Merck veterinary manual pancreatitis

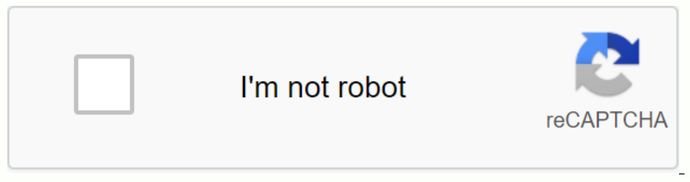

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Illustration of the dog's pancreas: Alveolus in the picture refers to the acinar cells of the exocrine pancreas. Cells form circular clusters. [1] These are cells that produce pancreatic enzymes needed to digest food. Canine pancreatitis is an inflammation of the pancreas that can occur in two very different forms. Acute pancreatitis[2] is sudden, while chronic pancreatitis is characterized by a recurring or persistent form of inflammation of the pancreas. Cases of both can be considered mild or serious. [3] The background of the Pancreas consists of two parts: the smaller endocrine part, which is responsible for the production of hormones such as insulin, somatostatin and glucagon, and the larger exocrine part[4], which produces the enzymes needed to digest food. Acinar cells make up 82% of the total pancreas; these cells are responsible for the production of digestive enzymes. [1] [5] Pancreatitis pathophysiology is caused by the automatic digestion of the pancreas, which is thought to begin with an increase in the secretion of pancreatic enzymes in response to stimulus[6][7], which can be any source from table residues to getting into the garbage for drugs, toxins and trauma. [3] [8] Digestive enzymes are released too quickly and begin to affect the pancreas instead of the food they normally digest. [2] [8] [9] [10] Once the process cascades, inflammatory mediators and free radicals are released and pancreatitis develops, causing an intensification of the process. [9] Clinical signs Clinical signs may vary from mild gastrointestinal agitation to death, with most dogs showing common gastrointestinal agitation symptoms such as vomiting, anorexia, painful abdomen, agitated posture, diarrhoea, fever, dehydration and lack of energy, with vomiting being the most common symptom. [8] [11] [12] These symptoms are not specific to pancreatitis only and may be associated with other gastrointestinal diseases and diseases. [3] [8] [13] Acute pancreatitis can provoke fluid accumulation, especially in the abdominal and thoramin (thorac) regions, acute kidney damage and cause inflammation of the arteries and veins. Inflammation triggers the clotting factors of the body, or exhausts them to the point of spontaneous bleeding. [8] [14] This form may be fatal in animals and humans. [12] Chronic pancreatitis may be present, although no clinical signs of the disease are found. [13] Pancreatitis may result in exocrine pancreatic insufficiency if the acinar cells of the organ are permanently damaged; pancreatic enzymes then need replacement with pancrepasis or similar medicines. Damage can also spread to the endocrine part of the pancreas, leading to diabetes mellitus. [15] Whether diabetes is transient (temporary) or permanent depends on the severity of damage to the beta cells of the endocrine pancreas. [14] Risk factors Although various causes of dog pancreatitis are known, such as fat diet, trauma, etc., pathophysiology is very complex. [2] [13] Pancreatitis may be idiopathic; no real causal factor can be found. [9] [12] Obese animals as well as animals fed a high fat diet may be more susceptible to the development of acute and chronic pancreatitis. [2] [11] [16] Some dog breeds are considered susceptible to the development of pancreatitis including miniature chnauzers, cocker spaniel, and some terrier breeds. [8] [9] [11] [17] Miniature schnauzers as a breed tend to develop hyperlipidemia, excess of circulating fats in the blood. [18] The breed, which appears to be at risk of an acute form of pancreatitis, is a Yorkshire terrier, while Labrador retrievers and miniature puddings appear to have a reduced risk of acute disease. Genetics can play a role in the risk factor. [2] Dogs suffering from diabetes mellitus, Cushing's disease (hyperadrenocorticism), hypothyroidism and epilepsy are at increased risk of pancreatitis. [2] [14] Diabetes and hypothyroidism are also associated with hyperlipidaemia. [19] [20] Persons with other types of gastrointestinal diseases and dogs who have had previous bouts of pancreatitis are also at increased risk of this disorder. [2] Treatment No treatment for canine pancreatitis has been approved. Treatment of this disease is supportive, and may require hospitalization to attend to the dog's nutritional and fluid needs, pain and solutions to any other processes of the disease (infection, diabetes, etc.) [15] while letting the pancreas heal on its own. [3] [11] Treatment often involves resting the pancreas for a short period of time, during which the patient does not receive any food or fluids by mouth, but is fed and hydrated with intravenous fluids and a feeding tube. [8] [12] Dehydration is also controlled by the use of liquid therapy. [21] [14] [15] However, a specialist from Texas A&M University stated, There is no evidence that food retention has any beneficial effect. Other experts also agreed with his view. [13] Canine pancreatitis is complex, often limiting the ability to access the disease. Postpancreatitis management A low fat diet is indicated. [3] Medications known to be associated with pancreatitis should be avoided. [13] [14] Some patients benefit from the use of pancreatic enzymes on a complementary basis. One study showed that 57% of dogs followed an acute pancreatitis attack for six months, either continued to show inflammation of the organ or had reduced acinar cell function, although they had no symptoms of pancreatitis. [13] [22] See also References to pancreatitis ^ and b Gross and microscopic anatomy of the pancreas. Colorado State University School of Veterinary Medicine. 