Pancreatic Acinar Atrophy in German Shepherds

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ABSTRACT: Pancreatic acinar atrophy (PAA) occurs most commonly in German shepherds and has been shown to be hereditary in this breed. In this disease, pancreatic acinar cells undergo atrophy probably subsequent to immune-mediated inflammation, while islet cells are spared. The exocrine pancreas has a large secretory reserve and only when pancreatic function is decreased to less than approximately 10% do affected dogs develop signs of exocrine pancreatic insufficiency (EPI). EPI causes nutrient malabsorption, particularly of fat and fat-soluble vitamins. In most affected dogs, enzyme deficiency is complicated by concurrent small intestinal bacterial overgrowth (SIBO), which probably contributes to cobalamin malabsorption that often leads to subnormal serum concentrations of this vitamin. Signs most commonly observed in dogs with PAA are weight loss, polyphagia, soft feces, poor haircoat, borborygmus, and flatulence. Vomiting and anorexia are less common signs. Clinical signs usually resolve completely in response to pancreatic enzyme supplementation, although fat absorption does not normalize completely. Fat-soluble vitamins and cobalamin should be supplemented as required. In cases with concurrent SIBO that do not respond to therapy with replacement enzymes alone, antibiotic therapy for concurrent SIBO may be useful, as may be feeding of a highly digestible diet that is low in fiber.

Pancreatic acinar atrophy (PAA) is the most common cause of exocrine pancreatic insufficiency (EPI) in dogs. The prevalence of PAA in German shepherds is higher than in any other breed. The disease is characterized by progressive atrophy of pancreatic acinar tissue. During the early stages of this disease, acinar atrophy is associated with inflammation, but there is minimal evidence of inflammation during the late stage of the disease. Islets of Langerhans are disorganized but remain otherwise unaffected. One case of EPI due to PAA combined with insulin-dependent diabetes mellitus (DM) has been described in a 3-month-old German shepherd. However, the vast majority of affected animals do not develop DM.

ETIOLOGY
Autosomal recessive inheritance of PAA was originally suggested in Swiss German shepherds and was subsequently also hypothesized in a family of Finnish...
Recent studies of two multigenerational families of German shepherds also indicated this condition to be an autosomal recessive trait in this breed in the United States, but multifactorial inheritance cannot be excluded. Identification of a genetic marker for this disease is currently being explored and might allow planning of a directed breeding program aimed at eliminating this trait in the future.

A study of 76 German shepherds with PAA found development of clinical signs of EPI at 1 to 4 years of age in 81% of all cases. However, one study reported early degenerative changes in pancreatic acinar tissue of a 6-week-old German shepherd that subsequently developed signs of EPI at 2 years of age, indicating that the disease is present long before clinical signs ensue. Both parents of this dog also had clinical signs of EPI.

Recent studies showed lymphocytic pancreatitis in German shepherds with subclinical EPI, leading to degeneration of pancreatic acinar cells. However, there was very little evidence of inflammation during the late stages of the disease. These findings suggest an immune-mediated pathogenesis of PAA.

Several alternative theories for the etiology of PAA have been suggested, including deficiency of specific nutrients. Severe protein/calorie malnutrition in children can lead to reversible atrophy of the exocrine pancreas. Copper deficiency in calves and rats and selenium deficiency in chickens can also cause atrophy of the exocrine pancreas. However, in 11 German shepherds with PAA, serum concentrations of copper and zinc were not significantly different from those of normal control dogs, suggesting that copper or zinc deficiency is less likely to play a role in the etiology of PAA. Other nutritional imbalances, acquired as a consequence of underlying intestinal mucosal abnormalities, is another hypothesis for the etiology of PAA in dogs. Most dogs with canine PAA have a history of intermittent signs of gastrointestinal disease long before the development of severe weight loss.

Pancreatic duct ligation in rats causes regression of pancreatic tissue with disappearance of pancreatic acini and the development of dilated pancreatic ducts. It is assumed that spontaneous pancreatic duct obstruction (e.g., due to pancreatic adenocarcinoma) may also result in PAA.

