

The Canine Gastrointestinal Tract: Exocrine Pancreas



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KEY POINTS

- The most commonly recognized diseases of the pancreas are pancreatitis and exocrine pancreatic insufficiency (EPI).
- Signs of acute pancreatitis are usually very severe, and initial management is based on fluid therapy, appropriate medication, and dietary rest; total starvation for a minimum of 2-3 days (or longer) is indicated to restrict pancreatic enzyme release and further inflammation. Initially, not even fluids should be given by mouth
- If starvation for pancreatitis is prolonged, total parenteral nutrition will be indicated.
- Reintroduction of oral intake in animals recovering from acute pancreatitis is based on initial fluid and electrolyte intake, followed by highly digestible carbohydrate, followed by addition of protein and finally some fat.
- Long-term prevention of recurrent bouts of pancreatitis can be difficult but may be helped by feeding a low-fat diet, dieting obese animals, and preventing scavenging.
- Most patients with EPI present with chronic diarrhea and weight loss and poor body condition despite a ravenous appetite.
- Nutrient digestion is impaired due to reduced pancreatic enzyme activity; because fat is the most complex nutrient to digest and lipase is found only in the pancreatic juice, fat digestion is most severely affected.
- Feeding a low-fat diet is generally indicated in patients with EPI; this will help to control diarrhea by reducing the digestive challenge to the gastrointestinal tract and is also useful in the control of small intestinal bacterial overgrowth (SIBO).
- Medium chain triglycerides (MCTs) are a source of easily available fat; supplementation with MCTs can increase energy density and supply essential fatty acids.
- It is very important to prevent dogs with EPI from scavenging inappropriate foods.
- A low-fiber diet is generally indicated in pancreatic disease, although some forms of soluble fiber may be beneficial in the SIBO often associated with EPI.
- All nutrients (protein, fat, and carbohydrate) should be highly digestible, as the activity of all pancreatic enzymes (protease, lipase, and amylase) is reduced.

- Parenteral vitamin B₁₂ administration may be necessary in patients with reduced levels.
- Feeding little and often helps overcome the reduced digestive capabilities and reduces the stimulant effect of stomach filling on pancreatic enzyme release in EPI.

The pancreas has both endocrine and exocrine functions. Its exocrine function involves the secretion of digestive enzymes into the proximal duodenum. These enzymes start the breakdown of dietary components to smaller molecules, which are then broken down further by small intestinal brush border enzymes, and therefore play a crucial role in the digestive process. Pancreatic lipase is particularly important, as the pancreas is the only significant source of lipase in the digestive tract.

In the dog, the pancreatic duct enters the duodenum close to the bile duct. Therefore diseases of the pancreas, small intestine, and liver are often interlinked.

Food intake is an important stimulus to pancreatic secretion, and dietary manipulations are vital in the treatment of the two most common canine pancreatic diseases: pancreatitis and exocrine pancreatic insufficiency (EPI).

ANATOMY

The pancreas is located in the right cranial abdomen in close proximity to the gastrointestinal (GI) tract. The right limb (lobe) lies close to the descending duodenum, and the left limb (lobe) lies close to the transverse colon and stomach. The pancreas is composed of exocrine acini and endocrine islets.



Blood is supplied via the cranial and caudal pancreaticoduodenal arteries and pancreatic branches of the splenic arteries. The parasympathetic vagus nerve supplies the acini.

Dogs have two pancreatic ducts taking enzymes from the acini into the duodenum: a large accessory duct that carries secretions from the right limb of the pancreas to a minor papilla in the duodenum and a smaller pancreatic duct that takes secretions from the left limb and opens next to the bile duct on the major duodenal papilla about 5 cm from the pylorus. The pancreatic ducts of the dog do not enter directly into the main bile duct, and reflux of bile into the pancreatic ducts is less likely in dogs than cats.

