exokrina del (med undantag av sjukdom med neoplastiskt ursprung). Materialet uppdelades kliniskt i två grupper: degenerativ atrofi i bukspottskörteln (PDA) och kronisk inflamation i bukspottskörteln. Femtio hundar led av PDA och av dessa var 45 schäfrar. Med undantag av en hund bildade PDA-fallen en både kliniskt och patoanatomiskt homogen sjukdomsgrupp. Alla hundar led av maldigestion och proteasaktiviteten i avföringen saknades hos samtliga hundar. De mest typiska makroskopiska förändringarna var en generell inanition, och en i mycket hög grad atrofierad bukspottskörtel. Histologiskt innehöll den exokrina bukspottskörteln atypisk acinar vävnad med infiltration av mononucleära celler. Fem hundar dog spontant och fyra av dem led av tarmomvridning och en av paralytisk ileus.

Tio hundar led av kronisk inflamation i bukspottskörteln. Denna grupp var både kliniskt och patoanatomiskt tämligen heterogen. Bukspottskörtlarna var något förstorade och konsistensen var hård. Den histologiska bilden dominerades av fibrotisk proliferation i vävnaden och infiltrering av inflamatoriska celler i mellanvävnaden. Hundarna var av normal vikt och proteasaktiviteten i avföringen var även normal.

*(Received May 25, 1982).*

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