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CLINICAL CASE

Diagnosis of acute biliary pancreatitis in the absence of imaging findings

Diagnóstico de pancreatitis biliar aguda en ausencia de hallazgos por imagen

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Abstract

Acute pancreatitis is defined as a sudden inflammation of the pancreas, which can range from a mild state to organ dysfunction. We present a 63-year-old male patient who was admitted with epigastric pain radiating to the back for 3 days, associated with oral intolerance. Suspecting acute pancreatitis, studies were requested that revealed elevated lipase as well as liver enzyme abnormalities, thus suggesting a biliary etiology; therefore, conservative treatment was initiated. In conclusion, this case emphasizes the relevance of clinical judgment and early management, demonstrating that a favorable outcome can be achieved even when imaging studies fail to confirm a biliary etiology.

Keywords: *Acute pancreatitis. Biliary etiology. Lipase elevation. Clinical judgment. Case report.*

Resumen

La pancreatitis aguda se define como una inflamación súbita del páncreas, la cual puede variar desde un estado leve hasta disfunción orgánica. Presentamos el caso de un paciente masculino de 63 años que ingresó con dolor epigástrico irradiado hacia la espalda de 3 días de evolución, asociado con intolerancia oral. Ante la sospecha de pancreatitis aguda, se solicitaron estudios que revelaron elevación de lipasa, así como alteraciones en las enzimas hepáticas, lo que sugiere una etiología biliar; por lo tanto, se inició tratamiento conservador. En conclusión, este caso enfatiza la relevancia del juicio clínico y el manejo temprano, demostrando que se puede lograr un desenlace favorable incluso cuando los estudios de imagen no logran confirmar una etiología biliar.

Palabras clave: *Pancreatitis aguda. Etiología biliar. Elevación de lipasa. Juicio clínico. Reporte de caso.*

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Introduction

Acute pancreatitis is defined as a sudden inflammation of the pancreas, and that can compromise adjacent structures, such as the peritoneum[1]. This condition represents one of the main gastrointestinal causes of hospitalization worldwide; with its incidence estimated between 13 and 45 cases/100,000 inhabitants per year, and it increases with each year; however, the incidence varies considerably between each geographic region[2]. The main causes of pancreatitis are of biliary origin, alcohol consumption, and hypertriglyceridemia, but it can also be due to other conditions such as hypercalcemia, drug-induced, abdominal trauma, infections, and genetic mutations such as cystic fibrosis, among others[3].

The diagnosis of pancreatitis continues to be a clinical challenge; however, it has been established that at least two of the following three criteria are required: characteristic abdominal pain, biochemical studies with pancreatic enzymes elevated above 3 times the upper normal limit, and imaging studies suggestive of pancreatitis, among which computed tomography is mainly found. Among the three criteria, the one that presents the most volatility is the clinical criterion, since pain can vary from person to person; and the disease can even present from mild self-limited forms to more severe pictures with even multiorgan failure and death; therefore, its identification and proper management are crucial[4].

In this manuscript, we present the case of a male patient with a personal history of chronic diseases, who developed a condition compatible with acute pancreatitis suspected of probable biliary origin despite not identifying stones or evident abnormalities in imaging studies; highlighting in this case the importance of a clinical and biochemical-based diagnostic suspicion, comprehensive approach, and the usefulness of early conservative treatment.

Case presentation

A 63-year-old male patient is presented, who was transferred to the emergency department from a private facility to the Hospital General de Ciudad Juárez (HGCJ) due to an abdominal pain syndrome consisting of epigastric pain with radiation in a hemibelt distribution of 3 days of evolution and that did not decrease with treatment taken by himself at home.

At the time of questioning, the patient had a history of systemic arterial hypertension diagnosed approximately 10 years ago under control with losartan, amlodipine, and carvedilol; dyslipidemia treated with atorvastatin; hyperuricemia treated with allopurinol; and intermittent asthma controlled with rescue salbutamol. He also had a history of total bilateral hip arthroplasty 20 years ago with a good outcome, as well as placement of an intraocular lens in the right eye due to cataracts with adequate recovery. The patient denied recent hospitalizations, as well as drug addictions; and regarding significant family history, he only had arterial hypertension and diabetes mellitus in his father and paternal grandfather.

