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BIOCHEMICAL AND HISTOPATHOLOGICAL CHANGES ASSOCIATED WITH RECORDED CASES OF CHRONIC PANCREATITIS AND PANCREATIC FAT NECROSIS IN CATTLE

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ABSTRACT

This study aimed to investigate the biochemical and pathological changes in chronic pancreatitis and pancreatic fat necrosis of cattle. The study included 32 cattle of different ages and sexes selected from animals slaughtered at El-Minia governorate slaughterhouses. Gross and histopathological examinations were used for the identification of pancreatic diseases. Among the 32 animals, 13 cases were identified as chronic pancreatitis, nine cases were diagnosed as pancreatic fat necrosis, and 10 cattle were clinically healthy and had no postmortem lesions represented the control group. Two blood samples were collected from each animal before slaughtering. Serum samples were used to estimate insulin, lipase, amylase, aspartate aminotransferase, gamma-glutamyl transferase (γGT), total proteins, albumin, globulin, creatinine, urea, and lipid profile. A plasma sample was used to estimate blood glucose levels. Pancreatic tissue specimens were subjected to histopathological examination. In chronic pancreatitis, there was a significant increase in serum amylase activity, creatinine level, and urea levels, while in pancreatic fat necrosis, there were significant increases in serum lipase activity, an increase in serum creatinine level, an increase in serum urea level and a decrease in serum γGT activity. It could be concluded that chronic pancreatitis and fat necrosis in cattle do not affect blood glucose and insulin levels. Serum urea and creatinine levels increase in both conditions. Serum amylase activity increases in chronic pancreatitis. However, serum lipase activity elevates in fat necrosis.

Keywords: Cattle Pancreas, Pancreatic disease, Chronic pancreatitis, fat necrosis, amylase, lipase.

INTRODUCTION

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). 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. Clinical

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diagnosis of chronic pancreatitis is difficult due to the typically mild or subclinical nature of the condition, and the fact that its clinical manifestations are nonspecific (Milastnaia and Dukhnitsky, 2019).

There is little research on 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 occur 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 spontaneous pancreatitis was discovered (Veiling, 1975; Groom, 1994; Doherty *et al.*, 1998; Braun *et al.*, 2001).

Pancreatic fat necrosis**,** 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 Japanese black (JB) cattle was higher than in other breeds, and 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, JB breeding cows that receive insufficient roughage and lack of exercise in their pens may develop abdominal fat necrosis (Shimada *et al.*, 1998).

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)

There is no indication that this disease is malignant, and the composition of the fatty deposits is similar to normal cow fat (Radostits *et al.*, 2007). Numerous clinical symptoms, including intestinal stenosis, urine retention, dystocia, and infertility are brought on by the disorder (Baker, 1983; 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 mesothelioma is necessary, because ultrasonography alone cannot guarantee an accurate diagnosis (Radostits *et al.*, 2007). The pathophysiology of bovine abdominal fat necrosis has not received much attention, with most reports focusing on pancreatic lesions (Tani *et al.*, 2017). In addition, there is a lack of research on the pancreas of cattle in Egypt. Consequently, this study aimed to investigate pancreatitis and pancreatic fat necrosis in cattle and associated histopathological and biochemical changes.

MATERIALS AND METHODS

Animals:

A total number of 32 cattle of both sexes and different ages were subjected to the study. Animals were selected from cattle presented at the Bani-Abaid slaughterhouse (Abu-Qurqas, Minya governorate, Egypt), and were included in the study based on inclusion criteria; cattle of different ages and sexes showed gross lesions on the pancreas, animals with any abnormal gross affections on any organ other than the pancreas were

excluded from the study. Data related to each animal was recorded in an examination sheet. Two blood samples were collected from each animal before slaughtering. Also, pancreatic tissue samples were collected after the animals had been slaughtered.

