Review Article

Journal of Advanced Veterinary Research
(2025) Volume 15, Issue 4, 529-534

Current insights into the most common pancreatic diseases in cattle: An updated review

Shimaa A. Abdelatty[1], Khaled M.A. Hassanein[2], Ghada A. Abou El-Ella[1], Mahmoud Rushdi[1]

[1]Department of Clinical Pathology, Faculty of Veterinary Medicine, Assiut University, Assiut, Egypt.
[2]Department of Pathology, Faculty of Veterinary Medicine, Assiut University, Assiut, Egypt.

ARTICLE INFO

Recieved: 26 April 2025

Accepted: 29 September 2025

*Correspondence:

Corresponding author: Shimaa Abdullah Abdelatty
E-mail address: shimaaabdullah6@gmail.com

Keywords:

Pancreatitis, Diabetes Mellites, Cattle pancreas, Pancreatic diseases, Amylase, Lipase.

ABSTRACT

Pancreas is one of the biggest and most important glands in cattle. The pancreas has both endocrine and exocrine functions. It produces hormones essential for controlling blood glucose levels, bicarbonate ions that maintain acid-base balance and enzymes that aid in the digestion of feed. Effective pancreatic function is essential for guaranteeing optimal digestion and nutrient intake. Nonetheless, different pancreatic conditions can hinder the effectiveness of this essential organ. The pancreatic diseases in cattle consist of acute pancreatitis, chronic pancreatitis, necrosis of pancreatic fat, pancreatic tumors, diabetes mellitus, pancreatic stones and pancreatic fluke infections. This research aimed to identify the different pancreatic diseases impacting cattle currently, examine their effects on pancreatic histopathology and investigate related biochemical alterations to enhance understanding of their influence on cattle health and productivity.

Introduction

The agricultural ecosystem depends on cattle for food, dairy products, natural fertilizer, economic possibilities and environmental advantages. Cattle are an important source of protein for human consumption primarily farmed for their flesh (Oerly *et al*., 2022).

Feed digestion is a vital step in converting nutrients into valuable outputs that are essential for productive purposes like meat or milk (Harmon and Swanson, 2020) . In high-producing ruminants, protein represents a high cost, environmental and dietary concern, whereas starch represents the major component of dietary energy thus, it is essential to provide both of them with optimal amounts. The way that these animals digest food is changing according to modern feeding practices veterinary nutritionists need to pay more attention to how they use starch and protein for optimal production (Harmon and Swanson, 2020).

Cattle are crucial to milk and meat production, supplying products such as milk, meat and dairy byproducts that people depend on significantly. To generate high-quality milk and meat, they need to be given an adequate and balanced diet. This diet needs to be properly digested and absorbed without depleting essential nutrients, a process that largely relies on the pancreas. The pancreas is a digestive gland with a complex structure located in the peritoneal cavity and posterior to the rumen. It has a head, body and tail without any boundaries between them. The head is located near the midline of the body of the animal. While the tail extends to the left, where it touches the spleen (Duncan, 2011). It is composed of both endocrine and exocrine tissues that perform several functions (Jenstad and Chaudhry, 2013). It plays a central role in breaking down ingested nutrients that are not fermented in the rumen and products of ruminal fermentation (Guo *et al*., 2021).

The endocrine portion of the pancreas is compact and nestled within the exocrine region and made up of clusters of islet cells. These islet cells are divided into α, β, δ and PP cells that are responsible for secreting different hormones: glucagon, insulin, somatostatin and pancreatic polypeptides, which regulate various functions like gastrointestinal motility, pancreatic secretion and gallbladder contraction, respectively (Brereton *et al*., 2015). Although they make up a very minor portion of pancreatic cells overall. Endocrine cells release hormones that have crucial regulatory roles in regulating blood glucose levels among other functions (Moullé and Parnet, 2019; Guo *et al*., 2021).

Pancreatic acini are the fundamental structural component that performs the exocrine function. These acini are made up of cone-shaped serous gland cells primarily responsible for producing, storing and releasing different pancreatic digestive enzymes. They include amylases, lipases and proteases that aid in the digestion of starch, fat and proteins within the chyme respectively (Guo *et al*., 2021). Pancreatic juice contains inorganic salts, electrolytes, zymogen particles and additional fluids. It is transported via a duct (accessory pancreatic duct) into the duodenum (Brereton *et al*., 2015). A duct system within the gland also connects with the common bile duct (Wass, 1965).

A better understanding of the normal physiological mechanisms controlling pancreatic function in ruminants could lead to innovative methods of improving nutrient digestion and thereby increasing the efficiency of growth and lactation (Croom *et al*., 1992). The intricate structure of the pancreas is one of the reasons why research on pancreatic exocrine control in ruminants is progressing slowly (Harmon and Swanson, 2020; Guo *et al*., 2021). In vitro investigations have reached great importance in pancreatology at the end of the 20th century, investigations in intact animals are still needed to answer several questions and to verify results obtained in vitro (Niebergall-Roth *et al*., 1997).

It is difficult to diagnose pancreatic disease as there are no specific clinical signs. Following a necropsy, the pancreas' histologic examination usually confirms the diagnosis (Ettinger *et al*., 2016). Furthermore, the diagnosis of pancreatic disease in a laboratory is challenging and not often undertaken. In addition, research on pancreatic diseases in cattle is

ISSN: 2090-6277/2090-6269/

lacking, consequently, this study was performed to investigate pancreatic diseases affecting cattle, with their underlying effect on blood biochemical measurements and associated histopathological changes.

Pancreatic diseases

The diseases of the bovine pancreas include Pancreatitis , fatty pancreas (Tani *et al*., 2017), pancreatic tumor, pancreatic lithiasis (Kelley *et al*., 1996), pancreatic fluke infestation , and diabetes mellitus (Jubb *et al*., 2012).

Acute and chronic pancreatitis

Pancreatitis is an inflammatory condition of the pancreas brought on by activating digestive enzymes before they are released into the small intestine. It results in pancreatic tissue self-digestion and inflammatory injuries such as pancreatic edema, bleeding and necrosis (Jia *et al*., 2018). Most cases of acute pancreatitis in cattle are triggered by chemicals such as chloroform in pancreatic tissue, which stimulate inflammation (Mohamed *et al*., 2003).

Chronic pancreatitis is defined as a persistent often progressive inflammation of the pancreas that leads to lasting damage to the pancreatic structure, which may result in irreversible dysfunction of both pancreatic exocrine and endocrine functions. Diagnosing chronic pancreatitis clinically is difficult due to the typically mild or subclinical nature of the condition and the fact that its clinical manifestations are nonspecific. Although there are no specific clinical symptoms in dogs with chronic pancreatitis, the most typical ones are diarrhea, stomach discomfort, persistent vomiting and appetite loss (Milastnaia and Dukhnitsky, 2019).

