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# Pancreatitis-induced prediabetes and hepatic lipidosis in a mixed breed cat

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**ABSTRACT:** Pancreatitis can lead to decreased insulin production and elevated blood glucose levels. Reduced insulin levels can cause excessive lipolysis, leading to hepatic lipidosis. This report aimed to illustrate how pancreatitis can result in predi-abetes and hepatic lipidosis, providing insight into selecting appropriate and causal therapies. This case report discusses a 5-year-old male mixed-breed cat weighing 3.9 kg presented with anorexia and hypersalivation. Despite a routine physical examination, blood tests revealed decreased creatinine and Ca2+ levels and elevated blood glucose, SGPT, cho-lesterol, and amylase levels. These findings suggested a diagnosis of pancreatitis accompanied by prediabetes and hepat-ic lipidosis. The treatment administered included intravenous Ringer's Lactate, subcutaneous Ornipural® injection, oral cephalexin (Rilexine®) and Samylin®, and dietary management with specialized renal wet food.

**Keywords:**
hepatic lipidosis, mixed breed cat, pancreatitis, prediabetes

## ■ INTRODUCTION

Pancreatitis is an inflammation of the pancreas with a prevalence of 67% among 115 cats (Forman *et al*. 2021). Blood chemistry is a diagnostic tool for pancreatitis that focuses on elevated amylase, lipase, cholesterol, and glucose activities. The clinical symptoms of pancreatitis in cats are non-specific, but anorexia, lethargy, and hypersalivation are often observed (Bazelle *et al*. 2014). There are no specific risk factors for pancreatitis; it can occur at any age, sex, or breed. The frequency of concurrent diseases in cats with acute pancreatitis is up to 92%, including hepatic lipidosis and diabetes. Pancreatitis reduces insulin levels, leading to glucose accumulation in the blood. The body uses fat and proteins as energy sources to compensate for this problem. However, continuous lipolysis can accumulate free fatty acids in the liver, causing hepatic lipidosis and increasing liver enzyme activity and blood glucose levels (Armstrong *et al*. 2012).

Currently, publishing such cases is difficult; therefore, sharing this information with veterinary medical professionals is crucial for better diagnosis and treatment strategies. This report aimed to illustrate how pancreatitis can result in prediabetes and hepatic lipidosis, providing insight into selecting appropriate and causal therapies.

## ■ CASES

**Signalment**: The patient is a 5-year-old male mixed breed cat weighing 3.9 kg. **History**: According to the owner, the cat has been refusing to eat and experiencing hypersalivation. The medical history of the cat is unknown. **Physical Examination**: The physical examination revealed dehydration and hypersalivation, while the temperature was normal at 39°C. **Diagnostic Laboratory**: Blood biochemistry. **Diagnosis**: Pancreatitis. **Treatment**: Administration of intravenous Ringer's Lactate, subcutaneous Ornipural® injection, oral cephalexin (Rilexine®) and Samylin®, and dietary management with specialized renal wet food.

## ■ RESULT AND DISCUSSION

Hypersalivation indicates nausea experienced by cats. Nausea can occur because of disorders in the digestive organs, particularly the liver and pancreas, leading to the need for blood chemistry analysis. This ancillary examination was limited to blood chemistry, which revealed decreased creatinine and Ca2+ levels and increased blood glucose, SGPT, cholesterol, and amylase levels (Table 1).

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Table 1. Blood chemistry examination in a mixed breed cat.

| Parameter | Results | Normal Value |
|---|---|---|
| **Kidney** | | |
| Blood Urea Nitrogen (mg/dL) | 18.9 | 14 - 36 |
| Creatinine (mg/dL) | 0.38 | 0.6 - 2.4 |
| Blood Glucose (mg/dL) | 248 | 74 - 114 |
| **Liver** | | |
| SGPT (U/L) | 106 | 10 - 100 |
| Alkali phosphatase (U/L) | 28 | 6 - 102 |
| Cholesterol (mg/dL) | 317 | 75 - 220 |
| Total protein (g/dL) | 6.3 | 5.2 - 8.8 |
| Albumin (g/dL) | 3.2 | 2.5 - 3.9 |
| Total bilirubin (mg/dL) | 0.13 | 0.1 - 0.4 |
| Creatinine kinase (u/L) | 224 | 56 - 529 |
| Ca2+ (mg/dL) | 6 | 8.2 - 10.8 |
| Phosphor (p) | 4.3 | 2.4 - 8.2 |
| Amylase (u/L) | 2,737 | 100 - 1,200 |
| Globulin (g/dL) | 3.1 | 2,3 - 5.3 |
| Amylase : Globulin Ratio | 1.03 | 0.45 - 1.2 |

Note: SGPT= Serum Glutamic Pyruvic Transaminase, Red=increase, Blue=decrease.

