Acute Pancreatitis Secondary to Hypercalcemia in Primary Hyperparathyroidism: A Case Report

Akutni pankreatitis zaradi hiperkalciemije pri primarnem hiperparatiroidizmu: prikaz primera

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***Ključne besede**: akutni pankreatitis; hiperkalcemija; primarni hiperparatiroidizem; adenom obščitnične žleze; nekrotizirajoči pankreatitis; večorganska odpoved*

ABSTRACT

Acute hypercalcaemic pancreatitis is a rare form of acute pancreatitis which develops when the serum calcium concentration exceeds 2.6 mmol/L. This case report presents a 29-year-old patient who developed a severe form of necrotising pancreatitis as a consequence of hypercalcemia due to primary hyperparathyroidism. The cause of hypercalcemia was a benign adenoma of the parathyroid gland, which was surgically removed. Despite the removal of the underlying cause, numerous complications developed that are characteristic of a severe course of the disease. The case highlights the importance of early recognition of hypercalcemia as a rare but serious cause of pancreatitis. It emphasises the complexity of treatment in patients with a complicated disease course.

IZVLEČEK

Akutni hiperkalcemični pankreatitis je redka oblika akutnega pankreatitisa, ki se razvije, ko koncentracija kalcija v serumu preseže 2,6 mmol/L. V tem poročilu o primeru predstavljamo 29-letnega bolnika, ki je razvil hudo obliko nekrotizirajočega pankreatitisa kot posledico hiperkalciemije zaradi primarnega hiperparatiroidizma. Vzrok hiperkalciemije je bil benigni adenom obščitnične žleze, ki je bil kirurško odstranjen. Kljub odstranitvi osnovnega vzroka so se razvili številni zapleti, značilni za hud potek bolezni. Primer poudarja pomen zgodnjega prepoznavanja hiperkalciemije kot redkega, a resnega vzroka pankreatitisa. Poudarja kompleksnost zdravljenja pri bolnikih z zapletenim potekom bolezni.

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INTRODUCTION

Acute pancreatitis

Acute pancreatitis is an acute inflammation of the pancreatic gland, presenting with abdominal pain and/or elevation of pancreatic enzymes three times above the normal value and/or typical changes on ultrasound or abdominal CT. According to the severity, it is classified into mild, moderate and severe. Severe pancreatitis is present in 5–20% of all cases.[1] In less than 1% of cases of acute pancreatitis, hyperparathyroidism is also present. Acute pancreatitis develops in less than 4% of patients with hyperparathyroidism.[2]

Aetiology of acute pancreatitis

Acute pancreatitis is one of the most common diseases of the gastrointestinal tract, with an annual incidence of 10–80 people per 100.000. The incidence is increasing by 3–4% per year. The most common causes of pancreatitis are gallstones (40%) and excessive alcohol consumption (20–35%), which together account for 75–80% of cases of acute pancreatitis in developed countries.[3] Other causes (20%) include hypertriglyceridemia, drug-induced pancreatitis, trauma, iatrogenic causes (e.g. endoscopic retrograde cholangiopancreatography (ERCP), pancreatic biopsy, etc.), infections, autoimmune causes, genetic causes, vascular causes (pancreatic ischemia) and others.[4] In around 5% of the patients, the underlying cause cannot be identified (idiopathic pancreatitis).[3]

Hypercalcemia is an infrequent cause of pancreatitis and occurs in 7–10% of patients with primary hyperparathyroidism (PHPT).[5]

Hypercalcaemic pancreatitis

Hypercalcemia is defined as a serum calcium level above 2.6 mmol/l or ionised calcium above 1.23 mmol/l.[1, 6] The most common causes of hypercalcemia are primary hyperparathyroidism (90%) and malignancies. Hypercalcemia is a rare cause of acute and chronic pancreatitis (1–7%)[1, 7], with its mechanism still not fully understood. Proposed mechanisms include premature activation of trypsinogen into trypsin inside the pancreas, obstruction of the pancreatic duct due to calcium deposits, and genetic mutations, which, in combination with hypercalcemia, may substantially increase the risk for acute pancreatitis in patients with PHPT.[8, 9]