EXOCRINE PANCREATIC FUNCTION

Pancreatic juice secreted from the pancreatic acini contains pancreatic enzymes, which are very important in the digestion of food. The pancreatic duct cells secrete bicarbonate to alkalinize the contents of the proximal small intestine, allowing maximal activity of digestive enzymes there. The pH of pancreatic juice is 8.4. In addition to this *exocrine* function, the pancreas also has *endocrine* tissue that secretes hormones. Hormones reach their area of action in the bloodstream and are not secreted via a duct. The endocrine part of the pancreas secretes insulin, glucagon, and pancreatic polypeptide.

The exocrine pancreas secretes the following:

- **Bicarbonate** (HCO_3^-), which neutralizes acid from the stomach, thereby providing an optimal environment for pancreatic enzyme activity
- **Lipase**, which breaks down fats to fatty acids and glycerol; this enzyme is very important - the pancreas is the body's only significant source of lipase
- **Trypsin**, which breaks down protein; to prevent autodigestion of the stomach, trypsin is secreted as trypsinogen, an inactive form, and then activated in the small intestine by an enterokinase secreted by the intestinal brush border
- **Chymotrypsin**, which also breaks down protein; this is secreted in its inactive form, chymotrypsinogen, which is then activated in the small intestine by trypsin
- **Peptidases**, which break down polypeptide chains to amino acids
- **Amylase**, which breaks down carbohydrates
- **Nucleotidases**, which break down RNA and DNA

In the normal pancreas, basal secretion is about 2-10% that of postprandial secretion. Secretion of the initial enzyme-rich fraction peaks at 1-2 hours after eating. The bicarbonate-rich secretion reaches its peak at 8-11 hours after meal. Secretion is under neural and hormonal control. Neural control is vagally mediated in response to the smell and anticipation of food; hormonal control is via cholecystokinin release from the small intestine, stimulated by fat or protein in the GI tract or stomach filling. This results in the release of the enzyme-rich fraction and the secretion of secretin from the small intestine (stimulated by acid in the duodenum or jejunum), which in turn results in the secretion of the bicarbonate-rich fraction.

Protection Against Autodigestion

The pancreas is protected against autodigestion by its own proteolytic enzymes by:

- Synthesis and storage of the enzymes as inactive precursors (zymogens), which are activated in the small intestine
- Segregation of enzymes in the cell in zymogen granules, separate from lysosomes
- Presence in the zymogens of pancreatic secretory trypsin inhibitor, which inhibits free trypsin but is inactivated in the acid environment of lysosomes

In addition, pancreatic proteases that escape into the circulation could be very damaging and are normally neutralized by α -antitrypsin and α -macroglobulin in the circulation. α -Antitrypsin binds proteases transiently and transfers them to α -macroglobulin, which binds them irreversibly and is cleared by the monocyte/macrophage system.

CLINICAL DISORDERS

There are a number of clinical disorders of the exocrine pancreas, including neoplasia and diseases, requiring surgical management. The two most commonly recognized diseases of the exocrine pancreas are pancreatitis and EPI.

Acute and Chronic Pancreatitis

Pancreatitis is an inflammatory condition of the pancreas in which the proteolytic enzymes of its own secretions are activated within the organ, resulting in its autodigestion. The systemic effects of the acute disease are severe and may be fatal. Preventing or inhibiting the release of pancreatic enzymes is therefore an essential component of therapy.

Acute pancreatitis is thought to result from fusion of lysosomes with zymogen granules with resultant activation of proteolytic enzymes within the pancreas, leading to autodigestion. The underlying cause is often unclear, but a number of factors have been implicated, including:

- Obesity
- High-fat diets
- Hyperlipidemia
- Certain drugs

- Toxins, such as anticholinesterases
- Hypercalcemia
- Pancreatic duct obstruction/duodenitis/cholangiohepatitis
- Pancreatic trauma (e.g., traffic accidents, surgery)
- Ischemia/reperfusion (e.g., with shock or general anesthesia)

Clinical signs very variable from mild and/or subclinical to severe, necrotizing, and lethal. Dogs may have one episode of pancreatitis or recurrent episodes. Chronic pancreatitis is, strictly speaking, a description of particular pathology found on pancreatic biopsy, describing chronic inflammation with macrophages and fibrous tissue. However, the term “chronic pancreatitis” is also used clinically for recurrent pancreatitis. Pancreatitis may eventually destroy enough pancreatic tissue to result in EPI and/or diabetes mellitus, although this is not the most common cause of EPI in dogs.