Whatever the etiology of acinar cell destruction, there is a large reserve capacity, and signs of malabsorption do not occur until approximately 90% of the acinar cells have disappeared.

PATHOPHYSIOLOGY

Once a dog shows clinical signs of EPI due to PAA, pancreatic acinar cells are almost totally absent from pancreatic tissue and all that remains is atypical parenchyma with islet cells of Langerhans, ductular structures, adipose tissue, blood vessels, and nerves (Figure 1). Thus dogs do not develop concurrent DM. In contrast, concurrent DM may occur in dogs with EPI caused by chronic pancreatitis, a condition usually seen in dogs older than 5 years of age. However, one case of PAA in a 3-month-old German Shepherd with concurrent DM has been reported. Also, abnormal glucose tolerance has been demonstrated in dogs with PAA. This abnormal glucose tolerance may reflect a compensatory mechanism for malnutrition in dogs with EPI since low serum insulin concentrations enhance lipolysis, resulting in an increased plasma concentration of free fatty acids that provides an energy source.
In dogs with experimental EPI after ligation of the pancreatic duct, daily fecal fat output increased from 1.7 ± 0.27 g/10 kg to 15.2 ± 1.2 g/10 kg. Lipase in the enzyme supplement is sensitive against gastric acid and is partially destroyed in the stomach. Therefore, even after supplementation with pancreatic enzymes, fat digestion remains impaired.\(^{19,20}\) The fat malabsorption leads not only to steatorrhea but also to malabsorption of fat-soluble vitamins, essential fatty acids, and cholesterol.\(^{21,22}\)

Lack of digestive enzymes is not the only factor responsible for malabsorption in EPI. Small intestinal mucosal function is altered and brush border enzyme activities are decreased due to altered protein degradation by pancreatic enzymes in the small intestine. This effect of EPI on the small intestine may reflect a lack of a direct effect that pancreatic secretions have on enterocytes. It may also be due to small intestinal bacterial overgrowth (SIBO) or may arise as a consequence of pathophysiologic changes associated with EPI, including malnutrition.\(^{23,24}\)

Concurrent SIBO has been observed in more than 70% of German shepherds with PAA. SIBO sometimes contributes to the clinical signs observed in dogs with EPI. Multiple factors may be responsible for concurrent SIBO. Large amounts of undigested food in the small intestine provide nutrients for bacterial growth.\(^{25}\) In addition, pancreatic juice in healthy dogs probably has bacteriostatic and antifungal effects, and therefore lack of pancreatic secretions may allow uncontrolled bacterial proliferation.\(^{26}\) Impaired immunologic function due to severe malnutrition has been discussed as another contributing factor.\(^{27}\) Moreover, many German shepherds show relative IgA deficiency in the small intestine, serum, or both, which may predispose them to SIBO.\(^{28}\) SIBO may worsen the clinical signs of EPI, especially if anaerobic bacteria (Clostridium spp.) are involved.\(^{29}\) These bacteria deconjugate bile salts, leading to a reduction of conjugated bile salts in the gut, which plays an important role in fat assimilation in healthy individuals.\(^{30}\) Finally, bacteria degrade fatty acids to hydroxy fatty acids, which may stimulate excessive secretion by the small intestinal mucosa and may cause watery diarrhea.\(^{31}\)

Serum cobalamin concentrations in dogs with EPI are commonly low and remain low even after treatment with pancreatic enzymes.\(^{32,33}\) This suggests that factors other than just the lack of pancreatic enzymes are responsible for malabsorption of cobalamin.\(^{34}\) Both pancreatic intrinsic factor and pancreatic proteases are necessary for absorption of cobalamin.\(^{35}\) Dietary cobalamin is bound to dietary protein and is not available for absorption. Gastric acid and pepsin predigest these dietary proteins in the stomach, and cobalamin is released.