Complications

The complications of acute pancreatitis are local and systemic. Local complications include peripancreatic fluid collections, pancreatic pseudocysts, acute necrotic collections, and walled-off necrosis (WON). Other possible complications are splenic or portal vein thrombosis, gastric outlet obstruction, and colonic necrosis. Systemic complications are defined as the worsening of pre-existing conditions, e.g. coronary artery disease, chronic pulmonary disease, etc.[1, 10]

CASE REPORT

A 29-year-old male with a euthyroid nodule, identified in August 2022, HLA B27-negative ankylosing spondylitis presented to the medical emergency department in September 2024 with abdominal pain and elevated serum amylase and lipase levels. He immediately received aggressive parenteral hydration; additional laboratory investigations were obtained. Laboratory tests revealed hypercalcemia. He progressed to shock, with markedly elevated lactate and inflammatory markers, hyperglycaemia, and biochemical evidence of acute kidney injury. Owing to severe acute pancreatitis with multi-organ failure, he was admitted to a level-3 intensive care unit, where his condition initially improved with conservative management.

He was subsequently transferred to the intensive care unit at the Gastroenterology Clinic, where CT imaging of the thorax and abdomen revealed extensive pancreatic necrosis and a nodular lesion in the

right thyroid lobe/parathyroid gland. PET-CT with a choline tracer demonstrated a minimally metabolically active lesion. Further workup confirmed hypercalcemia due to primary hyperparathyroidism, which was the cause of the severe necrotising pancreatitis.

He underwent surgery with excision of a large tumour in the right parathyroid gland. Pathohistological examination revealed an atypical adenoma. Postoperatively, he was transferred to the endocrinology department, where his condition deteriorated again. He developed acute respiratory failure and paralytic ileus of the small bowel. Imaging demonstrated progression of necrotic collections.

He was moved to the gastroenterology department, where he developed signs of infection that were treated with antibiotics. After maturation of the walled-off necrosis (WON), endoscopic drainage was performed via the gastric wall using a lumen-apposing metal stent (LAMS). This procedure initially improved the patient's clinical status for several weeks, but his condition worsened again.

He was transferred back to the level-3 intensive care unit, where he developed hypovolemic shock due to haemorrhage into the necrotic collection and required intubation. Abdominal compartment syndrome ensued, necessitating reoperation with abdominal lavage. Repeat CTA revealed a haematoma and a pseudoaneurysm adjacent to the LAMS. He underwent further surgery with the removal of the stent and necrotic pancreatic tail, followed by gastric wall suturing.

Because of severe pain, an epidural catheter was inserted. His symptoms deteriorated again, and CT imaging revealed gastric wall disruption, which was repaired during a third operation. Prolonged intubation required percutaneous tracheostomy. He received parenteral nutrition throughout hospitalisation.

During this period, left femoral vein thrombosis was detected and treated with standard heparin and a vena cava filter. This intervention provoked upper gastrointestinal bleeding. Emergency gastroscopy revealed a bleeding gastric ulcer, which was treated with haemostatic clipping. The procedure also revealed gastric wall dehiscence and pyloric stenosis, leading to nasojejunal tube placement. A right jugular vein thrombosis prompted reintroduction of unfractionated heparin, which resulted in haematemesis. Repeat gastroscopy demonstrated reflux esophagitis.

The patient's condition gradually improved. Small oral meals were introduced in conjunction with parenteral nutrition. After 2 months in the level-3 intensive care unit, he was transferred back to the gastroenterology intensive care unit. During hospitalisation, he developed insulin-dependent diabetes mellitus and electromyography confirmed critical illness myopathy. With gradual rehabilitation, he progressed to standing, walking with a walker, and tolerating everyday meals. As his condition improved, the tracheostomy and vena cava filter were removed.

After nearly six months of hospitalisation, he was transferred to the University Rehabilitation Institute Soča, where he completed rehabilitation and was discharged to home care in stable condition.