Samples

Blood samples

Two types of blood samples were collected from the jugular vein of each animal. The whole blood sample was collected in vacutainer tubes containing sodium fluoride as an anticoagulant to separate plasma for the determination of glucose level. The second blood sample was collected in plain tubes and processed for separation of serum. Serum samples were used for estimating insulin (Bovine INS ELISA kit, SunLong Biotech Co., Ltd.SL0018Bo), amylase (Spectrum, Egypt, cat.no ZL-219001) and lipase (Biomed, Egypt). serum AST, γGT, Tp, albumin and globulins levels (Spectrum, Egypt), serum levels of creatinine (Spectrum Diagnostics, Egypt, cat.no 234000), urea (Diamond Diagnostics, Egypt), and lipid profile (cholesterol, triglycerides HDL, LDL and VLDL (Biomed, Egypt). All biochemical parameters were measured using a Spectrophotometer (UV/VIS Spectrophotometer, Optizen 3220UV, Mecasys Co., Ltd. KOREA).

Pancreatic tissue samples:

Pancreatic specimens were fixed in 10% neutral buffered formalin. Then dehydration by ascending grades of alcohol, cleared by xylene, embedded in paraffin. Sectioning of the tissue with 4-5 µ and stained with hematoxylin and eosin stains (H&E) (Bancroft and Gamble, 2008).

Statistical analysis

Data was expressed as Mean ± SD, statistical analysis was conducted using SPSS 13.0 for Windows Discussion (SPSS, Chicago, USA). The differences in biochemical parameters were compared using one-way ANOVA, followed by the least significant difference (LSD) post-hoc analysis ($P<0.05$).

Ethical approval

All animals that were employed in this study were dealt with ethically by the Research Ethical Committee of the Faculty of Veterinary Medicine, Assiut University, Assiut, Egypt, which approved the study under No. 06/2024/0283.

RESULTS

Histopathological examination was the basis for the classification of pancreatic diseases. Among the 50 animals, 13 cases were classified as chronic pancreatitis, and nine cases were classified as pancreatic fat necrosis. In addition, 10 animals clinically healthy and free from any postmortem lesions, with a normal pancreas, were kept as a control. The remaining animals included some that were normal, while others had pancreatic diseases aside from chronic pancreatitis and pancreatic fat necrosis, and one was excluded due to liver condemnation. The lesions in most cases were localized and not distributed throughout the pancreas; that's why they may be the cause that masked the clinical signs.

Histopathological findings

Chronic pancreatitis:

Gross examination of the pancreas appeared pale in color, granular, and fibrosed (Fig. 1A). Histopathological examination of chronic pancreatitis cases revealed alteration in the blood vessels, acini, interstitium, and pancreatic ducts. There was severe congestion of blood vessels with swelling of endothelium, thinking, and hyperplasia of tunica media (vasculitis). The pancreatic acini showed degeneration, necrosis, and interstitial fibrosis with heavy infiltration, with chronic inflammatory cells as lymphocytes and macrophages. Metaplasia of the pancreatic duct with periductal fibrosis was also noticed (Fig. 1B-E).

Fat necrosis:

Gross examination of the pancreas with fat necrosis showed excessive multifocal whitish strips of infiltrative adipose tissues (Fig.2 A).

Histopathological examination of the pancreas with fat necrosis showed severe necrosis of the fat with the formation of fat-laden foam cells (Fig. 2B, C).