However, cobalamin is immediately bound by the so-called R-protein present in gastric content. In the small intestine, R-protein is digested by pancreatic proteases and the released cobalamin can bind to intrinsic factor for further transport. In dogs, most intrinsic factor is synthesized by the pancreas.\(^{36}\) Therefore, malabsorption of cobalamin in dogs with EPI may occur due to lack of both pancreatic proteases and intrinsic factor but also because an increased number of bacteria may compete with the body for cobalamin in the diet.

**CLINICAL SIGNS**

Clinical signs of PAA are those of EPI. It is impossible to differentiate between PAA and EPI due to other causes on the basis of clinical signs alone.

Depending on the duration of the disease, voluminous soft stools, severe weight loss, and ravenous appetite are usually the most common complaints from owners (Table 1).\(^{37}\) Polydipsia and increased frequency of defecation are also often reported. The feces are often gray or yellowish in color. Some dogs show vomiting, diarrhea, flatulence, borborygmus, or bouts of anorexia. Other dogs have a poor haircoat and smell rancid.\(^{7}\) Dermatologic signs were more commonly found in German shepherds with EPI, whether or not they were treated, than in healthy German shepherds or other breeds with EPI.\(^{57}\)

**DIAGNOSIS**

A specific diagnosis of PAA can be made only using invasive methods (e.g., laparoscopy, exploratory abdominal surgery, necropsy) and the finding of acinar atrophy despite no inflammatory changes or fibrosis on histopathologic examination. However, pancreatic function can be adequately evaluated by several indirect methods. This is sufficient for clinical purposes since PAA is treated like EPI due to any other cause.

The secretin–pancreozymin stimulation test is the gold standard for diagnosis of EPI in human patients with suspected EPI.\(^{38}\) However, this test is time consuming, technically difficult, and expensive and therefore not used in veterinary medicine.

**Serum Canine Trypsinlike Immunoreactivity**

In veterinary medicine, serum canine trypsinlike immunoreactivity (cTLI; determined by a double antibody cTLI kit, Diagnostics Products Corporation, Los Angeles, CA) testing has been established as highly sensitive and specific for the diagnosis of EPI in the dog. This immunoassay quantifies the amounts of circulating trypsinogen and trypsin in serum. Evaluation of a single serum sample has been shown to be extremely sensitive and specific for EPI in dogs.\(^{39}\) The reference
range for cTLI is 5 to 35 µg/L, but only a serum cTLI concentration of 2.5 µg/L or less is diagnostic for EPI in dogs. Dogs with serum cTLI concentrations between 2.5 and 5.0 µg/L need to be further evaluated. Ideally, the dog should be fasted for 12 hours (minimum, 3 hours) before blood collection because feeding can spuriously increase serum cTLI concentration. Porcine trypsin contained in the enzyme supplement.

Recent work indicates that a persistent serum cTLI concentration in the questionable range (2.5 to 5 µg/L) reflects subclinical EPI associated with diminished pancreatic mass and scattered areas of loss of glandular appearance within an otherwise normal pancreas. These dogs should be monitored for progression to clinically apparent EPI. Serum cTLI has been shown to be very stable, and samples do not need to be cooled during shipping (if this assay is not available locally, serum can