There is little research on acute and chronic pancreatitis in cattle. However, compared to studies on humans, dogs and cats. Pancreatitis is thought to occur less frequently in cattle. It's unclear how often pancreatitis occurs in cattle. However, they are seldom clinically recognized due to a lack of pathognomonic clinical symptoms and conclusive test results. Inflammatory and degenerative alterations in the bovine pancreas are seen during the necropsy of cows with different diseases, and spontaneously pancreatitis was discovered (Veiling, 1975; Groom, 1994; Doherty *et al*., 1998; Braun *et al*., 2001).

In induced acute pancreatitis, there was an increase in serum lipase and amylase levels in the first few days of the disease followed by a progressive decline in values. Also, there were increases in triglyceride and cholesterol levels. liver enzymes and glucose levels were not affected. Trypsin level was decreased and fat droplets were present in the fecal matter of the animals. The elevated levels of lipase and amylase enzymes are related to constriction or occlusion of the pancreatic duct due to inflammation. In addition, there was evidence of widespread pancreatic necrosis, which was followed by fibrosis and ultimately total pancreatic sclerosis (Hassan *et al*., 1980).

A previous study by Mohamed *et al*. (2003); the pancreas was found to have necrotic regions and hemorrhage with several fluid-filled cystic cavities. Several groups of inflammatory cells, mostly neutrophils, lymphocytes and interlobular septa were noticeably edematous. The inflammatory tissue has many venous thrombi dispersed throughout it. The authors reported rapid rises in the mean lipase (7.9-fold) and amylase (5.4-fold) activities, which parallel each other and peak 48 hours after pancreatitis induction. The mean blood lipase concentrations did not revert to baseline levels as quickly as the serum amylase value. There were very little variations in the mean levels of calcium, albumin and glucose in the experimental group and the controls. Additionally, the blood urea nitrogen (BUN) and creatinine tests were normal. Serum lipase and amylase concentrations don't seem to be very useful for diagnosing pancreatitis in cows. Evidence suggests that elevations in lipase and amylase levels cannot serve as a standard for definitive diagnosis, at least in cases with experimentally produced pancreatitis, although they can be used to support a diagnosis.

Fatty pancreas

The disease is also known as lipodystrophy or lipomatosis. In cattle, fat necrosis is a common condition marked by the accumulation of necrotic fat particles in the belly cavity. There are other locations where these necrotic fat masses can be detected such as the intestines, mesentery of the spiral colon, mesorectum and retroperitoneal region (Tani *et al*., 2017).

Since the 1960s, the cattle industry has recorded cases of fat necrosis, particularly in Japan (Katamoto *et al*., 1996; Oka *et al*., 2015; Tani *et al*., 2017; Lee *et al*., 2023). The development of fat necrosis has been linked to obesity, genetics, inactivity and consumption of a high-energy diet (Katamoto *et al*., 1996).

The incidence of fat necrosis in JB cattle was higher than in other breeds and it has been linked to economic loss in the Japanese cattle industry because it causes intestinal obstruction which can result in death (Yılmaz, 1995; Saisho *et al*., 2007; Tharwat and Buczinski, 2012). Additionally, Japanese black (JB) breeding cows that receive insufficient roughage and lack of exercise in their pens may develop abdominal fat necrosis (Shimada *et al*., 1988).

Fat necrosis in cows is also associated with excessive fattiness of the abdominal adipose tissue during the developing stage and consequent disruptions in lipid metabolism (Motoi *et al*., 1984). Excessive fattening is thought to produce fatty infiltration of the pancreas, acute or chronic pancreatitis, pancreatic enzymatic juice leakage and necrotic fat lesions in cattle (Katamoto *et al*., 1996). The pathophysiology of bovine abdominal fat necrosis has not received much attention, with most of the reports focusing on pancreatic lesions (Tani *et al*., 2017).

There is no indication that this disease is malignant and the composition of the fatty deposits is similar to that of normal cow fat (Radostits *et al*., 2006). Numerous clinical symptoms, including intestinal stenosis, urine retention, dystocia and infertility are brought on by the disorder (Jones *et al*., 1997). Little patches of fat necrosis can be noticed during an ordinary laparotomy of the right flank (Herzog *et al*., 2010). A differential diagnosis that includes intestinal adenocarcinoma, lymphosarcoma and peritoneal tumors such as mesothelium is necessary because ultrasonography alone cannot guarantee an accurate diagnosis (Radostits *et al*., 2006).

Macroscopically, an enlarged pancreas with fat necrosis revealed a fatty and light brown color. Additionally, excessive multifocal white strips of infiltrative adipose tissues accompanied by mineral deposits were seen on the cut surfaces of the pale and enlarged pancreases (Tani *et al*., 2017). Microscopically, the pancreases in the fat necrosis (FN) of JB cattle showed different stages of fatty infiltration (fatty pancreas), saponification (fat necrosis), acinar atrophy, infiltration of inflammatory cells and fibrosis (Tani *et al*., 2017).

A disruption in lipid metabolism was confirmed by the results of a blood lipid profile; nevertheless, serum activities of lipase and amylase were within reference values. A prior research (Oka *et al*., 1988), The blood concentrations of FFA were greater than the control data, whereas the concentrations of total cholesterol, triglycerides and phospholipids were lower, Cattle fed tall fescue also had lower blood cholesterol concentrations (Stuedemann *et al*., 1985; Tharwat and Buczinski, 2012).

Pancreatic tumor

Limited cases of pancreatic tumors in aged cattle have been documented. In cattle that had been slaughtered, 16 primary pancreatic tumors were found, including three exocrine carcinomas, one neurofibrosarcoma, one neurofibroma and eleven malignant islet cell tumors. There were many sites of metastasis (Kelley *et al*., 1996). The fact is that animals raised for food are typically slaughtered shortly before they reach an age at which carcinomas may be expected among other factors. When they are slaughtered, many steers, calves and cattle are in good health and are under, close to, or slightly above puberty. Italy has reported on several cattle instances involving both non-metastasizing and metastasizing islet tumors. The histology findings are convincing and it doesn't seem plausible that they are confused with non-neoplastic alterations (Hayes and Sass, 1987).

