Acute Pancreatitis without Increased Pancreatic Enzymes

Pancreatite Aguda sem Elevação das Enzimas Pancreáticas

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DOI: https://doi.org/10.29315/gm.965

RESUMO

A pancreatite aguda é uma doença comum, heterogénea e imprevisível, com morbilidade e mortalidade significativas associadas. O reconhecimento precoce e a gestão atempada desta condição são essenciais para prevenir complicações.

É caracterizada por inflamação pancreática, frequentemente associada ao aumento dos níveis de amilase e lipase séricas. No entanto, este critério não é um requisito obrigatório para o diagnóstico desta doença. Tal apresentação é rara e pode constituir um dilema diagnóstico, potencialmente conduzindo a um diagnóstico incorreto e a um tratamento tardio.

Este artigo apresenta um caso de pancreatite aguda em que tanto os níveis de amilase como os de lipase estavam dentro dos limites de referência.

PALAVRAS-CHAVE: Amilase; Dor Abdominal; Lipase; Pancreatite Aguda

ABSTRACT

Acute pancreatitis is a common, heterogeneous, and unpredictable disease with significant associated morbidity and mortality. Early recognition and timely management of this condition are essential to prevent complications.

It is characterized by pancreatic inflammation, most commonly associated with increased serum amylase and lipase levels. However, this criterion is not a mandatory requirement for the diagnosis of this disease. Such a presentation is rare and may constitute a diagnostic dilemma, potentially leading to misdiagnosis and delayed treatment.

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Recebido/Received: 2024-11-19 Aceite/Accepted: 2025-10-16. Publicado online/Published online: 2025-11-12.

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This article presents a case of acute pancreatitis in which both amylase and lipase levels were within reference limits.

KEYWORDS: Abdominal Pain; Amylases; Lipase; Pancreatitis, Acute/diagnosis

INTRODUCTION

Acute pancreatitis is a common condition associated with significant morbidity and mortality,¹ and recent studies have demonstrated a steadily rising incidence over the past decades, affecting both sexes and all age groups.² It is characterized by a local and systemic inflammatory response, and is frequently accompanied by elevated levels of serum pancreatic enzymes, most commonly amylase and lipase, due to the destruction of the pancreatic parenchyma.³

Nevertheless, although rare, acute pancreatitis can present with normal pancreatic enzyme levels.^{3,4} Such presentation constitutes a diagnostic dilemma, which can lead to misdiagnosis and delayed treatment.¹

CASE REPORT

A 63-year-old male with a past medical history of well-controlled essential hypertension presented to the Emergency Department with abdominal pain persisting for two days. He described the pain as sharp, sudden in onset, moderate to severe, located in the upper abdominal region and radiating to his back, worse when lying flat, and slightly better when standing. The pain was persistent and worsening despite the use of over-the-counter paracetamol. He noted that the pain was unrelated to food intake and that this was his first experience of such an episode. He had associated nausea and poor appetite. The patient also reported having fever with a maximum of 39.2°C measured with an axillary thermometer, which subsided after taking paracetamol. He denied vomiting, weight loss, blood loss, diarrhea, or constipation. He had no respiratory, neurologic or genitourinary symptoms. He denied tobacco, illicit drug, and alcohol use, as well as newly introduced medication or herbal supplements. He had no prior hospitalizations or surgeries. There was no known personal or family history of gallstones, hypertriglyceridemia, malignancy, endocrine or autoimmune disorders.

Upon admission, his vital signs were unremarkable except for a slightly elevated blood pressure. Physical evaluation revealed tenderness in the epigastric region and left upper abdominal quadrant on palpation, but the abdomen was soft, without organomegaly, guard-

ing or rebound tenderness. The remaining physical examination was unaltered.

Laboratory testing showed leukocytosis ($19.2\times10^3/\mu$ L; reference range – RR: $4.0\text{-}10.0\times10^3/\mu$ L) with neutrophil predominance ($14.6\times10^3/\mu$ L; RR: $2.0\text{-}7.0\times10^3/\mu$ L) and elevated C-reactive protein (20.6 mg/dL; RR: 0.05-1.00 mg/dL). Pancreatic enzymes were within normal limits, with amylase at 35 IU/L (RR: 25-115 IU/L) and lipase at 108 IU/L (RR: 73-393 IU/L). Hemogram, troponin, calcium, glycose, renal and hepatobiliary panels were normal. Triglycerides were also within the reference range (145 mg/dL; RR: <150 mg/dL). Blood and urine cultures were collected and returned negative. At admission, the patient had a Ranson's score of two, indicating a mild risk of mortality from acute pancreatitis.

A contrast-enhanced computed tomography (CT) scan of the abdomen demonstrated peripancreatic adipose and fascial densification around the body and tail of the pancreas, suggestive of edematous pancreatitis (Fig. 1). An abdominal magnetic resonance imaging (MRI) scan was also performed, which was consistent with acute acalculous pancreatitis. Neither the CT nor the MRI revealed any pancreatic calcifications or fibrosis.