### TABLE 1

<table>
<thead>
<tr>
<th>Typical Signs in EPI</th>
<th>Group</th>
<th>Breed</th>
<th>EPI ± Standard Error</th>
<th>Control ± Standard Error</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>%</td>
<td>%</td>
<td>%</td>
<td></td>
</tr>
<tr>
<td>Yellow feces</td>
<td></td>
<td>GS</td>
<td>55.0 ± 7.4</td>
<td>0</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td></td>
<td>RCC</td>
<td>43.0 ± 8.9</td>
<td>5.0 ± 2.7</td>
<td>.00001</td>
</tr>
<tr>
<td>Increased fecal</td>
<td></td>
<td>GS</td>
<td>48.0 ± 7.5</td>
<td>17.0 ± 4.4</td>
<td>.0223d</td>
</tr>
<tr>
<td>volume</td>
<td></td>
<td>RCC</td>
<td>29.0 ± 8.2</td>
<td>8.0 ± 3.2</td>
<td>.00001</td>
</tr>
<tr>
<td>Pulpy feces</td>
<td></td>
<td>GS</td>
<td>36.0 ± 7.1</td>
<td>3.0 ± 1.9</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td></td>
<td>RCC</td>
<td>30.0 ± 8.2</td>
<td>1.0 ± 1.4</td>
<td>.75</td>
</tr>
<tr>
<td>Coprophagia (sometimes)</td>
<td></td>
<td>GS</td>
<td>32.0 ± 6.9</td>
<td>24.0 ± 5.0</td>
<td>.001d</td>
</tr>
<tr>
<td></td>
<td></td>
<td>RCC</td>
<td>26.0 ± 7.9</td>
<td>0</td>
<td>.0043</td>
</tr>
<tr>
<td>Indoor defecation</td>
<td></td>
<td>GS</td>
<td>18.0 ± 5.7</td>
<td>8.0 ± 3.3</td>
<td>NS</td>
</tr>
<tr>
<td>(sometimes)</td>
<td></td>
<td>RCC</td>
<td>32.0 ± 8.4</td>
<td>14.0 ± 4.0</td>
<td>.76</td>
</tr>
<tr>
<td>Flatulence (often)</td>
<td></td>
<td>GS</td>
<td>32.0 ± 6.9</td>
<td>0</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td></td>
<td>RCC</td>
<td>26.0 ± 7.9</td>
<td>1.0 ± 1.4</td>
<td>.21</td>
</tr>
<tr>
<td>Defecation frequency</td>
<td></td>
<td>GS</td>
<td>28.0 ± 6.8</td>
<td>4.0 ± 2.4</td>
<td>NS</td>
</tr>
<tr>
<td>(&gt;3 times/day)</td>
<td></td>
<td>RCC</td>
<td>19.0 ± 7.1</td>
<td>3.0 ± 1.9</td>
<td>.93</td>
</tr>
<tr>
<td>Thinness</td>
<td></td>
<td>GS</td>
<td>18.0 ± 5.7</td>
<td>4.0 ± 2.4</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td></td>
<td>RCC</td>
<td>26.0 ± 7.9</td>
<td>4.0 ± 2.3</td>
<td>.63</td>
</tr>
<tr>
<td>Ravenous appetite</td>
<td></td>
<td>GS</td>
<td>23.0 ± 6.2</td>
<td>4.0 ± 2.4</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td></td>
<td>RCC</td>
<td>19.0 ± 7.1</td>
<td>3.0 ± 1.9</td>
<td>.81</td>
</tr>
<tr>
<td>Diarrhea (≥1–2</td>
<td></td>
<td>GS</td>
<td>16.0 ± 5.4</td>
<td>1.0 ± 1.4</td>
<td>NS</td>
</tr>
<tr>
<td>times/wk)</td>
<td></td>
<td>RCC</td>
<td>19.0 ± 7.1</td>
<td>3.0 ± 1.9</td>
<td>.75</td>
</tr>
<tr>
<td>Borborygmus (often)</td>
<td></td>
<td>GS</td>
<td>9.0 ± 4.2</td>
<td>3.0 ± 1.9</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td></td>
<td>RCC</td>
<td>16.0 ± 6.6</td>
<td>0</td>
<td>.06</td>
</tr>
</tbody>
</table>


The EPI group comprised 45 GSs and 31 RCCs; the control group comprised 72 GSs and 73 RCCs. Significant (*P* < .01) differences in all clinical signs were found between dogs with EPI and control dogs.

Low (*P* < .05) goodness-of-fit *P* value indicates significant interaction, which means that the difference between EPI- and control-group dogs varies also by breed.

Significant difference in fecal volume (*P* < .05) and coprophagia (*P* < .01) between breeds.

EPI = exocrine pancreatic insufficiency; GS = German shepherd; RCC = rough-coated collie; NS = not significant.
be evaluated at the Gastrointestinal Laboratory at Texas A&M University).