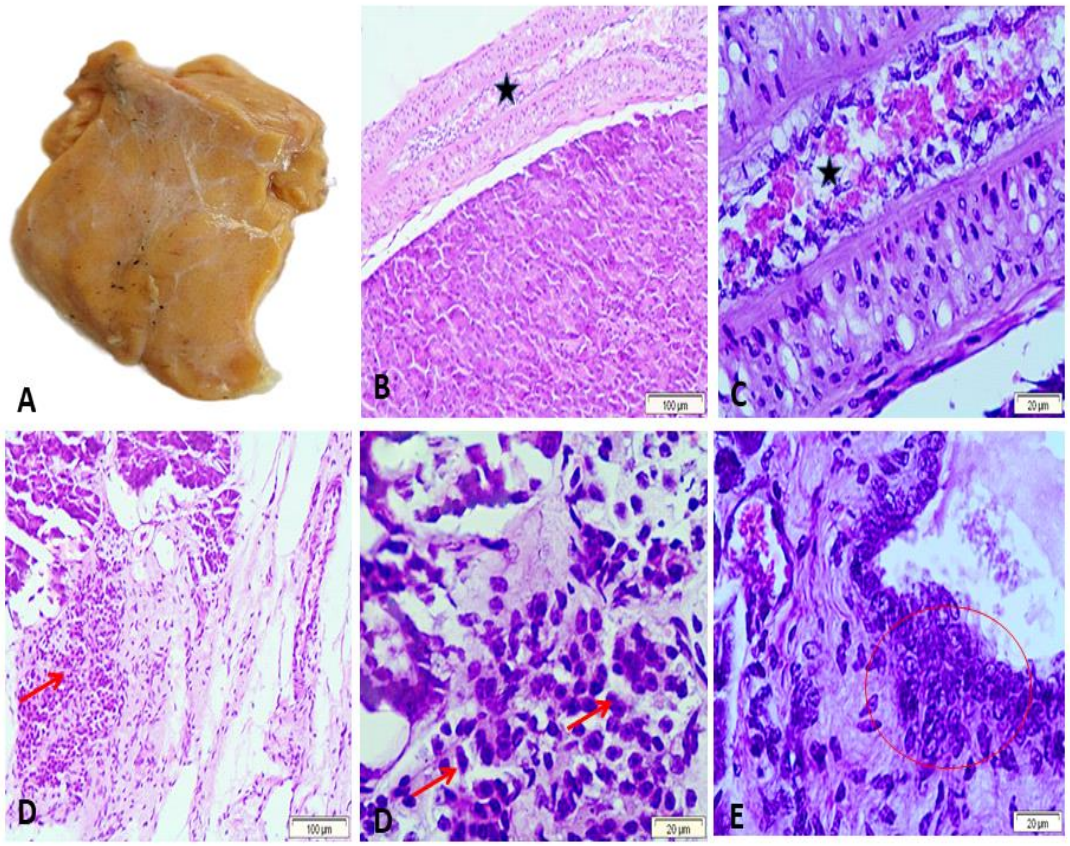

Fig.1: Macroscopical and microscopical findings in chronic pancreatitis. **A)** chronic pancreatitis that appeared pale and fibrosed. B-E) Representative micrograph of the pancreas with chronic pancreatitis stained by HE showing severe congestion of blood vessels with swelling of its endothelium (stars) and hyperplasia of tunica media, interstitial fibrosis with heavy infiltration with chronic inflammatory cells (red arrows) and metaplasia of pancreatic duct with periductal fibrosis (red circle).

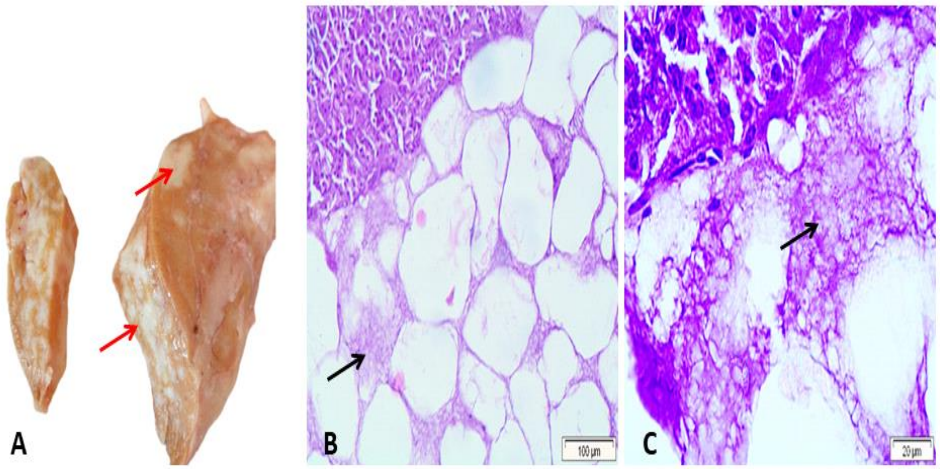

Fig. 2: Macroscopical and microscopical findings in pancreatic fat necrosis. A) Showing fat necrosis appeared in excessive multifocal whitish strips of infiltrative adipose tissues (red arrows). B, C) Representative micrograph of the pancreas with fat necrosis stained by HE stains showing severe fat necrosis with the formation of fat-laden foam cells (black arrows).