Exocrine Pancreatic Insufficiency

Exocrine pancreatic insufficiency describes a deficiency of pancreatic enzymes. Deficiency only occurs with more than 85% loss of the pancreatic acini. Typically, EPI is caused by pancreatic acinar atrophy. This is an apparently inherited condition seen particularly in young German Shepherds, which account for 52% of dogs with EPI in the USA, but it is also reported in English and Irish Setters.

Exocrine pancreatic insufficiency may also be the result of chronic pancreatitis, severe acute pancreatitis, or pancreatic neoplasia, but only once most of the pancreatic tissue has been destroyed. In these circumstances, concurrent diabetes mellitus may also be recognized. Although carbohydrate and protein digestion may be impaired, it is the digestion of fat that is most severely affected, since lipases are absent from the normal array of brush border enzymes.

Up to 70% of patients with EPI show concurrent small intestinal bacterial overgrowth (SIBO) and secondary damage to the small intestine. SIBO may also adversely affect fat digestion and absorption, because bacteria deconjugate bile salts and produce hydroxy fatty acids. As a consequence, fat absorption is diminished, bile acid recycling is impaired, and the presence of malabsorbed nutrients within the intestinal lumen can cause osmotic or secretory diarrhea. This will contribute to the clinical picture and should be considered when treating affected dogs.

A number of dogs, particularly those presenting with cachexia, also show reduced duodenal enzyme activity. This may be partly due to the SIBO but may also be due to the effects of malnutrition on the GI tract and possibly to the loss of the trophic influence of pancreatic secretions.

CLINICAL AND PHYSICAL FINDINGS

The clinical signs in acute pancreatitis are very variable and range from mild symptoms, like anorexia and depression, to very acute and severe signs. Most cases show acute vomiting, anterior abdominal pain, and anorexia. Some severe cases will also present with shock, disseminated intravascular coagulation (DIC) with petechiae, and rapid death. Additionally, patients may develop jaundice due to temporary occlusion of the bile duct.

Mild colitis caused by local irritation of the transverse colon by the inflamed pancreas is common. Furthermore, the resulting fat maldigestion may lead to secretory diarrhea and colitis. Further clinical signs may relate to other underlying causes, such as hypercalcemia of malignancy or hyperlipidemia. The clinical signs of chronic pancreatitis tend to be waxing and waning anorexia, vomiting, and depression, with or without diarrhea.

Most patients with EPI present with chronic diarrhea and cachexia and a ravenous appetite. The diarrhea tends to contain undigested fat (steatorrhea) as a result of fat maldigestion. However, this can be variable; if digestion is interrupted early, the osmotic effect of molecules may be less significant and not a prominent feature.

General cachexia and fat maldigestion may be accompanied by a deficiency of essential fatty acids, visible as poor skin and coat condition or in more severe cases as seborrheic skin disease. When pancreatitis is the cause of EPI, there may be concurrent signs of diabetes mellitus.

Laboratory Tests in Pancreatic Disease

Laboratory Tests

For Pancreatitis

- Routine biochemistry and hematology tests
- Amylase, lipase, and trypsin-like immunoreactivity (TLI) measurement
- Possibly abdominocentesis

For EPI

- TLI
- B12 and folate
- Possibly fecal tests, including pancreatic elastase, sudan stain, and trypsin activity

Biochemical and Hematologic Blood Screens

Blood samples for hematology and biochemistry screens (including electrolytes) are indicated in pancreatic disease, particularly in acute pancreatitis, to rule out underlying causes such as hypercalcemia or hyperlipidemia. These tests are also useful to assess other organs (e.g., the liver for cholangiohepatitis).

These tests also allow assessment of electrolyte, plasma protein, acid-base, and hydration status. Prerenal failure is common in pancreatitis and causes azotemia and a high urine specific gravity. Hypokalemia is common in vomiting associated with acute pancreatitis; hypokalemia causes GI hypomotility and thus may inhibit recovery. Hypocalcemia is seen in some cases of acute pancreatitis, reportedly due to saponification in surrounding fat. Hyperglycemia may be seen as a stress or “prediabetic” response.