The condition began approximately 72 h before admission with epigastric pain of 4/10 intensity on the visual analog scale (VAS) that progressively increased to 9/10 VAS, with hemibelt radiation of stabbing nature; accompanied by nausea without vomiting, oral intolerance mainly to solids, and early satiety. Due to the pain, the patient self-medicated with omeprazole and hyoscine without specifying doses; however, he did not experience significant improvement, so he decided to seek medical attention. On arrival at the hospital, he reported that the pain slightly improved, he had no fever, jaundice, or abnormalities in bowel movements.

On physical examination at the time of admission, he presented with blood pressure of 110/80 mmHg, heart rate of 109 beats/min (bpm), respiratory rate of 21 breaths/min (bpm), oxygen saturation (SpO_2) of 90% in room air, temperature of 36.5 °C, weight of 130 kilograms (kg), height of 170 cm, and a body mass index of 44.08 kg/m^2. The patient was conscious, oriented in all 4 spheres, dehydrated skin and mucosa, without mucocutaneous jaundice. At the abdominal level, pain was noted on superficial and deep palpation in the epigastrium 8/10 VAS currently, with hemibelt radiation, without signs of peritoneal irritation. The rest of the physical exam showed no evident abnormalities.

Given the abnormal laboratory studies shown in table 1 and clinical presentation, acute pancreatitis of probable biliary origin was suspected; however, alkaline phosphatase was not elevated for the bilirubin level, so an abdominal ultrasound was requested and general surgery was consulted.

At the time of admission to the general surgery service, the patient continued with the previously described pain, without new added symptoms; with good bilateral ventilation and supported by inhalotherapy through nasal cannula at 3 L/min. On thoracic examination, bilateral basal crackles were revealed without signs of consolidation; the abdomen continued without signs of

Table 1. Laboratory tests with abnormal results at the time of hospital admission, consistent with acute pancreatitis

Parameter	Patient's result	Reference range
Amylase	1100 U/L	30-110 U/L
Lipase	6458 U/L	13-60 U/L
Total bilirubin	8.0 mg/dL	0.2-1.2 mg/dL
Direct bilirubin	7.3 mg/dL	0-0.3 mg/dL
Aspartate aminotransferase (AST)	268 U/L	10-40 U/L
Alanine aminotransferase (ALT)	314 U/L	7-56 U/L
Alkaline phosphatase	370 U/L	40-130 U/L
Glucose	145 mg/dL	70-99 mg/dL (fasting)
Urea	69 mg/dL	15-40 mg/dL
Blood urea nitrogen	32 mg/dL	7-20 mg/dL
Creatinine	2.6 mg/dL	0.6-1.3 mg/dL
Calcium	7.8 mg/dL	8.5-10.5 mg/dL
Magnesium	1.4 mg/dL	1.7-2.2 mg/dL
Phosphorus	6.7 mg/dL	2.5-4.5 mg/dL

peritoneal irritation, preserved diuresis, and no focal neurological signs or signs of hypoperfusion. For all the above, the general surgery service also considered acute pancreatitis of probable biliary origin as the diagnostic impression.

Given the diagnosis, absolute fasting and intravenous hydration with Hartmann's solution were indicated; omeprazole, analgesic medications, hyoscine, calcium gluconate, and magnesium sulfate were administered intravenously; and strict monitoring of vital signs and fluid balance was continued. Hepatobiliary ultrasound was requested again, revealing no abnormalities with a common bile duct of 5 mm with a wall thickness of 2 mm.

With the current treatment, the patient showed clinical improvement, with decreased abdominal pain and improved oral tolerance; regarding follow-up laboratory studies, a downward trend in pancreatic and hepatic enzymes, improved renal function, and correction of hydroelectrolytic disorders were observed. The patient was discharged from hospital stay with dietary instructions and warning signs for the recurrence of symptoms, in addition to close follow-up by outpatient consultation.

Discussion

The present work presented a clinical case of a 63-year-old male patient with a history of systemic arterial hypertension and controlled dyslipidemia, as well as grade III obesity; who developed a condition compatible with acute pancreatitis (AP), being that the condition was diagnosed highlighting a serum lipase of 6458 U/L, which is significantly higher than 3 times the normal limit; accompanied by typical abdominal pain; and therefore, the international diagnostic criteria for AP were met.

AP is a sudden inflammation of pancreatic tissue which, according to the 2012 revised Atlanta classification, can vary from mild pancreatitis if there is no organ failure or any complication; to moderately severe if there is transient organ failure (< 48 h) or with the presence of local complications; and to severe if there is already persistent organ failure for more than 48 h[5]. In the present case, the patient developed signs of a systemic inflammatory response but without evidence of persistent organ failure, which is why it was classified as moderately severe; and in addition to this, it was of biliary etiology, which is the most frequent cause of pancreatitis (40-70%), and although the exact mechanism is unknown, it is estimated that a gallstone can migrate to the common bile duct and reach the ampulla of Vater, which is where the pancreatic duct of Wirsung also drains; and by preventing the drainage of pancreatic fluids, the intraductal pressure increases, causing a premature activation of pancreatic enzymes inside the pancreas, and therefore, autodigestion and inflammation[6,7].