Biochemical findings:

In chronic pancreatitis, there were significant increases in serum amylase level (P=0.040), creatinine level (P=0.000), and urea level (P=0.000).

In the case of fat necrosis, there were significant increases in serum Lipase level (P=0.04), serum creatinine level (P=0.001), and serum urea level (P=0.007). However, serum γGT level showed a significant decrease (P=0.04).

Table 1: Biochemical findings in control and pancreatic diseases.

	Control	Chronic Pancreatitis	Pancreatic fat necrosis
Glucose(mg/dL)	91.73±26.49 [a]	81.66±22.15 [a]	84.47±21.93 [a]
Insulin (mU/L)	10.95±3.25 [a]	15.70±6.51 [a]	9.77±2.76 [a]
Lipase (U/l)	8.48±5.41 [a]	13.79±7.57 [ab]	14.23±2.99 [b]
Amylase (U/l)	41.66±18.07 [a]	70.49±48.06 [b]	61.28±13.55 [ab]
AST (U/l)	38.03±23.26 [a]	34.26±17.70 [a]	43.23±14.11 [a]
γGT (U/l)	11.64±3.84 [ab]	16.94±7.40 [a]	11.10±5.31 [b]
TP (g/dL)	7.21±0.35 [a]	7.32±0.60 [a]	7.51±1.23 [a]
Albumin (g/dL)	3.01±0.43 [a]	3.00±.77 [a]	3.30±0.68 [a]
Globulin (g/dL)	4.20±0.62 [a]	3.93±1.31 [a]	4.24±1.50 [a]
Creatinine (mg/dL)	1.09±0.17 [a]	1.88±0.38 [b]	1.97±0.62 [b]
Urea (mg/dL)	20.84±4.17 [a]	48.55±16.88 [bc]	62.44±32.96 [c]
Cholesterol (mg/dL)	143.53±46.32 [a]	138.79±43.57 [a]	123.16±19.83 [a]
TG (mg/dL)	15.27±12.74 [a]	24.03±13.46 [ab]	21.43±12.12 [ab]
HDL (mg/dL)	89.20±46.25 [a]	82.41±30.97 [a]	80.18±26.48 [a]
LDL (mg/dL)	51.27±46.65 [a]	51.57±40.94 [a]	38.69±17.93 [a]
VLDL (mg/dL)	3.05±2.55 [a]	4.82±2.69 [ab]	4.28±2.42 [ab]

Data are presented as Mean±SD. In each row, values followed by different superscripts ([a, b, c, ...]) are significant (P<0.05). AST: aspartate aminotransferase; γGT: Gamma-glutamyl transferase; TP: total protein. TG: triglycerides. HDL: High-density lipoproteins; LDL: Low-density lipoproteins; VLDL: Very low-density lipoproteins.

DISCUSSION

The diseases of the bovine pancreas include pancreatitis (Kholad *et al.*, 1981) and fatty pancreas. Other pancreatic diseases include pancreatic lithiasis (Kelley *et al.*, 1996), pancreatic fluke infestation (Florence, 1939), diabetes mellitus due to an infection with the foot-and-mouth disease virus (Jubb *et al.*, 2012), pancreatic atrophy associated with nutritional deficiencies (Jubb *et al.*, 2012). Dairy cows with obesity and abdominal fat necrosis had significant histological pancreatic abnormalities, including fatty pancreas and pancreatitis (Tani *et al.*, 2017).

The clinical signs of pancreatic diseases are nonspecific. They may be atypical, as most clinical signs resemble those of digestive disturbances and largely vary from one animal to another, which makes the clinical diagnosis of pancreatic diseases difficult. The most common signs include severe abdominal pain, weight loss, jaundice, and an inability to stand (Mair and Love, 2013; Lv *et al.*, 2021). The gold standard for diagnosing pancreatic diseases is a histo-pathological examination. Biochemical findings and clinical signs assist histo-pathology in diagnosing the disease (Webb and Trott, 2008).