Nonspecific, mild to moderate elevations of hepatocellular enzymes are often seen in acute pancreatitis because of hepatocellular reaction and peri-biliary pancreatic inflammation. Increased bilirubin is also seen in some cases. Hematology screens may show neutrophilia or a stress leukogram in acute pancreatitis. Dehydration may result in hemoconcentration. Some severe, necrotizing cases of pancreatitis progress to DIC with thrombocytopenia, and prolonged coagulation times may be seen. Hematology and biochemistry screens are usually normal in dogs with EPI, although creatinine and albumin may be marginally low because of malnutrition.

Amylase, Lipase, and Trypsin-Like Immunoreactivity

Amylase and/or lipase levels are elevated in some cases of acute pancreatitis but may also be normal due to underlying chronic fibrosis reducing the amount of pancreatic tissue or discharge of enzymes into the peritoneum. There are other sources of both enzymes, but large rises (2.5-3 times above normal or more) are usually of pancreatic origin. Both may alternatively be elevated in renal failure or steroid application.

Elevation in TLI are more specific and sensitive for acute pancreatitis, as the pancreas is the only source for trypsin. However, as the half-life of TLI in the plasma is shorter than amylase and lipase, measurements must be made early.

Measurement of plasma TLI showing low levels is the diagnostic test of choice for EPI. However, in the rare cases where EPI is the result of chronic pancreatitis, diagnosis can be complicated by intermittent elevations in TLI associated with pancreatic inflammation.

Abdominocentesis

Abdominocentesis and analysis of abdominal fluid can be useful in acute pancreatitis, both to rule out other causes of severe abdominal pain (e.g., bowel rupture) and to aid diagnosis. Amylase and lipase levels may be measured in the fluid and are usually markedly elevated and higher than serum levels. In pancreatitis, cytology of the fluid usually reveals a sterile inflammatory exudate.

Vitamin B12 and Folate

Measurement of these is particularly indicated in EPI. Approximately 70% of cases present with concurrent SIBO, which may result in elevated folate and low B₁₂ levels. In addition, lack of pancreatic intrinsic factor in animals with EPI results in poor absorption of vitamin B₁₂, which in some cases can lead to very low blood levels. In these animals, parenteral B₁₂ supplementation is indicated, as this deficiency predisposes to further intestinal damage.

Fecal Samples

The assessment of fecal samples is indicated in EPI to rule out infectious causes of diarrhea and weight loss. Fecal trypsin activity is low in EPI; however, this test can be unreliable with both false-negative and false-positive results.

Sudan stain may be used to show undigested fat, but this is nonspecific. More recent work suggests the measurement of reduced fecal pancreatic elastase, but this is not yet commercially available.

Diagnostic Imaging in Pancreatic Disease

Plain abdominal radiographs help to rule out acute intestinal obstruction, which may show very similar clinical signs.

The pancreas itself is not radiographically visible, and acute pancreatitis may not reveal any radiographic evident changes. Nonspecific changes caused by local peritonitis may include a local loss of detail in the right cranial quadrant (or sometimes even a mass effect) and a dilated, fixed “C”-shaped proximal duodenum with delayed gastric emptying due to functional pylorospasm.

The administration of barium is contraindicated in acute pancreatitis, as the resultant stomach filling will increase the release of pancreatic enzymes. Ultrasonography is very useful in the hands of an experienced operator, where it will reveal a swollen, mottled, hypoechoic pancreas with acute inflammation. Ultrasonography can also guide local paracentesis.

MANAGEMENT

Dietary management is central to the long-term treatment of EPI and the long-term control of pancreatitis. However, the initial acute treatment of pancreatitis requires starvation and aggressive fluid therapy combined with appropriate medication.

The treatment of EPI requires long-term supplementation of pancreatic enzymes. Additionally, treatment for associated SIBO may be required.