Pancreatic tumors are rare and seldom appear on radiography unless they are extremely big, however, they can be seen on sonography (Kelley *et al*., 1996). Spasmodic spasms that involve head retraction, eye-rolling and limb stiffness in a cow whose islet cell cancer was later shown to be non-metastasizing. The animal died before the blood sugar level could be determined, but in the previous study, the author concluded that the convulsive symptoms were consistent with hyperinsulinism after studying reports of deliberately induced hypoglycemia in cattle (Tokarnia, 1961). Upon antemortem examination of the cows with islet cell tumors, no clinical symptoms were found, except two cattle's inability to stand. In a single case of functioning bovine islet cell tumors, seizures have been seen (Tokarnia, 1961).

Macroscopically, Endocrine tumors were characterized as multilocular solid nodular masses ranging from white to yellow in the pancreas or near the small intestine. Some primary tumors had a well-defined shape and were enclosed by a thick collagenous connective tissue capsule, while others lacked encapsulation and penetrated the pancreatic tissue. Both encapsulated and invasive islet cell tumors frequently arise within the same animal. Numerous animals exhibited hemorrhagic tumor formations (Kelley *et al*., 1996).

Microscopically, the neoplastic cells appeared rounded to multi-sided. Featuring finely granular eosinophilic cytoplasm. Seldom, tumor cells had several sizable eosinophilic intragranules in the cytoplasm. Nuclei were frequently located in the center were elliptical to circular with moderate quantities of spotted chromatin and possessed large single or double nucleoli centered. Anisokaryosis was sometimes apparent although mitotic figures were rare. Cytoplasmic edges were at times unclear when cells were tightly clustered. These cells were organized in lobules, clusters, or strands. frequently encircled by vascular sinuses or limited by delicate fibrovascular tissue (Kelley *et al*., 1996).

Macroscopic appearance in cattle with exocrine tumors was significant fat necrosis, mineral deposits in abdominal fat and fibrosis encircling the pancreas and intestine leading to notable thickening of the peritoneum. In some cases, there was icteric with several firm lobulated lesions in the liver (Kelley *et al*., 1996).

Exocrine carcinomas showed a wide range of histologic characteristics. poorly differentiated, comprising mainly of cells that are oval to circular vesicular nuclei, one to three notable nucleoli, and eight to fourteen mitotic figures. Another case was poorly defined carcinoma made up of columns and clusters of cells divided by dense collagenous connective tissue. Neoplastic cells appeared small, featuring pleomorphic nuclei and limited eosinophilic, finely granular cytoplasm. well-differentiated acinar cell carcinoma. The anisokaryotic nuclei, which are oval to round in shape, frequently had a basal orientation within the cells. Two to four mitotic figures were observed. The cytoplasm exhibited bright eosinophilia and had a vacuolar or granular appearance. Numerous metastatic sites in the liver exhibited an acinar or small tubular arrangement (Kelley *et al*., 1996).

To the best of the author's knowledge, there is a lack of research on biochemical findings in pancreatic tumors in cattle. However, biochemical finding in pancreatic tumors was reported in animal models and humans.

Increased levels of the laboratory markers total bilirubin (TBil) and carbohydrate antigen 19-9 (CA19-9) were linked to human pancreatic cancer (Zhang *et al*., 2018). Laboratory results in pancreatic tumors show raised serum levels of immunoreactive elastase (IRE) in 70% of individuals and carbohydrate antigen (CA 19-9) in 68%. Amylase and carcinoembryonic antigen (CEA) demonstrated increases of 30% and 28%, respectively, in humans (Hayakawa *et al*., 1988).

Bovine pancreatolithiasis

Pancreatic stones are often numerous, hard, white and resemble tiny sand grains (Jubb *et al*., 2012). The condition is usually neglected during postmortem examination unless the gross pancreatic lesions are severe enough to trigger a pathologist's detailed investigation of the gland (López and McKenna, 2018). Typically, it is an accidental finding during autopsy or discovered by meat inspectors at the slaughterhouse (Jubb, 1993). The animals have no clinical symptoms of pancreatolithiasis (Groom, 1994).There were clinical signs of colic in the case history in some cases (Jubb *et al*., 2012).

It is proposed that pancreatolithiasis in the affected cow may be caused by or result from persistent inflammation of the pancreatic duct (Nourani and Dehkordi, 2017). Pancreolithiasis seems to be associated with the existence of silicate in the soil (Longinelli and Verine, 1977). Season, breed and age all affect its occurrence. There have been cases documented in dairy breeds, with Jerseys having the lowest incidence, Holstein-Friesians having the lowest and Red Danish having the greatest prevalence. In animals less than four years old, the frequency was 0.1%, while in animals older than four years, it was 0.82%. Females seem to be impacted more often than males, and more instances are often seen in the fall and early winter. Although a genetic predisposition has been proposed, nutrition has also been implicated as a contributing factor (Veiling, 1975).

The pancreas had a hard texture and an increased nodular appearance under a microscope (López and McKenna, 2018). The pancreatic lobes were both solid and swollen, with an uneven capsular surface. Calculi ranged in diameter from 0.5 cm to 1.2 cm and were spherical, white, smooth, and firm. They may be single or multiple and may exist as small concretions or well-developed calculi. Pancreatic calculi have a dull brown color (Tharwat *et al*., 2013).

Calcium carbonate made up the majority of the stones' chemical makeup, with traces of magnesium ammonium phosphate (Groom, 1994). On a cutting surface, the pancreatic ducts seemed fibrotic and included scores of round-faceted pancreatoliths with a diameter of 0.5–2.0 mm. The pancreatic ducts are found to be cystic (duct ectasia), rough-surfaced and white stones of varying sizes were seen inside the ducts (Nourani and Dehkordi, 2017).

Microscopically, When the pancreas was examined under a microscope, the lamina propria and submucosa of the principal and secondary ducts showed widespread fibrosis, and the lumens were enlarged. Epithelial hyperplasia alternated with focal to patchy epithelial necrosis. There was a modest, widespread, submucosal infiltration of neutrophils and macrophages, along with a lower number of lymphocytes and plasma cells and intraepithelial lymphocytes were prevalent. With irregular clusters of rather normal acinar divided by thick fibro collagenous trabeculae, acinar atrophy was evident. Throughout the acinar structures, islets were sporadically distributed. and significant acinar atrophy (Groom, 1994; López and McKenna, 2018).

Although there was a decrease in the exocrine and endocrine components of the pancreas, there was probably insufficient impact on pancreatic function to be clinically noticeable (Groom, 1994). Serum and hematological biochemical profiles showed elevated lactate dehydrogenase activity, leukocytosis, neutrophilia, lymphopenia, hypoalbuminemia, hypergammaglobulinemia, and hyperglycemia. Lipase and amylase values were within normal ranges(Tharwat *et al*., 2013).

Chronic interstitial pancreatitis and chronic wasting disease caused by Eurytrema spp.