DISCUSSION

The case of a 29-year-old man illustrates a rare but clinically significant complication of primary hyperparathyroidism: acute hypercalcaemic pancreatitis. Although hypercalcemia as a cause of pancreatitis is well documented, it occurs in fewer than 10% of patients with PHPT, which is asymptomatic in most cases. The condition is commonly due to a benign parathyroid adenoma and is often detected incidentally.

Hypercalcemia as a cause of pancreatitis is well described but extremely rare. Pancreatitis occurs in less than 7–10% of patients with PHPT, which is asymptomatic in 80% of cases.[1] The most common cause (80–85%) of PHPT is benign parathyroid adenoma.[9] The disease is often detected only when

hypercalcemia is found incidentally on routine laboratory testing. The clinical presentation depends on the duration of the disease, degree of hypercalcemia, and the effects of parathyroid hormone on target organs.[1] It may present solely with acute pancreatitis, as in this case.[9] Patients with PHPT are 28 times more likely to develop acute pancreatitis compared to the general population.[9, 11]

In this case, the cause of hypercalcemia was primary hyperparathyroidism due to an atypical adenoma of the parathyroid gland. Marked hypercalcemia caused severe acute necrotising pancreatitis with multi-organ failure (renal, circulatory, respiratory).

After surgical removal of the parathyroid adenoma, hypercalcemia normalised, but the clinical course was complicated. The patient developed an infected WON requiring endoscopic transluminal drainage with LAMS. Despite initial improvement, severe complications occurred – bleeding into the necrotic collection with hypovolemic shock, abdominal compartment syndrome, gastric wall perforation, and recurrent gastrointestinal bleeding. These required multiple surgeries, endoscopic haemostatic procedures, and complex intensive care.

The course was further complicated by thromboembolic events (femoral and jugular vein thrombosis), sepsis, paralytic ileus, long-term respiratory support with tracheostomy, critical illness myopathy and newly diagnosed diabetes due to pancreatic destruction – prolonged treatment required several months of intensive rehabilitation, which led to almost complete functional recovery.

Rapid recognition of rare causes of acute pancreatitis is crucial for optimal treatment and management. While gallstones and alcohol are the most common causes, they are less frequent in young adults; Baeza-Zapata et. al. report a biliary cause frequency of 56.4%–64.9% in older patients and 20.2%–37.3% in younger patients.[12] Therefore, in young patients without typical risk factors, rare causes must be considered.

Hypercalcemia is considered an emergency if calcium levels exceed 3,5 mmol/L, which may lead to a life-threatening condition known as a hypercalcaemic crisis. It presents with the acute onset of neurological symptoms, gastrointestinal disturbance, dehydration, acute kidney injury, and potentially fatal arrhythmias.[13]

The case highlights the importance of a multidisciplinary approach in managing acute pancreatitis. Multiple organ failure occurs in approximately 18% of cases.[15] Combined with the extended hospitalisation typical of necrotising pancreatitis, which predisposes to additional complications, this underscores the need for close cooperation between multiple specialities.[16] In this case, gastroenterologists, endocrinologists, oncologists, surgeons, intensivists, infectious disease specialists, otorhinolaryngologists, dietitians, physiatrists, physiotherapists, psychiatrists, and many others were involved.

Patients with severe pancreatitis often develop long-term complications. Hollemans et. al. report recurrent pancreatitis in 26%, exocrine pancreatic dysfunction in 38% and endocrine dysfunction in 34% of cases.[17] Both exocrine and endocrine dysfunction developed in our patient, along with critical illness myopathy, maldigestion, and psychophysical exhaustion.

A key intervention in this case was the insertion of a lumen-apposing metal stent (LAMS) for drainage of infected WON. LAMS enables the minimally invasive treatment of infected collections and is generally considered the first-line treatment when the anatomy is favourable. However, complications may occur during or after the procedure, including bleeding, perforation, dislocation, or obstruction.

In conclusion, this case illustrates how a rare endocrine disorder can trigger a cascade of life-threatening complications. In acute pancreatitis of unclear aetiology, rare causes must always be considered. In severe disease, prolonged, specialised, multi-level care is often required. Nevertheless, with timely diagnosis and persistent multidisciplinary treatment, favourable long-term recovery is achievable.

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