Fluid Therapy

The initial treatment of acute pancreatitis requires nil per os, including no water, for 2 to 3 days. Intravenous fluid therapy is essential to provide maintenance levels (in mild cases) or increased levels in severe cases associated with vomiting or shock.

Pancreatic ischemia will perpetuate inflammation, and maintaining circulating fluid volume is therefore very important. Hartmann's solution is usually used, with extra potassium added for hypokalemic patients. In severe necrotizing pancreatitis, plasma transfusions are advisable to replace depleted α -macroglobulin in the plasma, allowing free proteases to be neutralized and preventing DIC.

Medical Management of Pancreatitis

Fluid therapy and initial starvation followed by appropriate dietary management are essential in the treatment of acute pancreatitis. In addition, analgesics are advisable during the acute stages, and appropriate antibiotic cover will help to prevent secondary infection of the inflamed tissue.

There is no specific treatment to prevent or resolve pancreatic inflammation, and therapy is therefore supportive. Antiemetics may be helpful; however, care should be taken not to enhance gastric motility, which may stimulate further pancreatic enzyme release.

Some dogs develop concurrent GI ulceration requiring therapy. Steroids are contraindicated in acute pancreatitis, as they reduce macrophage activity and thus reduce clearance of α -macroglobulin-protease complexes. They also predispose the patient to ulceration of the already compromised stomach and duodenum.

It has been suggested that the supplementation of pancreatic enzymes may have some value in the long-term treatment of pancreatitis, reducing endogenous pancreatic enzyme release by negative feedback mechanism and thus reducing inflammation and pain.

Medical Management of Exocrine Pancreatic Insufficiency

In addition to careful dietary management, the treatment of EPI requires long-term pancreatic enzyme replacement. Enzymes are usually provided as powder, tablets, or capsules sprinkled on the food. It is important to empty the capsules or crush the tablets to allow maximum enzyme activity. Fresh, raw pancreas, which can be frozen in aliquots, may be used alternatively.

Much enzyme activity (up to 83% of lipase activity and 65% of trypsin activity) is lost in the acid pH of the stomach, and the enzyme dose needs to be tailored to the individual. To overcome gastric losses, the dose of enzymes can be increased or gastric pH can be medically raised.

Preincubating the enzymes with the food is generally not successful. In the long-term, it is often possible to slowly reduce the dose of enzymes over time. This may be due to resolution of secondary SIBO and of the effects of chronic malnutrition on enterocytes and brush border enzymes.

Cases of EPI with concurrent SIBO will require long courses of appropriate antibiotic therapy. Cases with low serum cobalamin will require parenteral B₁₂ administration to allow a satisfactory recovery. Patients with EPI as a result of chronic pancreatitis may require additional insulin therapy for diabetes mellitus.

Dietary Management

Efficient nutrient absorption is an important goal in patients with pancreatic disease, both in terms of physically stopping diarrhea and in promoting rapid recovery. Carbohydrate and protein are degraded into their structural units by the dual activities of enzymes secreted into the GI tract and of others originating from the luminal brush border membrane. Fat, however, is more complex to digest and absorb. Some stages of this process are rate limiting and take considerably longer than the degradation of proteins and fats. In the diseased state, this process is particularly vulnerable.

In acute pancreatitis, starvation is initially indicated, followed by careful reintroduction of a low-fat and, initially, low-protein diet. Long term, the prevention of recurrences of pancreatitis relies almost exclusively on dietary control.

Appropriate long-term dietary management is crucial in the successful treatment of patients with EPI. Meeting energy requirements and alleviating fat maldigestion are particularly important considerations in these animals, as the pancreas is the only significant source of lipase.

Dietary Management of Acute Pancreatitis

It is essential in all cases of acute pancreatitis to give nil per os, including no water, for 2-3 days, as stomach filling is a potent stimulator of pancreatic enzyme release. Thereafter, if the animal has stopped vomiting, oral intake can be gradually introduced in the following order:

- Fluids with electrolytes first
- Then carbohydrates, which are least stimulatory to pancreatic enzyme release
- Then some protein, which will stimulate enzyme release
- Finally, some fat, which stimulates enzyme release most

However, some animals require starvation for considerably longer than 2 days before pancreatic inflammation subsides; in some cases, this can be up to several weeks. In these individuals, total parenteral nutrition is indicated. After resolution of the acute inflammation, the animal should be fed a low-fat diet long term.