In the present study, pathological examination of chronic pancreatitis revealed obvious gross and histopathological changes. The gross examination revealed a pale and fibrosed pancreas. Histopathologically, there were vasculitis, interstitial fibrosis with heavy infiltration of lymphocytes, and metaplasia of the pancreatic duct with periductal fibrosis. The obtained findings agree with those reported in dogs and cats (Xenoulis *et al.*, 2008; Washabau and Day, 2012), and in humans (Iglesias-García *et al.*, 2006).

The primary mediators of fibrosis are pancreatic stellate cells, which cause the extracellular matrix to develop in interstitial spaces and regions where duct cells are damaged or acinar cells are absent. This process eventually results in substantial alterations to the arrangement and makeup of the islets, abnormal distortion of the major ducts, and a gradual loss of the pancreatic lobular morphology and structure. Malnutrition and/or diabetes finally result from the irreversible fibrotic deterioration of the pancreatic gland and the morphological and structural alterations that impede exocrine and endocrine activities (Brock *et al.*, 2013).

In fat necrosis cases, the pancreas macroscopically showed multifocal whitish streaks of infiltrative adipose tissue, and microscopically showed fat necrosis with the formation of fat-laden foam cells. These findings are consistent with those reported in cattle by (Tani *et al.*, 2017) and in humans (Paul and Shihaz, 2020; Mahyoub *et al.*, 2023). The researchers suggested that acute pancreatitis may trigger necrotic fat accumulation in the pancreas and inflammation due to the leakage of active enzymes. The main pathological findings were fat necrosis (saponification) and adipocyte infiltration. Macroscopically the pancreas was hard, enlarged, pale, and showed excessive multifocal whitish strips of infiltrative adipose tissue. While under the microscope, there was formation of fat-laden foam cells, severe fat necrosis, and infiltration of inflammatory cells, especially macrophages and lymphocytes. The body tends to redistribute the fat into other tissues other than the adipose tissue, like the liver and pancreas, resulting in inflammation of the pancreas either acute or chronic. The enzymes leak from the inflamed pancreas, leading to the autodigestion of fat and making fat necrosis, followed by fibrosis.

The significant increase in serum amylase activity in chronic pancreatitis agrees with those reported by (Nothman and Callow, 1971; Murtaugh and Jacobs, 1985) in dogs. Increased serum amylase activity could result from production occurring in areas aside from the pancreas, such as the small intestine and liver, indicating that higher levels may be related to non-pancreatic production. Additionally, elevated amylase activity might be a result of reduced amylase clearance by the kidneys due to kidney disease, as renal failure or the formation of macroamylesaemia can raise amylase enzyme levels without the presence of pancreatitis (Matull *et al.,* 2006). In contrast, Oh *et al.* (2017) noted that in chronic pancreatitis, serum amylase activity was below the normal limit, as the secretory capacity of the pancreas declined during chronic pancreatitis. The high level of creatinine and urea in the study may be attributed to renal disease. More research is needed to investigate the relationship between chronic pancreatitis and kidneys.

Insulin and glucose levels in this study showed non-significant changes in cases of chronic pancreatitis and fat necrosis. This result agrees with (Ciccarelli *et al.*, 2024) in humans. In the absence of diabetes mellitus, chronic pancreatitis may not affect β-cells that produce insulin, especially in the first stage of chronic pancreatitis, so the level of glucose is in the normal range (Lundberg *et al.*, 2016; Ciccarelli *et al.*, 2024). In contrast, Sasikala *et al.* (2012) reported that in the early stages of CP, β-cell dysfunction arises, although clinical diabetes appears later when fibrosis is severe. Several compensatory or disease-related variables

that preserve a transient equilibrium despite the pancreatic damage can result in normal insulin and glucose levels in the pancreatic necrosis setting. The remaining intact beta cells can release enough insulin to keep glucose levels within the normal range if the necrosis is localized and does not cause severe damage to the islets of Langerhans. Blood glucose levels can be stabilized during stressful times, with the support of adequate hepatic glucose synthesis and storage. The increase in serum lipase activity in fat necrosis agrees with the findings of (Hameed *et al.*, 2015; Pirahanchi and Sharma, 2019) in humans. Fat necrosis is considered a specific marker for the first stage of pancreatic inflammation (Tani *et al.*, 2017; Mahyoub *et al.*, 2023), which is associated with high serum lipase activity. It may also be related to a decreased clearance rate from the kidney due to renal disease (Hameed *et al.*, 2015).