The diagnosis of pancreatitis is established following the 2012 revised Atlanta criteria, which require at least two of the following three elements:

- Characteristic abdominal pain in the epigastrium of sudden onset, which radiates in a belt-like pattern to the patient's back, of high intensity, and transient in nature[8]
- Biochemical studies with an elevation of amylase and/or lipase ≥ 3 times the upper limit of normal[8]
- Imaging studies with characteristic findings[8].

In our case, the patient met the clinical and biochemical criteria, so there was no confirmatory need to request an imaging study; being that the study of choice within the first 48 h of symptom onset is abdominal ultrasound, especially to rule out biliary etiology, since it has low sensitivity for the visualization of pancreatic tissue[9]. In the case of an unfavorable evolution to treatment or when diagnostic doubts persist, a computed

tomography scan is recommended once 72 h have passed, mainly to assess the severity of the case and to see the existence of local complications[10]. In this case, the suspicion of biliary etiology was supported by the elevation of total and direct bilirubin, as well as hepatic transaminases; however, the hepatobiliary ultrasound did not show any stones or abnormality of the biliary tract; being that this absence of findings does not rule out a biliary etiology, since it has been shown that in 20-30% of cases of acute pancreatitis of biliary origin, no visible stones are present in initial studies, especially in obese patients, as is the case[11].

Once the diagnosis was made, absolute fasting, intravenous hydration with Hartmann's solution, analgesia, and correction of hydroelectrolytic disorders were indicated. Current guidelines were followed regarding aggressive hydration, using only Hartmann's solution, since it is superior to saline solution in reducing systemic inflammation, preventing early complications, and avoiding hyperchloremic acidosis typically associated with the use of saline solution[12]; however, hydration should be an individualized and closely monitored process to avoid fluid overload, especially in patients at risk of abdominal compartment syndrome; and although in this patient, hydration was adequate, the patient presented considerable signs of systemic inflammatory response[8].

Early enteral nutrition was achieved within the first 72 h, since the initiation of oral feeding is highly recommended once the patient tolerates food, even when the patient continues to report pain, as it improves clinical outcomes and reduces the likelihood of infections. Regarding pain management, non-steroidal anti-inflammatory drugs were used, as their efficacy is similar to that of opioids and with fewer adverse effects, especially in mild and moderate cases[12].

At present, other treatments are being studied that are aimed at modulating pancreatic inflammation early; such as the use of tumor necrosis factor-α modulators[13], interleukin 1β[14], and caspase inhibitors[15]; as well as stem cell therapy strategies to reduce the progression of inflammation and necrosis[16]; however, these treatments are not yet part of the standard management of pancreatitis and are under investigation.

In summary, the present case was managed according to what is established in the international literature; however, there were considerable limitations, such as the lack of an imaging study to assess the structural integrity of the pancreas; in addition to the absence of the use of a prognostic index for risk stratification such as BISAP, Ranson, or APACHE II. Another thing to consider was the lack of cholecystectomy during hospitalization, since it is recommended that in cases of biliary-origin AP, cholecystectomy be performed to prevent recurrences; so discharge without this procedure implies a risk for new pancreatitis events of the same origin.

Conclusion

This case highlights the importance of a comprehensive clinical evaluation in the suspicion of acute pancreatitis; demonstrating that even in the absence of evidence that is typically confirmatory, other aspects should be assessed and taken into account, such as biochemical studies and the patient's clinical presentation; therefore, the diagnosis of biliary pancreatitis should always be considered even without conclusive imaging evidence, always placing medical care first as an individualized process with a multidisciplinary approach.

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Conflicts of interest

The authors declare no conflicts of interest.

Ethical considerations

Protection of humans and animals. The authors declare that no experiments involving humans or animals were conducted for this research.

Confidentiality, informed consent, and ethical approval. The authors have followed their institution's confidentiality protocols, obtained informed consent from patients, and received approval from the Ethics Committee. The SAGER guidelines were followed according to the nature of the study.

Declaration on the use of artificial intelligence. The authors declare that no generative artificial intelligence was used in the writing of this manuscript.

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