**Other Tests for Evaluating Pancreatic Function**

Alternative tests for determining pancreatic function are microscopic examination for undigested nutrients in feces, indirect methods for determining fat absorption (n-benzoyl-1-tyrosyl-p-aminobenzoic acid), assay of pancreatic enzyme activities or concentrations in feces (elastase, trypsin, chymotrypsin), or tests for chymotryptic enzyme activity (bentiromide-p-aminobenzoic acid test). Many of these methods are expensive, and the sensitivities and specificities of all these tests are lower than those for cTLI. In addition, most of these tests are time consuming, expensive, and no longer recommended. A new ELISA for measuring elastase in feces was recently introduced in Europe and has been advertised in the United States. However, this assay shows a lower sensitivity and specificity than serum cTLI determination and would be clinically useful only in dogs that have an obstruction of the pancreatic duct and that have functional EPI but a normal serum cTLI. However, to our knowledge such cases have never been reported in the veterinary literature and are probably extremely rare.

**Serum Cobalamin and Serum Folate Concentrations**

Many dogs with EPI have hypocobalaminemia due to malabsorption of this vitamin as described above. Also, more than 70% of dogs with EPI have concurrent SIBO and/or other concurrent small intestinal disease. Therefore, determination of serum cobalamin and folate concentrations in dogs with EPI is recommended. While neither low serum cobalamin concentration nor high serum folate concentration is very sensitive for SIBO, these tests have been shown to be very specific and may be useful in suggesting antibiotic therapy in patients with EPI that do not respond satisfactorily to enzyme supplementation. It should be noted that folate can be falsely elevated in hemolyzed serum samples since erythrocytes contain high concentrations of folate. Therefore, serum must be separated from the blood clot before submission to avoid hemolysis in transit.

**THERAPY**

Most dogs with EPI due to PAA respond favorably to supplementation of the maintenance diet with pancreatic enzymes. In cases that do not respond to enzyme supplementation alone, vitamin supplementation, antimicrobial therapy, dietary modification, or H₂-blockers can also be useful.
Enzyme Supplementation

Many different preparations of pancreatic enzymes are commercially available, but powdered formulations have been shown to be most effective in dogs. In contrast, enteric-coated tablets are used commonly in humans with EPI. Enteric-coated tablets are less effective in dogs, probably due to a difference in gastric emptying between enteric-coated products and food.

The dose of pancreatic enzymes that is needed for successful therapy of EPI in dogs differs widely. Initial supplementation with pancreatic enzyme powder (2 tsp/20 kg) mixed with each meal is recommended. As clinical improvement becomes apparent, the amount of enzymes can be reduced to the smallest effective dose.

Most dogs with EPI will need at least one teaspoonful of pancreatic enzyme powder daily to provide relief of clinical signs. A single daily meal may be adequate and economical, but two meals are preferable initially if logistically possible.

An economical alternative to commercially available enzyme preparations is chopped raw bovine or porcine pancreas (0.2 oz/kg) given with the food. Porcine pancreas is as effective as bovine pancreas, but there is a potential risk of infection with Aujeszky's disease since the pancreas needs to be fed raw. Fresh pancreas can be stored frozen for at least 3 months without loss of enzymatic activity. The owner should be reminded that raw pancreas, like all raw meat products, should be handled using proper kitchen hygiene to decrease the risk of possible transmission of zoonotic diseases such as salmonellosis. One study has shown that feeding of raw pancreas instead of dried pancreatic powder results in higher lipase activity in the duodenum. However, there is no difference in therapeutic response between dogs that are treated using pancreatic powder or raw pancreas.

Vitamin Supplementation

Although clinical signs of EPI resolve with pancreatic enzyme supplementation alone in most cases, serum concentrations of cobalamin and vitamin E are often decreased in dogs with PAA. Vitamin E deficiency may cause degeneration of skeletal muscle leading to muscle weakness, elevated plasma creatinine phosphokinase concentration, lipofuscinosis of small intestinal muscle, increased dialytic acid hemolysis, failure of spermatogenesis, and gestational failure. In puppies, retinal degeneration has also been associated with vitamin E deficiency. To avoid these complications, vitamin E should be supplemented. A daily dose of 250 to 500 mg tocopherol (vitamin E) given in the food should be administered for 1 month. Anemia with hypoplastic erythropoietic centers in the bone marrow has been described as a consequence of cobalamin deficiency in the dog. In humans with cobalamin deficiency, megaloblastic anemia, metabolic abnormalities (e.g., methylmalonic aciduria or homocystinuria), and neurologic defects (e.g., degeneration of the spinal cord) are observed clinically. Serum cobalamin should be assayed, and if decreased, cobalamin (a weekly dose of 500 µg SC or IM for 1 month, repeated every 6 months) should be administered.