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Obtained from of WikiVet English Introduction For general introduction see Pancreatitis General Disease Signaling susceptible breeds include: Labradors, Miniature Puddings, Miniature Schnauzers, Yorkshire Terriers Increased risk of disease occurs with obesity, diabetes mellitus, hyperadrenocorticalism, previous gastrointestinal illness or recurrent seizures. In addition, middle-aged dogs are more likely to be affected and male and spayed females are affected more frequently than whole females. History and clinical symptoms Often a history of eating fat food. Clinical symptoms include anorexia, vomiting, abdominal pain, lethargy, depression and nausea. Diarrhea is also often a common feature sometimes with blood, fresh or melaena occurs due to the proximity of the inflamed pancreas to the duodenum and colon. More severe cases may occur in shock, acute renal failure, jaundice (due to ftal hepatic necrosis) or cardiac arrhythmias. Pulmonary edhemia, pleural effusion, widespread bleeding, DIC, mild ascites, dehydration (mild to moderate) and pyrexia may also be present. Crab abdominal mass can be palpated. Laboratory tests On hematology may be leukocytosis, increased volume of packed cells due to dehydration, thrombocytopenia, neutrophilia and left displacement. For biochemical changes may include azotemia, increased liver enzymes, hyperbilirubinemia, hyperglycaemia in cases of necrotizing pancreatitis and hypoglycemia in cats with purulent pancreatitis. Hypercholesterolaemia and hypertriglyceridaemia are also common in dogs. An increase in digestive enzymes of the pancreas (amylase, lipase, trypsin-like immunoreactivity (TLI), phospholipase A2 and pancreatic lipase immunoreactivity (PLI) will also be present. Laboratory tests specific to the pancreas All pancreatic enzymes increase after renal failure (except PLI), which makes it difficult to determine the true cause of the increase. However, the increase threefold are mainly due to pancreatitis, while a five-fold increase rarely came out as pancreatitis. An increase in lipase, amylase and phospholipase A2 may also be of hepatic, gastric, intestinal or neoplastic origin. In cats: Amylase and lipase have no diagnostic value. Serum cat trypsin-like immunoreactivity (fTLI) is a specific test for exocrine pancreatic function, but the sensitivity of the test varies between 30% and 60%. By comparison, the feline pancreas immunoreactivity test (fPLI) was found to be more specific and sensitive in the diagnosis of feline pancreatitis. In dogs: A significant increase in serum lipase is a more reliable marker of Amylase. However, administration of corticosteroids increases lipase activity up to five times. Immunoreactivity of serum pancreatic lipreactivity (cPLI) is the most sensitive and sparing test for the diagnosis of canine pancreatitis. Diagnostic imaging Survey X-ray graphs are rarely useful, but findings may include increased density in the right craybar abdomen, decreased contrast, reduced granularity and the stomach can be shifted to the left. In addition, the descending duodenum can be shifted to the right, with the presence of medial matter and thickened walls. Gastric distention may be visible and the passage of barium may be delayed, indicating abnormal peristalsis. Radiography is useful for excluding differentials. Abdominal ultrasound is highly specific with a sensitivity of 70% in dogs and 30% in cats, but is dependent on the operator. Findings include enlargement of the pancreas, peritoneal effusion, hypoetogenic pancreas (pancreatic necrosis) and hyperecogenic surrounding tissue. Explorive laparotomy/autopsy Findings the Pancreas will be edematous and soft with fibrinous attachments to surrounding organs, there may be free fluid in the peritoneal cavity and pancreatic liquefaction if it is severe enough. Pseudocysts may be present, as well as mental and pancreatic bleeding and areas of fat necrosis. A biopsy should be taken to provide evidence of inflammation. Treatment Acute treatment General treatment includes correction and maintenance of fluids, while any underlying cause is treated. Then support is given for the inflammatory process to subside. Oral feeding should be excreted for a short time in patients with vomiting, but enteral and parenteral feeding may be well tolerated. Analgesia should always be administered without symptoms of pain. Recommended options include subcutaneous pethidine, intravenous or continuous infusion rate of morphine or transdermal fentanyl. Dogs may also be given intraperitoneal lidocaine or bupivicain. If pancreatic infection is suspected, then antibiotics should be administered, trimetoprim-sulfonamide and enrofloxacin have good penetration into the pancreas. Foods can be gradually introduced low in protein and fat because they are more likely to cause symptoms. Fat can be further introduced if symptoms have not yet returned. If symptoms appear, then further hunger should be carried out. Total parenteral nutrition can be used to keep animals that are unable to tolerate food at all. Cases often require supportive care, aggressive treatment with fluids will be necessary to treat dehydration