The increase in serum urea and creatinine levels in fat necrosis agrees with the findings of (Lankisch *et al.*, 2010; Nassar and Qunibi, 2019) in humans and (Dulude *et al.*, 2024) in cats. The increased levels can be explained by pancreatic fat necrosis that occurs after acute pancreatitis and acute kidney injury may result from hypovolemia, systemic inflammation, or direct kidney damage from circulating pancreatic enzymes and cytokines in pancreatitis leads to a rise in urea levels and creatinine, as a result of decreased excretion due to impaired renal function (Nassar and Qunibi, 2019; Dulude *et al.*, 2024) and increased levels of creatinine are considered a specific marker in pancreatic necrosis (Lankisch *et al.*, 2010). The release of active proteases from the pancreatic acinar cells may result in an increase in urea levels due to increased trypsinogen, which promotes protein digestion. The waste product of digesting proteins is urea. Therefore, in certain pancreatic conditions, its level may be elevated (Kaphalia, 2019).

As the pancreas produces γGT along with the liver and kidney (Chiyanika *et al.*, 2024), the decreased activity of γGT in fat necrosis may be attributed to pancreatic diseases in which fat accumulates in the cells that produce γGT and affect the production of γGT.

CONCLUSION

Chronic pancreatitis and fat necrosis in cattle do not affect blood glucose and insulin levels. Serum urea and creatinine levels increase in both conditions. Serum amylase activity increases in chronic pancreatitis. However, serum lipase activity elevates in fat necrosis.

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التغيرات البيوكيميائية والنسيجية المرضية المرتبطة بالحالات المسجلة من التهاب البنكرياس المزمن ونخر الدهون البنكرياسية في الأبقار

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هدفت هذه الدراسة إلى دراسة التغيرات البيوكيميائية والمرضية في التهاب البنكرياس المزمن ونخر دهون البنكرياس لدى الأبقار. شملت الدراسة ٣٢ رأسًا من الماشية من مختلف الأعمار والجنس، اختيرت من حيوانات مذبوحة في مسلخ محافظة المنيا. واستُخدمت الفحوصات العينية والهيستوباثولوجية لتحديد أمراض البنكرياس. من بين ٣٢ رأسًا، تم تشخيص ١٣ حالة على أنها التهاب بنكرياس مزمن، وتسع حالات على أنها نخر دهون البنكرياس، و ١٠ أبقار سليمة ولا تعاني من أي آفات ما بعد الذبح، ومثلت المجموعة الضابطة. تم جمع عينتي دم من كل رأس قبل الذبح. استُخدمت عينات مصل الدم لتقدير مستويات الأنسولين، والليباز، والأميليز، وأسبارتات أمينوترانسفيراز، وجاما-غلوتاميل ترانسفيراز (γGT)، والبروتينات الكلية، والألبومين، والغلوبيولين، والكرياتينين، واليوريا، ومستوى الدهون. كما استُخدمت عينة بلازما لتقدير مستويات سكر الدم. وخضعت عينات أنسجة البنكرياس للفحص النسيجي المرضي. في حالات التهاب البنكرياس المزمن، لوحظت زيادة ملحوظة في نشاط الأميليز، ومستوى الكرياتينين، ومستويات اليوريا في المصل، بينما في حالات نخر دهون البنكرياس، لوحظت زيادات ملحوظة في نشاط الليباز، وزيادة في مستوى الكرياتينين، وزيادة في مستوى اليوريا، وانخفاض في نشاط γGT في المصل. ويمكن الاستنتاج أن التهاب البنكرياس المزمن ونخر الدهون في الماشية لا يؤثران على مستويات سكر الدم والأنسولين. ترتفع مستويات اليوريا والكرياتينين في المصل في كلتا الحالتين. يزداد نشاط الأميليز في المصل في التهاب البنكرياس المزمن. ومع ذلك، يرتفع نشاط الليباز في المصل في نخر الدهون.