Cases of vitamin K deficiency–dependent coagulopathies have occasionally been described in dogs with EPI and should initially be treated with 2.5 mg/kg vitamin K1 parenterally, followed by 0.25 to 2.5 mg/kg PO.

Antibiotics

Secondary SIBO can cause diarrhea, weight loss, and malabsorption. One study showed that these clinical signs improved with oral supplementation of pancreatic enzymes alone in a group of dogs with experimental EPI. However, enzyme supplementation did not have a significant effect on jejunal microflora in a group of dogs with naturally occurring EPI. Therefore, antibiotic therapy is indicated if signs of EPI persist despite pancreatic enzyme supplementation—particularly if serum cobalamin concentration is decreased and/or serum folate concentration is increased. Administration of tylosin (10 to 15 mg/kg PO bid) or metronidazole (10 to 20 mg/kg PO bid) may improve overall response to therapy. It is our clinical impression that dogs with EPI and complicating SIBO respond more quickly to antibiotic therapy than do dogs with SIBO but without EPI. Therefore, a treatment interval of 2 to 4 weeks may be adequate in most cases. However, some dogs may require a longer antibiotic treatment interval. Alternatively, feeding a diet containing fructo-oligosaccharides (FOSs) may help to alleviate clinical signs by modifying the intestinal bacterial flora.

Diet

Most dogs with EPI do well on a commercial maintenance diet. A strict feeding routine is important for success of therapy. Many dogs with EPI display signs of dietary sensitivity during the course of the disease. SIBO in combination with EPI may be responsible for morphologic or functional changes of the intestinal mucosa. Changing feeding times, amount of diet, or the diet fed or giving treats or table scraps may be responsible for failure of therapy.

Fecal fat decreases when a low-fat diet is fed. However, a low-fat diet will also decrease the absorption of fat, fat-soluble vitamins, essential fatty acids, and cholesterol. It has also been shown that a low-fat diet does not ameliorate signs of EPI. In one experimental
study, it was shown that feeding a high-fat, high-protein diet in combination with porcine lipase maximized fat absorption in dogs with EPI.\textsuperscript{55}

The diet should contain highly digestible protein and carbohydrate sources. Large amounts of undigested, high-molecular-weight protein may cause the development of dietary sensitivity.\textsuperscript{56} Rice is more digestible than corn or wheat.\textsuperscript{57} Undigested carbohydrates in the gut have an osmotic effect and should therefore be avoided in dogs with watery diarrhea.\textsuperscript{58} High concentrations of dietary fiber should also be avoided because certain fiber types (e.g., wheat bran, pectin) have been shown to decrease pancreatic enzyme activity.\textsuperscript{59,60}

Different measures have been suggested to improve fat absorption. However, preincubation of the food with pancreatic enzymes, administration of antacids, and enteric coating of pancreatic enzymes have all been shown to be ineffective. Another approach is using a fat source that is easily digestible and not dependent on pancreatic lipase for absorption. There is evidence that medium-chain triglycerides can be directly absorbed into the portal blood, while dietary fats containing mainly long-chain triglycerides need to be digested by lipase and incorporated into chylomicrons before they can be absorbed.\textsuperscript{61} We are currently investigating the effect of medium-chain triglycerides on the well-being of dogs with EPI.

In dogs with concurrent SIBO, dietary supplementation with FOS may be useful to reduce the number of bacteria in the small intestine. Pathogenic bacteria may not be able to metabolize FOS as effectively as they can other carbohydrates.\textsuperscript{52} Reduced numbers of pathogenic bacteria might contribute to more effective digestion in dogs with EPI.