Eurytrema sp. is a trematode parasite that often lives in the pancreatic ducts but can also be found in the bile ducts and less frequently in ruminants' small intestines. *Eurytrema* spp including *Eurytrema coelomaticum*, *Eurytrema pancreaticum* and *Eurytrema ovis* (Bassani *et al*., 2007).

Eurytrema spp are found across Europe, Asia, South America and Oriental Russia (Yamaguti, 1975). The only species known to exist in Brazil is *E. coelomaticum* (Travassos, 1944). The parasites *Eurytrema coelomaticum* and *Eurytrema pancreaticum* are mostly found in the pancreatic ducts of buffalo, sheep, poultry and cattle (Vianna, 1987; Dorny *et al*., 1996; Rachid *et al*., 2011a). It affects the animals at the age of two to three years (Pantiu Andrea *et al*., 2024).

This trematode has an 80–100 day prepatent period in sheep and cattle (Soulsby, 1982). This fluke's life cycle consists of two intermediate hosts: grasshoppers of the species *Conocephalus* and land snails of the genus *Bradybaena* (Soulsby, 1982).

The life cycle of *Eurytrema pancreaticum* involves multiple hosts. Eggs are passed in feces and ingested by land snails (*B. similaris* in Brazil) (Mattos Junior, 1987). Inside the snail, two generations of sporocysts develop, producing cercariae that are released onto pastures before dawn. Grasshoppers (*Conocephalus* in Brazil) or tree crickets (*Oecanthus* in other regions) ingest the cercariae, which develops into infective metacercariae within three weeks. Final hosts are infected by accidentally consuming infected grasshoppers. The metacercariae encyst in the duodenum, migrate to the pancreas via ducts and cause chronic interstitial pancreatitis, particularly affecting the left lobe (Jubb, 1993).

It is hypothesized that *Eurytrema coelomaticum* and *E. pancreaticum* might lead to pancreatic diseases as well as affect the mechanisms that the pancreas relies upon for digestion and metabolism (Surian *et al*., 2021). The disease is silent and results in losses in milk and meat production but does not show any clinical symptoms (Silva Júnior, 2017). Furthermore, people can contract this pancreatic trematode (Ogawa *et al*., 2019). In other study, the clinical signs were Progressive weight loss, poor physical health despite an abundance of high-quality fodder and mortality (Ilha *et al*., 2005; Pantiu Andrea *et al*., 2024).

Macroscopically, One of three different patterns of lesions was seen at necropsy: the pancreas was typical in size and color or it was tiny, shrunken, white, noticeably and widely hard (fibrosis) or it was black, slightly expanded and had a shriveled capsular surface. The genus *Eurytrema* had many leaf-shaped trematodes that were either hidden in the residual pancreatic parenchyma or packed inside several dilated ducts with thicker, white fibrous walls (Ilha *et al*., 2005; Pantiu Andrea *et al*., 2024). Microscopic observations revealed ductal hyperplasia, intralesional flukes, eggs and a large loss of pancreatic parenchyma with replacement fibrosis. Around the trematode eggs, there was an a granulomatous response along with varying degrees of inflammation (Ilha *et al*., 2005). This might to varied degrees result in abnormalities in the pancreas's secretory functions (Bassani *et al*., 2006; Rachid *et al*., 2011b; Pantiu Andrea *et al*., 2024).

The pancreas is often condemned during regular inspection when *E. coelomaticum* is discovered, which is typically treated as an unintentional discovery during necropsy or at slaughterhouses. Yet, herds of beef cattle have reported cases of bovine *Eurytrema*tosis linked to persistent emaci-

Table 1. Serum biochemical findings in different pancreatic diseases.

Diseases	Biochemical changes	References
Acute pancreatitis	-Increase in serum lipase and amylase levels. -Increases in triglyceride and cholesterol levels. -Liver enzymes and glucose levels were not affected. -Trypsin level was decreased.	(Hassan *et al.*, 1980)
	-Rises in the mean lipase (7.9-fold) and amylase (5.4-fold) activity levels. - Very little variations in the mean levels of calcium, albumin, and glucose. -BUN and creatinine tests were normal.	(Mohamed *et al.*, 2003).
Chronic pancreatitis	-Decreased blood levels of pancreatic-specific enzymes, including lipase, pancreatic iso-amylase, and trypsin.	(Schneider, 1986).
	-very low (LLN) pancreatic serum enzymes. -Low fecal elastase-1.	(Löhr, 2016).
Pancreatic fat necrosis	-normal levels of amylase and lipase.	(Tani *et al.*, 2017).
	- Decrease levels of total cholesterol, triglycerides and phospholipids. - Increased level of FFA.	(Oka *et al.*, 1988).
	-Lower blood cholesterol concentrations.	(Stuedemann *et al.*, 1985; Tharwat and Buczinski, 2012).
Pancreatic tumors	Increased total bilirubin (TBil) and carbohydrate antigen 19-9 (CA19-9).	(Zhang *et al.*, 2018)
	Raised serum levels of immunoreactive elastase (IRE) in 70% of individuals and carbohydrate antigen (CA 19-9) in 68%. Amylase and carcinoembryonic antigen (CEA) demonstrated increases of 30% and 28%, respectively.	(Hayakawa *et al.*, 1988).
Pancreatic lithiasis	Elevated lactate dehydrogenase activity, leukocytosis, neutrophilia, lymphopenia, hypoalbuminemia, hypergammaglobulinemia, and hyperglycemia. Lipase and amylase values were within normal ranges.	(Tharwat *et al.*, 2013)
Diabetes Mellitus	-Low insulin levels, high fructosamine, and hyperglycemia.	(Senturk *et al.*, 2003).
	Glucose intolerance, low plasma insulin concentration, hyperglycemia and glycosuria.	Baker.
	Mild neutrophilia and elevated levels of plasma AST, inorganic phosphorus, and glucose.	(Hasegawa *et al.*, 1999).
	Urine ketones and glucose levels significantly increased triglycerides, blood sugar, cholesterol and b-hydroxybutyrate were significantly higher than normal.	(Nazifi *et al.*, 2004).
	Pancreatic amylase concentration decreases.	(Ben Abdeljlil *et al.*, 1965).
	Amylase, lipase and carboxypeptidase were either undetectable or significantly reduced.	(Bendayan *et al.*, 1982)
Pancreatic erythematosis	-Regenerative anemia, low hemoglobin and hematocrit concentration. -Alkaline phosphatase, aspartate aminotransferase, gamma-glutamyl transferase, total bilirubin, albumin, and total protein levels were all within acceptable limits. -Slight neutrophilia and a high plasma amylase. -Blood smears stained with Giemsa's stain showed no signs of rickettsiae or blood protozoa. - No anomalies were found in the urine examination. -Few eggs may be present in feces.	(Ilha *et al.*, 2005).

ation (Bassani *et al*., 2006).