Energy

Energy intake is an important consideration in both EPI and acute pancreatitis.

Starvation is required to help resolution of pancreatic inflammation in pancreatitis. At the same time, patients can develop hypermetabolism, and this combination can rapidly result in protein-calorie malnutrition. Total parenteral nutrition may therefore be indicated in these patients. In the long term, control of recurrent bouts of pancreatitis can be difficult, and feeding a low-fat diet appears to help, as does dieting in obese individuals. In low-fat diets, calories are predominantly obtained from carbohydrate and protein; all these nutrients need to be highly digestible, as all pancreatic enzymes are reduced.

It is important to feed patients according to their current bodyweight, not their ideal bodyweight, as overfeeding can further trigger diarrhea. If the patient needs to gain weight, this needs to be done by increasing the volume of the chosen diet in small steps; this may take considerable time but is required to avoid further bouts of diarrhea.

Supplying sufficient energy in the diet becomes a major consideration in animals with EPI, since compromised digestive function may result in cachexia. Also, EPI is often seen in large breeds with high calorie requirements (e.g., German Shepherds).

As fat is a very energy dense nutrient, it can be a challenge to design a low-fat diet that is energy dense at the same time. Low-fat diets can be supplemented with medium-chain triglycerides (MCTs), such as coconut oil. These MCTs contain chains of 8-12 carbons (compared with the 16-18 carbons in normal fatty acids), do not require bile acids for absorption, and are taken directly into capillaries rather than lymphatics. However, MCTs must not be oversupplemented as they can cause osmotic diarrhea when fed in large doses; a general recommendation is to add 1/4 to 4 teaspoons per dog per day in divided doses. In some individuals, MCTs can cause vomiting. They are contraindicated in liver disease as they may worsen encephalopathy. MCTs do not carry fat-soluble vitamins, which may have to be supplemented separately. However, when feeding a commercially available well-balanced low-fat diet, no supplementation will be required.

Protein

Short-term feeding of a low-protein diet can be indicated in the management of acute pancreatitis, as dietary protein will stimulate pancreatic enzyme release. However, it is important not to restrict dietary protein in the long-term management of either pancreatitis or EPI, as this will worsen cachexia and protein malnutrition and further compromise digestion because of the deleterious effects of protein malnutrition on the small intestine. Sufficient protein intake is required for adequate small intestinal epithelial turnover and maintenance of GI immune function. Glutamine is an important energy source for small intestinal epithelial cells.

Additionally, protein provides a crucial source of calories in fat-restricted diets. The chosen protein should be of high biologic value and easily digestible, as all pancreatic enzymes, including proteases, are compromised.

Fat

The pancreas is central to fat digestion. This is emphasized by the absence of lipases in the normal array of brush border enzymes, making the process dependant on normal pancreatic function.

Fat is the most potent stimulator of pancreatic enzyme release. Dogs with EPI show a severe disruption of fat digestion in response to the lack of pancreatic lipase; this is often accompanied by concurrent SIBO, further impairing fat digestion. Restriction of dietary fat intake is therefore a cornerstone in the management of pancreatic disease. Fat restriction will help to minimize the challenge to the GI tract and restrict the amount of unabsorbed molecules within the GI lumen, thereby decreasing the potential for osmotic and secretory diarrhea. In addition, this allows the intestinal mucosa to rest and inflammation to subside.

Fat intake is not, as such, essential in the dog, except for the delivery of essential fatty acids and fat-soluble vitamins. These are, however, only required in small amounts. Fat usually contributes a significant proportion of the daily energy intake, because fat is more energy dense than carbohydrate or protein. In cachectic patients, it can be a considerable challenge to achieve weight gain with a low-fat diet, particularly in large breeds, which are more prone to EPI. Weight gain may only be achieved very slowly over a considerable time span. Fat can partly be replaced by medium-chain triglycerides.