**Inhibition of Gastric Acid Secretion**

It has been shown that only a high dose of cimetidine (300 mg/20 kg) effectively increases fat absorption.\textsuperscript{19} However, this therapy does not decrease fecal weight.\textsuperscript{19} For treatment of routine cases of EPI, H\textsubscript{2}-blockers are not economically feasible or even beneficial and most dogs respond well to therapy with pancreatic enzymes alone. However, in one case of a German shepherd with PAA, the use of cimetidine (300 mg/20 kg) in addition to pancreatic enzyme supplementation was necessary to achieve therapeutic success.\textsuperscript{62} The use of sodium bicarbonate to neutralize gastric acid is not recommended.\textsuperscript{19}

**Further Diagnostics**

If none of the therapeutic measures described is successful in controlling the clinical signs of the dog, further diagnostics need to be initiated in order to diagnose...
concurrent disorders such as lymphocytic-plasmacytic gastroenteritis.

**PROGNOSIS**

Atrophied pancreatic acinar tissue in dogs with PAA does not regenerate. However, if the owner accepts the expense and effort of a life-long therapy, prognosis for dogs with PAA is good. Some dogs do not reach their former weight and high fecal volume and flatulence may persist, but diarrhea and polyphagia can be controlled in most cases.37 Mesenteric torsion is the most common fatal complication in German shepherds with EPI in Finland.66 The cause of this complication is unknown but may possibly be due to excessive gas in the intestinal lumen.66 Clinical signs in dogs with mesenteric torsion are not as fulminating as in those with gastric torsion, and the disease is usually fatal as it is rarely diagnosed soon enough after it occurs to allow for appropriate intervention. It is unclear why this condition does not appear to be associated with EPI in dogs in the United States or the United Kingdom. Owners of dogs with EPI should be educated about the clinical presentation of this condition, which is characterized by clinical signs suggestive of intestinal obstruction (e.g., anorexia, vomiting, abdominal distension, abdominal pain, and defecation of small amounts of bloody mucoid feces).55

**REFERENCES**


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**About the Authors**

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1. What is the most common cause of EPI in dogs?
   a. chronic pancreatitis
   b. pancreatic parasites
   c. PAA
   d. hepatic necrosis

2. In EPI, digestive enzymes are lacking. Which enzyme in pancreatic enzyme supplements is most sensitive to degradation by gastric acid?
   a. trypsin
   b. amylase
   c. lipase
   d. elastase

3. Canine pancreatic juice contains intrinsic factor, which is needed for the absorption of
   a. fat-soluble vitamins.
   b. cobalamin.
   c. carbohydrates.
   d. zinc.

4. More than 70% of dogs with EPI have concurrent
   a. SIBO.
   b. gastric volvulus.
   c. degenerative myelopathy.
   d. skin disease.

5. A serum cTLI of __________ is diagnostic for EPI.
   a. 2.5 µg/L or more
   b. 2.5 µg/L or less
   c. greater than 5 µg/L
   d. less than 35 µg/L

6. In dogs with EPI, serum folate concentration is often increased because of
   a. increased dietary intake.
   b. increased bacterial synthesis.
   c. diarrhea.
   d. vomiting.

7. The concentration of which fat-soluble vitamin is significantly decreased in most canine patients with EPI?
   a. A
   b. D
   c. E
   d. K

8. What is the most important reason against reducing the fat content in the diet for dogs with EPI?
   a. The caloric content of the diet is severely reduced.
   b. The absorption of essential fatty acids, fat-soluble vitamins, and cholesterol is further decreased.
   c. Palatability of the diet decreases.
   d. Fat is less osmotic than carbohydrates.

9. Which formulation of pancreatic enzyme supplements is most effective in dogs with EPI?
   a. enteric-coated products
   b. tablets
   c. powder
   d. granules

10. What are the most common clinical signs of dogs with EPI?
    a. weight loss and ravenous appetite
    b. diarrhea and vomiting
    c. flatulence and borborygmus
    d. lethargy and changes in character