Laboratory findings, regenerative anemia, low hemoglobin and hematocrit concentration. Alkaline phosphatase, aspartate aminotransferase, gamma-glutamyl transferase, total bilirubin, albumin, and total protein levels were all within acceptable limits, despite slight neutrophilia and a high plasma amylase. Blood smears stained with Giemsa's stain showed no signs of rickettsiae or blood protozoa. No anomalies were found in the urine examination. When the feces of the afflicted cattle were examined, only a few *Eurytrema* sp. eggs were found in the majority of the instances (Ilha *et al*., 2005).

Animals with severe or moderate parasitemia did not have pancreatic digesting enzymes in their stools. Furthermore, increased lipase and serum amylase levels with a light load, further demonstrated that the disease is affecting pancreatic functions of the exocrine gland. Currently, the necropsy of the animal or post-mortem investigations in slaughterhouses are the only ways to determine the definitive diagnosis (Surian *et al*., 2021).

Pancreatic disease and Diabetes Mellitus

Diabetes Mellitus (DM) results from a dysfunctional regulation of carbohydrate metabolism. Adenocarcinoma, chronic pancreatitis and islet degeneration are linked to pancreatic destruction, which results in DM. It is an uncommon disease that affects cattle more than small animals and is characterized by low insulin levels, high fructosamine, and hyperglycemia (Senturk *et al*., 2003). There are two types of DM, insulin-dependent DM (IDDM) and non-insulin dependent. The most common one to occur in animals is (IDDM). Humans with insulin-dependent diabetic mellitus (IDDM) are thought to have an autoimmune disease caused by a breakdown in self-immunotolerance caused by a variety of environmental factors, such as viruses and other chemicals (Taniyama *et al*., 1995).

Although the definitive cause of DM in cattle is unknown. The pancreatic islets included more alpha cells than beta cells, according to immunohistochemical examination (Ono *et al*., 1989). These results raised the possibility that DM may be virus-induced or immune-mediated. Young calves in Japan have been observed to have autoimmune-mediated DM linked to Bovine Viral Diarrhea (BVD) virus infection, DM in Japanese Black cattle was caused by a chronic BVD viral infection (Taniyama *et al*., 1993). Diseases like fatty liver, fat cow syndrome, parturition and chronic insulitis are known to cause DM in cattle (Kitchen and Roussel Jr, 1990).

Different viral types have been known to be IDDM inducers. Two potential pathways of virus-induced IDDM have been suggested: The first one is that the virus destroys β cells of the pancreas, which lowers insulin output (Notkins, 1977; Yoon *et al*., 1979).The second pathway is that the animal body produces an immune response against the viral infection but this immune response fits the body tissue (Rayfield *et al*., 1986; Onodera *et al*., 1990). No research indicates that the incidence of IDDM in cattle is significantly influenced by genetic background (Tajima *et al*., 1999).

Pancreatic atrophy, abdominal adiponecrosis and hepatic lipidosis were all discovered during necropsy of cattle with DM. Histopathological examination revealed insulitis with mononuclear cell infiltration and a reduction in the total number and size of pancreatic islets (Hasegawa *et al*., 1999).The pancreatic acinar tissue showed evidence of a fatty alteration and the organ's overall structure was normal. Islets' size and number were decreased (Nazifi *et al*., 2004).

The majority of ruminant diabetes reports typically included pathology and clinical results. The final diagnosis in these animals was made based on certain laboratory tests, including glucose intolerance, low plasma insulin concentration, hyperglycemia and glycosuria (Taniyama *et al*., 1993; Taniyama *et al*., 1995). Mild neutrophilia and elevated levels of plasma AST, inorganic phosphorus, and glucose (Hasegawa *et al*., 1999).

Urine ketones and glucose levels significantly increased, according to biochemical tests. The levels of triglycerides, blood sugar, cholesterol and b-hydroxybutyrate were significantly higher than normal (Nazifi *et al*., 2004). It is generally known that insulin is essential for the synthesis of amylase, and biochemical investigations have demonstrated that pancreatic amylase concentration decreases in alloxan diabetic rats (Ben Abdeljlil *et al*., 1965).

Normal bovine pancreatic acinar cells contain seven secretory proteins (chymotrypsinogen A, trypsinogen, carboxypeptidase A, RNase, DNase, a-amylase, and lipase); however, in diabetic animals, amylase, lipase and carboxypeptidase were either undetectable or significantly reduced. The reduction in the three pancreatic enzymes in these diabetic animals could be connected to the endocrine tissue breakdown of the pancreas (Bendayan *et al*., 1982). The most important signs are persistent hyperglycemia and glucose intolerance (Bendayan *et al*., 1982).

Biochemical changes in pancreatic diseases in cattle

The biochemical components in the blood are important to maintain the proper physiology of the animal body. Consequently, like other organs, the pancreas depends on optimal blood biochemical components for its function. Serum enzyme measurements are a crucial diagnostic tool for both human and veterinary clinical practice. The majority of enzymes that have diagnostic uses work inside the cells that synthesize them and are found in high concentrations, especially in certain tissues (Barson *et al*., 2019).

These tests include those that show insufficiency of the exocrine or endocrine glands and those that detect varying degrees of reduced secretory capability. Tests for pancreatic function are most frequently used to identify chronic pancreatitis (Chowdhury and Forsmark, 2003). A summary for the biochemical changes that associate pancreatic diseases are presented in Table 1.

Conclusion

There are many diseases affecting the pancreas of cattle. Lack of research on the pancreas especially in cattle, denotes the necessities for further studies, which provide the clue for neglected clinical findings of that organ and help in diagnosis and treatment.

Conflict of interest

The authors declare no conflict of interest.