Carbohydrate and Fiber

When a low-fat diet is indicated, carbohydrates tend to provide the main source of calories in the long-term management of pancreatitis and EPI. Carbohydrates provide the least stimulus to pancreatic enzyme release; therefore a diet based almost exclusively on highly digestible carbohydrates, such as rice, is recommended for initial feeding after a bout of pancreatitis. However, patients with reduced pancreatic function also show impaired amylase activity, and all dietary carbohydrates must therefore be highly digestible.

It has also been shown that soluble fiber may impair the activity of or possibly even absorb pancreatic enzymes. Additionally, dietary fiber may also reduce small intestinal absorption of essential nutrients, such as minerals. Insoluble indigestible fiber will also reduce overall digestibility of the diet.

In healthy dogs, specific soluble fiber sources, such as sugar beet pulp, have shown to have a beneficial effect in small intestinal bacterial flora, encouraging the growth of “beneficial” bacteria and helping to prevent the development of potentially “harmful” bacteria, thereby helping to ensure a healthy GI flora. The clinical significance of this in

SIBO has not yet been determined but may have future implications for dogs with EPI and concurrent SIBO.

Vitamins and Minerals

Cobalamin (vitamin B₁₂) may be reduced in EPI because of a deficiency of the pancreatic intrinsic factor needed to absorb vitamin B₁₂. Additionally, there may also be effects of concurrent SIBO with bacteria metabolizing cobalamin. Vitamin B₁₂ is necessary for small intestinal health; thus when diagnostic tests reveal low intestinal levels, supplementation is recommended. Supplementation should be parenteral rather than enteral, as intestinal absorption is impaired.

Supplementation with fat-soluble vitamins may be considered when feeding a home-prepared low-fat diet, particularly in EPI associated with significant fat maldigestion. Commercial low-fat diets, however, should be balanced and contain all essential nutrients; thus additional supplementation is not indicated, as this could lead to toxicity. Medium-chain triglycerides do not act as carriers for fat-soluble vitamins.

Vomiting and anorexia often result in hypokalemia, and potassium is the most important macromineral to consider in pancreatic disease. This is particularly important in dogs receiving intravenous fluids that are low in potassium. Initial potassium supplementation via fluids, and later via the diet, may therefore be required when blood levels are low.

Some studies have suggested a depression of zinc absorption as a consequence of EPI (Boosalis *et al.* 1983), and possibly a similar mechanism for copper (Abdulla *et al.* 1978).

SUMMARY

The pancreas has both endocrine and exocrine functions. Its exocrine function is crucial for digestion, as the pancreatic enzymes protease, amylase, and lipase are responsible for the breakdown of dietary proteins, carbohydrates, and fats.

The most commonly seen pancreatic diseases are pancreatitis and EPI. Both are characterized by a severe impairment of nutrient digestion, particularly fat, leading to diarrhea, weight loss, and cachexia. Nutritional management is therefore a crucial aspect of therapy, focusing on supplying adequate energy and nutrient intake and utilization.

With acute pancreatitis, initial starvation is indicated to restrict enzyme release and further inflammation. Fluid therapy, and possibly total parenteral nutrition, may be required during this time. Dietary rest is then followed by feeding a highly digestible low-fat, and initially low-protein, diet. Feeding a highly digestible low-fat diet will also help in the long-term prevention of recurrence.

Dietary fat restriction is indicated in EPI to allow resolution of clinical signs, particularly diarrhea. Fat restriction will help to minimize the challenge to the GI tract and restrict the amount of unabsorbed molecules within the GI lumen, thereby decreasing the potential for osmotic and secretory diarrhea. In addition, this allows the intestinal mucosa to rest and inflammation to subside.

It is important to feed patients according to their current bodyweight, not their ideal bodyweight, as overfeeding can further trigger diarrhea. If weight gain is required, food intake can be increased in small steps over a considerable period of time. It is important to feed the chosen diet consistently and prevent the dog from scavenging. All nutrients need to be highly digestible, as all pancreatic enzyme function is impaired.

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