Based on the physical and laboratory examination results, pancreatitis was suspected in the cat. Pancreatitis can cause enzymes to leak into the bloodstream, leading to elevated amylase levels. It also causes insulin levels to decrease due to pancreatic cell damage. Reduced insulin levels result in glucose accumulation in the blood because less glucose enters the cells. Blood chemistry results showed prediabetes symptoms, with a blood glucose level of 248 mg/dL. Prediabetes is indicated by fasting blood glucose levels of 100–126 mg/dL or 140–200 mg/dL post-meal (Kodali & Ali, 2016). Pancreatic cell damage can also activate Toll-like Receptors (TLRs) as an inflammatory response, reducing cholesterol efflux and causing cholesterol to accumulate in the blood. TLR explains the elevated cholesterol levels observed in the blood chemistry results (Hong *et al*. 2020).

The lack of glucose in cells causes the body to create new energy sources from the breakdown of fats (lipolysis) and proteins. Lipolysis produces free fatty acids, which then circulate in the blood and are stored in the liver (Dilworth *et al*. 2021). However, excess free fatty acids can lead to hepatic lipidosis (fatty liver disease). This condition can impair hepatocyte function and cause liver inflammation, leading to leakage of liver enzymes into the bloodstream. Consequently, there was an increase in SGPT levels in the blood chemistry results. Hypocalcemia in blood chemistry results can occur due to the binding of free fatty acids with calcium, forming insoluble soaps in the blood, resulting in the excretion of calcium through feces (Zini *et al*. 2007). The decrease in creatinine level is likely related to malnutrition or dehydration in the patient.

The treatments administered to the patient included intravenous Ringer's lactate infusion to restore body fluids due to dehydration, subcutaneous Ornipural® injection to enhance liver and kidney function, and Samylin® containing a complex of antioxidants to protect the liver. Additionally, the patient was administered cephalexin (Rilexine®) antibiotics to prevent bacterial infections that could worsen the pancreatitis. The patient was provided specialized renal wet food (Royal Canin K/D®) to prevent further kidney deterioration caused by elevated blood glucose levels.

## ■ CONCLUSION

Pancreatitis in cats is often associated with prediabetes and hepatic lipidosis, leading to elevated blood glucose levels and fatty liver disease due to decreased insulin levels. Treatment included Ornipural®, Samylin®, Rilexine®, and specialized renal food with possible additional antiemetics and appetite stimulants. If the prediabetes progresses to diabetes, insulin therapy should be considered. The limited equipment availability and the owner's ability to afford them prevented a definitive diagnosis; therefore, further tests such as fructosamine measurement, urinalysis, and abdominal ultrasound or X-ray are recommended.

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## ■ REFERENCES


Armstrong PJ, Williams DA. 2012. Pancreatitis in cats. Topics in Companion Animal Medicine. 27(3):140–147.

Bazelle J, Watson P. 2014. Pancreatitis in cats. Journal of Feline Medicine and Surgery. 16(5):395–406.

Dilworth L, Facey A, Omoruyi F. 2021. Diabetes Mellitus and Its Metabolic Complications: The Role of Adipose Tissues. Inter-national Journal of Molecular Sciences. 22(14):7644.

Forman MA, Steiner JM, Armstrong PJ, Camus MS, Gaschen L, Hill SL, Mansfield CS, Steiger K. 2021. ACVIM consensus statement on pancreatitis in cats. Journal of Veterinary Internal Medi-cine. 35(2):703–723.

Hong W, Zimmer V, Basharat Z, Zippi M, Stock S, Geng W, Bao X, Dong J, Pan J, Zhou M. 2020. Association of total cholesterol with severe acute pancreatitis: A U-shaped relationship. Clini-cal Nutrition. 39(1):250–257.

Kodali VRR, Ali I. 2016. Prediabetes: the cat is out! International Journal of Diabetes in Developing Countries. 36(2):143–145.

Zini E, Hauser B, Ossent P, Dennler R, Glaus T. 2007. Pansteatitis and severe hypocalcaemia in a cat. Journal of Feline Medicine and Surgery. 9(2):168–171.