References

Barson, R.K., Padder, S., Sayam, A.S.M., Rahman, M.M., Bhuiyan, M.M.U., Bhattacharjee, J., 2019. Serum glucose, urea nitrogen, cholesterol, and total proteins in crossbred repeat breeder and normally cyclic cows. J. Adv. Vet. Anim. Res. 6, 82-85. https://doi.org/10.5455/javar.2019.f316

Bassani, C.A., Sangioni, L.A., Saut, J.P.E., Headley, S.A., Yamamura, M.H., 2007. Euritrematose bovina. Semina: Ciências Agrárias 28, 299-315. https://doi.org/https://doi.org/10.5433/1679%E2%80%910359.2007v28n2p299

Bassani, C.A., Sangioni, L.A., Saut, J.P.E., Yamamura, M.H., Headley, S.A., 2006. Epidemiology of *Eurytrema*tosis (*Eurytrema* spp. Trematoda: Dicrocoeliidae) in slaughtered beef cattle from the central-west region of the State of Paraná, Brazil. Veterinary Parasitology 141, 356-361. https://doi.org/https://doi.org/10.1016/j.vetpar.2006.06.003

Ben Abdeljlil, A., Palla, J.C., Desnuelle, P., 1965. Effect of insulin on pancreatic amylase and chymotrypsinogen. Biochemical and Biophysical Research Communications 18, 71-75. https://doi.org/https://doi.org/10.1016/0006-291X(65)90884-3

Bendayan, M., Ito, S., Manocchio, I., 1982. Alterations of exocrine pancreatic enzymes in virus-induced diabetic cattle as revealed by immunohistochemistry. Diabetologia 23, 65-68. https://doi.org/https://doi.org/10.1007/BF00257734

Braun, U., Dürr, M., Diener, M., Ossent, P., Hammon, H., Blum, J.W., 2001. [Diabetes mellitus caused by pancreatitis in a bull]. Schweizer Archiv fur Tierheilkunde 143, 99-104. http://europepmc.org/abstract/MED/11234631

Brereton, M.F., Vergari, E., Zhang, Q., Clark, A., 2015. Alpha-, delta-and PP-cells: are they the architectural cornerstones of islet structure and co-ordination? Journal of Histochemistry & Cytochemistry 63, 575-591. https://doi.org/https://doi.org/10.1369/0022155415583535

Chowdhury, R., Forsmark, C., 2003. Pancreatic function testing. Alimentary Pharmacology and Therapeutics 17, 733-750. https://doi.org/https://doi.org/10.1046/j.1365-2036.2003.01495.x

Croom, W.J., Bull, L.S., Taylor, I.L., 1992. Regulation of Pancreatic Exocrine Secretion in Ruminants: A Review1,2,3. The Journal of Nutrition 122, 191-202. https://doi.org/https://doi.org/10.1093/jn/122.1.191

Doherty, M., Healy, A., Donnelly, W., 1998. Diabetes mellitus associated with lymphocytic pancreatitis in a cow. Vet. Rec. 142, 493. doi: 10.1136/vr.142.18.493.

Dorny, P., Batubara, A., Iskander, M., Pandey, V.S., 1996. Helminth infections of sheep in North Sumatra, Indonesia. Veterinary Parasitology 61, 353-358. https://doi.org/https://doi.org/10.1016/0304-4017(95)00826-8

Duncan, L.K., 2011. Prasse's Veterinary Laboratory Medicine. Clinical Pathology. 5th ed. Wiley-Blackwell.

Ettinger, S.J., Feldman, E.C., Côté, E., 2016. Textbook of Veterinary Internal Medicine-eBook: Textbook of Veterinary Internal Medicine-eBook. Elsevier health sciences.

Groom, S., 1994. Pancreolithiasis in a Holstein-Friesian cow. The Canadian Veterinary Journal 35, 244.

Guo, L., Yao, J., Cao, Y., 2021. Regulation of pancreatic exocrine in ruminants and the related mechanism: The signal transduction and more. Animal Nutrition 7, 1145-1151. https://doi.org/https://doi.org/10.1016/j.aninu.2021.09.004

Harmon, D.L., Swanson, K.C., 2020. Review: Nutritional regulation of intestinal starch and protein assimilation in ruminants. Animal 14, s17-s28. https://doi.org/10.1017/S1751731119003136

Hasegawa, T., Uchida, K., Yanase, J., Kitazaki, K., Uchino, Y., Nakamura, S., Sakimoto, H., 1999. A case of diabetes mellitus in Japanese Black cattle. Journal of Veterinary Medical Science 61, 965-966. https://doi.org/https://doi.org/10.1292/jvms.61.965

Hassan, M., Amer, A., El-Amrousi, S., Bayumi, A., 1980. Some studies on pancreatic function in cattle and buffaloes 2. Induced acute pancreatitis. Assiut Veterinary Medical Journal 7, 295-306. https://doi.org/https://doi.org/10.21608/avmj.1980.192953

Hayakawa, T., Kondo, T., Shibata, T., Hamano, H., Kitagawa, M., Sakai, Y., Ono, H., 1988. Sensitive serum markers for detecting pancreatic cancer. Cancer 61, 1827-1831. https://doi.org/https://doi.org/10.1002/1097-0142(19880501)61:9%3C1827::AID-CNCR2820610918%3E3.0.CO;2-8

Hayes, H., Sass, B., 1987. Testis neoplasia in captive wildlife mammals: comparative aspects and review. The Journal of Zoo Animal Medicine 18, 162-165. https://doi.org/https://doi.org/10.2307/20094835

Herzog, K., Burgdorf, W., Hewicker-Trautwein, M., 2010. Mobile Encapsulated Bodies Comprising Fat Necrosis and Fibrous Tissue in the Abdominal Cavity of Cows. Journal of Comparative Pathology 143, 309-312. https://doi.org/https://doi.org/10.1016/j.jcpa.2010.04.002

Ilha, M.R.S., Loretti, A.P., Reis, A.C.F., 2005. Wasting and mortality in beef cattle parasitized by *Eurytrema coelomaticum* in the State of Paraná, southern Brazil. Veterinary Parasitology 133, 49-60. https://doi.org/https://doi.org/10.1016/j.vetpar.2005.02.013

Jenstad, M., Chaudhry, F.A., 2013. The amino acid transporters of the glutamate/GABA-glutamine cycle and their impact on insulin and glucagon secretion. Frontiers in Endocrinology 4, 199. https://doi.org/https://doi.org/10.3389/fendo.2013.00199

Jia, J., Wu, Q., Kou, J., Yang, M., 2018. Relationship between fatty liver and pancreatitis. International Journal of Clinical Medicine 9, 243-248. https://doi.org/https://doi.org/10.4236/ijcm.2018.94021

Jones, T., Hunt, R., King, N., 1997. The digestive system: peritoneum. Veterinary Pathology. 6th ed. Baltimore, MD: Williams & Wilkins 1087-1089.

Jubb, K.V., 1993. The pancreas. Pathology of Domestic Animals 2, 407-424.

Jubb, K.V.F., Kennedy, P.C., Palmer, N., 2012. Pathology of Domestic Animals. Academic press.

Katamoto, H., Aoki, M., Shimada, Y., Hakogi, E., 1996. Lipoprotein lipase activity of post-heparin-plasma in Japanese Black cattle affected with fat necrosis. British Veterinary Journal 152, 339-345. https://doi.org/https://doi.org/10.1016/S0007-1935(96)80106-0

Kelley, L., Harmon, B., McCaskey, P., 1996. A retrospective study of pancreatic tumors in slaughter cattle. Veterinary Pathology 33, 398-406. https://doi.org/https://doi.org/10.1177/030098589603300405

Kitchen, D. L., Roussel Jr, A.J., 1990. Type-I diabetes mellitus in a bull. Journal of the American Veterinary Medical Association 197, 761-763.