and fluid loss from diarrhea and vomiting. Renal function and potassium levels should be monitored and potassium supplemented if necessary. Patients may develop metabolic acidosis in acute pancreatitis or alkaline due to vomiting. If diabetes mellitus develops, this may require insulin therapy. Further management may respiratory distress, bleeding disorders, renal failure, cardiovascular problems and neurological disorders. In addition, transfusions of whole blood or plasma with a serious disease that may α macroglobulins may be administered. Albumin also provides oncical support and limits of pancreatic ischaemia and edema. For short-term use in fulmination pancreatitis, corticosteroids can be administered together with fluids. Long-term treatment can lead to unwanted complications. Long-term treatment In most patients who have one episode, they may need only to avoid fat foods. Recurrent hypertriglyceridaemia may need pharmacological intervention. Prognosis The disease varies greatly, and the prognosis can vary from complete recovery to death. In general, if it is uncomplicated one episode patients will make a good recovery. Acute pancreatitis Introduction In dogs, the condition is possibly caused by reflux of duodenal contents into the pancreas of the duct causing the release and activation of enzymes through inflammation and therefore leakage. Most cases showed necrosis and fibrosis and only 3% had acute purulent pancreatitis. This condition is quite common in dogs and is caused by sudden ingestion of fat food. However, the risk increases when concomitant conditions such as obesity, diabetes mellitus, hyperadrenocorticalism, previous GIT disease or epilepsy are present because these have a relationship with the pancreas in some way. Signaling Yorkshire terriers, laboratories, minature poodles are susceptible. It is most common in middle-old dogs. Males and speyed females are at greater risk than intact females. Clinical signs Clinical signs are acute at onset and include severe abdominal pain, vomiting, diarrhea and anorexia. May or may not be the presence of ikterus. Acute hemorrhagic pancreatitis can present itself as shock and collapse. Diagnosis Clinical signs and history e.g. Lipase measurement may have normal or elevated results in pancreatitis. In dogs, you would expect a 3- to 5-fold increase in acute lipase pancreatitis, and this is indicative of disease. Amylase is not specific in dogs and is not commonly used to diagnose this disease. Immunoreactivity serum trypsin immunoreactivity (TLI) is also of limited value. Serum immunoreactivity of pancreatic lipase (PLI) usually increases and cPLI & fPLI look promising as sensitive and specific markers of pancreatic inflammation. Treatment and control Intitially the animal should be starved for 1-2 days maximum and placed on intravenous fluid therapy (crystalloids, colloids or plasma). Precipitation therapies (azathikoprin) and antiemetics and intestinal protectors may be administered. The dog will need analgesy. If the condition is very severe, surgical intervention to remove necrotic tissue may be indicated. Control may include changing the on a low fat diet Chronic pancreatitis Repeated mild episodes of acute pancreatitis and pancreatic necrosis result in progressive destruction and pancreatic fibrosis and chronic pancreatitis. If a significant proportion of the pancreas is affected, epi symptoms develop +/- DM. the pancreas appears distorted ans shrivelled. Nodular matter with fibrous adhesions to adjacent tissues may be present. The condition may be an accidental autopsy detection. Phalonal pancreatitis may occur during the parvovirus of the canine parvovirus and infection with the distemper virus. Clinical signs Clinical signs include chronic abdominal pain, anorexia and occasional vomiting and diarrhoea. There may also be the presence of ikterus. Diagnosis Diagnosis is usually blood tests. These may show that lipase is normal or elevated. In dogs, a 3-5-fold increase in lipase is indicative of pancreatitis. Amylase levels are usually normal and not much help. Serum trypsin immunoreactivity (TLI) is normal or elevated in chronic disease and is also of limited value. The immunoreactivity of serum pancreatic lipase (PLI) will increase, and cPLI & fPLI will look promising as sensitive and specific markers of inflammation of the pancreas. Treatment and control fluids may be necessary and analgesia. Placing the dog on a low fat diet is useful in controlling the disease. It may be important to perform subsequent blood tests later to test for EPI, as this is often a consequence of chronic pancreatitis. Pancreatitis – Dog Learning Resources Literature Search recent publications via CAB Abstract (CABI log in required) Pancreatitis in Cats and Dogs Publication Full Text Articles Full text articles are available from CAB Abstract (CABI sign up in required) updates on pancreatitis in dogs. Simpson, K. W.; Svoboda, M. ; Czech Veterinary Association of Small Animals, Prague, Czech Republic, 2006 World Congress Proceedings. 31. World Congress of the Association of Small Animals, 12. dealing with a spectrum of diseases In practice article Blood, DC and Studdert, V.P. (1999) Saunders Comprehensive Veterinary Dictionary (2nd Edition), Elsevier Science Ettinger, S.J. and Feldman, E.C. (2000) Textbook of Veterinary Diseases of Internal Medicine of Dog and Cat Volume 2 (Fifth Edition), W.B. 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