The final diagnosis was acute idiopathic edematous pancreatitis. The patient was managed conservatively with intravenous fluids and analgesics. Fasting was



FIGURE 1. Contrast-enhanced CT – axial cut: Acute edematous pancreatitis

initiated, followed by the gradual introduction of oral feeding. Amylase and lipase levels remained normal across several determinations. He reported symptom resolution and was discharged with follow-up in an Internal Medicine consultation.

DISCUSSION

Acute pancreatitis has an annual incidence of approximately 34 cases per 100 000 person-years¹ and a recurrence rate of around 20%.³ The overall mortality rate is about 5%, but it can rise to 47% in patients with multiorgan failure.⁵

The most common causes of acute pancreatitis are gallstones and alcohol abuse, while less common etiologies include hypertriglyceridemia, hypercalcemia, medications, endoscopic retrograde cholangiopancreatography, and surgical trauma.^{1,4} However, approximately 20% of cases remain idiopathic.⁴

The pathogenesis of acute pancreatitis involves increased conversion of trypsinogen to trypsin, leading to pancreatic injury and inflammation, which in turn triggers the release of amylase and lipase from pancreatic acinar cells.⁴

Acute pancreatitis is most often accompanied by an increase in pancreatic enzymes due to the destruction of the pancreatic parenchyma, with amylase and lipase being the most readily available, and included as part of the diagnostic criteria.^{3,6}

According to the American College of Gastroenterology, the diagnosis of acute pancreatitis is based on meeting at least two of the following three criteria: i) characteristic abdominal pain, ii) serum amylase and/ or lipase levels at least three times the upper limit of normal, and iii) abdominal imaging findings consistent with the disease.⁶

Therefore, normal serum levels of amylase and lipase do not exclude the diagnosis of acute pancreatitis and, although rare, several case reports document this presentation.^{3,4,7-9}

Several factors have been linked to acute pancreatitis with normal amylase and lipase levels. One factor pertains to the timing of testing: while amylase levels increase rapidly after the acute event and remain elevated for about three to five days, lipase levels peak at 24 hours and can remain elevated for one to two weeks, as lipase is reabsorbed by the kidney tubules.⁴ Therefore, early or late testing can yield normal results.

Another explanation is the onset of an acute-onchronic pancreatitis episode. Both enzyme concentrations may not be elevated if significant damage to the pancreatic parenchyma has previously occurred, with consequent loss of pancreatic function.^{3,7}

Gallstone pancreatitis is another cause related to acute pancreatitis without increased pancreatic enzymes, but the mechanism remains unclear.³

In cases of alcoholic pancreatitis, it has been reported that up to 20% of patients lack an increase in amylase due to exocrine pancreatic insufficiency.^{3,10}

In hypertriglyceridemia-induced pancreatitis, around half of patients have amylase and lipase levels below the diagnostic criteria limit due to the dilutional effects of the lipemia. 3.7.9

Regarding our case, none of the etiological causes were identified, the patient was evaluated 48 hours after symptom onset, and acute-on-chronic pancreatitis was also considered an unlikely explanation based on clinical and radiological findings.

Further studies are required to better comprehend this condition and identify ideal markers for its early assessment.

LEARNING POINTS

This case report highlights the importance of a strong index of suspicion and early recognition in cases where acute pancreatitis presents with normal serum amylase and lipase levels.

When faced with a suggestive clinical presentation, it is essential to perform imaging tests for confirmation.

The mechanisms underlying this presentation are not yet fully understood, and the role of conventional biomarkers in diagnosing acute pancreatitis may not apply to every case.

DECLARAÇÃO DE CONTRIBUIÇÃO /CONTRIBUTORSHIP STATEMENT

JTP, DM, AC, AR,CT - Elaboração, revisão e aprovação final do artigo

Todos os autores aprovaram a versão final a ser publicada.

JTP, DM, AC, AR, CT - Drafting, revision and final approval of the article

All authors approved the final version to be published.

RESPONSABILIDADES ÉTICAS

CONFLITOS DE INTERESSE: Os autores declaram a inexistência de conflitos de interesse na realização do presente trabalho.

FONTES DE FINANCIAMENTO: Não existiram fontes externas de financiamento para a realização deste artigo.

CONFIDENCIALIDADE DOS DADOS: Os autores declaram ter seguido os protocolos da sua instituição acerca da publicação dos dados de doentes.

CONSENTIMENTO: Consentimento do doente para publicação obtido.

PROVENIÊNCIA E REVISÃO POR PARES: Não comissionado; revisão externa por pares.

ETHICAL DISCLOSURES

CONFLICTS OF INTEREST: The authors have no conflicts of interest to declare.

FINANCING SUPPORT: This work has not received any contribution, grant or scholarship.

CONFIDENTIALITY OF DATA: The authors declare that they have followed the protocols of their work center on the publication of patient data.

PATIENT CONSENT: Consent for publication was obtained.

PROVENANCE AND PEER REVIEW: Not commissioned; externally peer-reviewed

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