Lee, S.-J., Cho, H.-S., Noh, S., Kim, Y.H., Seo, H.-W., Oh, Y., 2023. A Postmortem Case Study—An Analysis of microRNA Patterns in a Korean Native Male Calf (Bos taurus coreanae) That Died of Fat Necrosis. Animals 13, 2149. https://www.mdpi.com/2076-2615/13/13/2149

Löhr, J.-M., 2016. Clinical and laboratory diagnosis of chronic pancreatitis. Pancreapedia: The Exocrine Pancreas Knowledge Base. https://doi.org/10.3998/panc.2016.14

Longinelli, A., Verine, H.J., 1977. Isotopic composition of cattle pancreatic stones: biological and geochemical implications. Experientia 33, 1419-1420. https://doi.org/10.1007/bf01918778

López, A., McKenna, S., 2018. Bovine pancreatolithiasis: case report and review of the literature. Journal of Veterinary Diagnostic Investigation 30, 760-762. https://doi.org/https://doi.org/10.1177/1040638718780190

Mattos Junior, D.G., 1987. *Eurytrema coelomaticum* (Giard et Billet, 1892) Looss, 1907: alguns aspectos do seu relacionamento com os hospedeiros intermediários e definitivo sob condições experimentais. Universidade Federal Rural do Rio de Janeiro. https://doi.org/https://rima.ufrrj.br/jspui/handle/20.500.14407/11772

Milastnaia, A., Dukhnitsky, V., 2019. Clinical and pathogenetic changes in dogs with chronic pancreatitis. Scientific Messenger of LNU of Veterinary Medicine and Biotechnologies. Series: Veterinary Sciences 21, 174-178. https://doi.org/https://doi.org/10.32718/nvlvet9432

Mohamed, T., Sato, H., Kurosawa, T., Oikawa, S., Nitanai, A., 2003. Ultrasonographic Imaging of Experimentally Induced Pancreatitis in Cattle. The Veterinary Journal 165, 314-324. https://doi.org/https://doi.org/10.1016/S1090-0233(02)00179-X

Motoi, Y., Kinno, S., Minamino, K., Shimbayashi, K., Ushimi, C., 1984. Treatment and Clinicobiochemical Observations of Cows Affected with Fat Necrosis. The Japanese Journal of Veterinary Science 46, 281-289. https://doi.org/10.1292/jvms1939.46.281

Moullé, V. S., Parnet, P., 2019. Effects of Nutrient Intake during Pregnancy and Lactation on the Endocrine Pancreas of the Offspring. Nutrients 11, 2708. https://www.mdpi.com/2072-6643/11/11/2708

Nazifi, S., Karimi, T., Ghasrodashti, A. R., 2004. Diabetes mellitus and fatty liver in a cow: case report. Comparative Clinical Pathology 13, 82-85. https://doi.org/https://doi.org/10.1007/s00580-004-0519-1

Niebergall-Roth, E., Teyssen, S., Singer, M. V., 1997. Pancreatic exocrine studies in intact animals: historic and current methods. Comparative Medicine 47, 606-616.

Notkins, A., 1977. Virus-induced diabetes mellitus. Archives of Virology 54, 1-17. https://doi.org/https://doi.org/10.1007/BF01314374

Nourani, H., Dehkordi, A. J., 2017. Pathological findings of severe pancreatolithiasis in a cow. Journal of the Hellenic Veterinary Medical Society 68, 487-490. https://doi.org/https://doi.org/10.12681/jhvms.15545

Oerly, A., Johnson, M., Soule, J., 2022. Economic, social, and environmental impacts of cattle on grazing land ecosystems. Rangelands 44, 148-156. https://doi.org/https://doi.org/10.1016/j.rala.2021.12.003

Ogawa, H., Takehara, Y., Naganawa, S., Yamaguchi, J., Nakaguro, M., 2019. A case of human pancreatic eurytremiasis. Abdominal Radiology 44, 1213-1216. https://doi.org/https://doi.org/10.1007/s00261-019-01925-4

Oka, A., Iwamoto, E., Tatsuda, K., 2015. Effects of clay on fat necrosis and carcass characteristics in J apanese B lack steers. Animal Science Journal 86, 878-883. https://doi.org/https://doi.org/10.1111/asj.12367

Oka, A., Yamasaki, T., Shibatani, M., Suzuki, T., Saito, T., 1988. Efficacy of isoprothiolane for the treatment of fat necrosis in cattle. British Veterinary Journal 144, 507-514. https://doi.org/https://doi.org/10.1016/0007-1935(88)90091-7

Ono, K., Hasegawa, T., Hasegawa, A., Tomoda, I., 1989. The Cellular Composition in the Pancreatic Islet of a Cow with Spontaneous Diabetes Mellitus. The Japanese Journal of Veterinary Science 51, 1067-1069. https://doi.org/10.1292/jvms1939.51.1067

Onodera, T., Taniguchi, T., Yoshihara, K., Shimizu, S., Sato, M., Hayashi, T., 1990. Reovirus type 2-induced diabetes in mice prevented by immunosuppression and thymic hormone. Diabetologia 33, 192-196. https://doi.org/https://doi.org/10.1007/BF00404795

Pantiu Andrea, J., Ruben, B., Leandro, O., Marcelo, S., Aguirre Laura, S., Agustín, A.-C., Micheloud Juan, F., 2024. *Eurytrema*tosis: An emerging parasitosis in cattle from Northeast Argentina. Veterinary Parasitology: Regional Studies and Reports 56, 101133. https://doi.org/https://doi.org/10.1016/j.vprsr.2024.101133

Rachid, M., Aquino Neto, H., Facury-Filho, E., Carvalho, A., Valle, G., Vasconcelos, A., 2011a. Chronic interstitial pancreatitis and chronic wasting disease caused by *Eurytrema coelomaticum* in Nelore cow. Arquivo Brasileiro de Medicina Veterinária e Zootecnia 63, 741-743. https://doi.org/https://doi.org/10.1590/S0102-09352011000300028

Rachid, M., Aquino Neto, H., Facury-Filho, E., Carvalho, A., Valle, G., Vasconcelos, A., 2011b. Pancreatite crônica intersticial e caquexia causada por *Eurytrema coelomaticum* em vaca Nelore. Arquivo Brasileiro de Medicina Veterinária e Zootecnia 63, 741-743. https://doi.org/https://doi.org/10.1590/S0102-09352011000300028

Radostits, O. M., Gay, C. C., Hinchcliff, K. W., Constable, P. D., 2006. Veterinary Medicine E-Book: Veterinary Medicine E-Book. Elsevier Health Sciences.

Rayfield, E. J., Kelly, K. J., Yoon, J.-W., 1986. Rubella Virus–Induced Diabetes in the Hamster. Diabetes 35, 1278-1281. https://doi.org/10.2337/diab.35.11.1278

Saisho, Y., Butler, A., Meier, J., Monchamp, T., Allen-Auerbach, M., Rizza, R., Butler, P. C., 2007. Pancreas volumes in humans from birth to age one hundred taking into account sex, obesity, and presence of type-2 diabetes. Clinical Anatomy 20, 933-942. https://doi.org/https://doi.org/10.1002/ca.20543

Schneider, M., 1986. Serum enzymes in diagnosis of chronic pancreatitis. In: Diagnostic procedures in pancreatic disease. pp. 247-258. Springer.

Senturk, S., Kennerman, E., Golcu, E., Temizel, M., 2003. Diabetes mellitus in a cow. Indian Vet. J. 80, 809-810.

Shimada, Y., Katamoto, H., Ishida, S., Kobayashi, K., Tohzyoh, H., 1988. Therapeutic Effect of Isoprothiolane on Bovine Fat Necrosis. The Japanese Journal of Veterinary Science 50, 1017-1024. https://doi.org/10.1292/jvms1939.50.1017

Silva Júnior, F. A., 2017. Ocorrência e caracterização morfológica de *Eurytrema coelomaticum* (Giard & Billet, 1892)(Digenea, Dicrocoeliidae) em rebanhos bovinos provenientes da região sul de Minas Gerais, Brasil

Soulsby, E. J. L., 1982. Helminths, arthropods and protozoa of domesticated animals. London: Baillière Tindall, 809 pp., illus. ISBN 0-7020-0820-6.

Stuedemann, J., Rumsey, T., Bond, J., Wilkinson, S., Bush, L., Williams, D., Caudle, A., 1985. Association of blood cholesterol with occurrence of fat necrosis in cows and tall fescue summer toxicosis in steers. American Journal of Veterinary Research 46, 1990-1995. https://doi.org/https://doi.org/10.2460/ajvr.1985.46.09.1990

Surian, C.R.S., Surian, S.R.S., Carneiro, C., Perosa, F.F., Horn, V.W., Fronza, N., Bonassi, D.E., Peripolli, V., Santarosa, B.P., Gomes, T.M.A., 2021. *Eurytrema coelomaticum* infection: correlation between parasite burden and impairment of pancreatic exocrine enzyme secretion. Ciência Rural 52, e20210041. https://doi.org/https://doi.org/10.1590/0103-8478cr20210041

Tajima, M., Yuasa, M., Kawanabe, M., Taniyama, H., Yamato, O., Maede, Y., 1999. Possible causes of diabetes mellitus in cattle infected with bovine viral diarrhoea virus. Journal of Veterinary Medicine, Series B 46, 207-216. https://doi.org/https://doi.org/10.1046/j.1439-0450.1999.00230.x

Tani, C., Pratakpiriya, W., Tani, M., Yamauchi, T., Hirai, T., Yamaguchi, R., Ano, H., Katamoto, H., 2017. Histopathological changes in the pancreas of cattle with abdominal fat necrosis. Journal of Veterinary Medical Science 79, 52-59. https://doi.org/https://doi.org/10.1292/jvms.16-0282

Taniyama, H., Shirakawa, T., Furuoka, H., Osame, S., Kitamura, N., Miyazawa, K., 1993. Spontaneous diabetes mellitus in young cattle: histologic, immunohistochemical, and electron microscopic studies of the islets of Langerhans. Veterinary Pathology 30, 46-54. https://doi.org/https://doi.org/10.1177/030098589303000106

Taniyama, H., Ushiki, T., Tajima, M., Kurosawa, T., Kitamura, N., Takahashi, K., Matsukawa, K., Itakura, C., 1995. Spontaneous diabetes mellitus associated with persistent bovine viral diarrhea (BVD) virus infection in young cattle. Veterinary Pathology 32, 221-229. https://doi.org/https://doi.org/10.1177/030098589503200302

Tharwat, M., Abdelaal, A., Selim, H., Floeck, M., 2013. Ultrasonographic identification of pancreolithiasis in a Holstein cow: A case report. Wiener tierärztliche Monatsschrift, 100. https://doi.org/https://www.researchgate.net/publication/261376505_Ultrasonographic_identification_of_pancreolithiasis_in_a_Holstein_cow_A_case_report

Tharwat, M., Buczinski, S., 2012. Diagnostic ultrasonography in cattle with abdominal fat necrosis. The Canadian Veterinary Journal 53, 41.

Tokarnia, C., 1961. Islet cell tumor of the bovine pancreas.

Travassos, L., 1944. Revisao da familia Dicrocoeliidae Odhner, 1910. In Revisão da família Dicrocoeliidae Odhner, 1910. pp. 124-124.

Veiling, K., 1975. Bovine Pancreolithiasis in Denmark. Acta Veterinaria Scandinavica 16, 327-340. https://doi.org/10.1186/BF03546651

Vianna, S., 1987. O *Eurytrema coelomaticum* (Trematoda: Dicrocoeliidae) no Brasil. Arquivos Fluminenses de Medicina Veterinaria, 2.

Wass, W., 1965. The duct systems of the bovine and porcine pancreas. Am. J. Vet. Res. 26, 267-272.

Yamaguti, S., 1975. A synoptical review of life histories of digenetic trematodes of vertebrates, with special reference to the morphology of their larval forms. Published by Yugaku -sha.

Yılmaz, A., 1995. Massive Fat Necrosis In A Cow. Ankara Üniversitesi Veteriner Fakültesi Dergisi, 42, 1. https://doi.org/https://doi.org/10.1501/Vetfak_0000000790

Yoon, J. W., Austin, M., Onodera, T., Notkins, A. L., 1979. Isolation of a virus from the pancreas of a child with diabetic ketoacidosis. The New England Journal of Medicine 300, 1173-1179. https://doi.org/10.1056/nejm197905243002102

Zhang, S., Huang, X., Tian, Y., Aimaiti, S., Zhang, J., Zhao, J., Chen, Y., Wang, C., 2018. Clinicopathologic characteristics, laboratory parameters, treatment protocols, and outcomes of pancreatic cancer: a retrospective cohort study of 1433 patients in China. PeerJ, 6, e4893. https://doi.org/https://doi.org/10.1097/mpa.0000000000000939