Colon necrosis secondary to acute pancreatitis: A case report and literature review

Necrosis del colon secundaria a pancreatitis aguda. Reporte de un caso y revisión literaria

A 39-year-old woman presented with obesity as the single comorbidity and a body mass index of 36 kg/m². She came to the emergency department, 2h before her hospital admission, complaining of sharp, stabbing epigastric pain of 8/10 intensity, accompanied by nausea and vomiting of the gastric and biliary content with no evident factors that either lessened or increased the pain. Upon arrival, the patient presented with tachycardia and the rest of her vital signs were within normal parameters. Physical examination revealed she was alert and mentally oriented as to place, time, and person. Cranial nerve and neurologic exam was unremarkable and cardiopulmonary exam was normal. The patient had a distended abdomen, reduced peristalsis, diffuse pain upon palpation of the upper hemiabdomen, and no limb alterations.

The laboratory work-up reported: hemoglobin 11.3 g/dl; hematocrit 33.5%; leukocytes 13.77 K/µL; glucose 167 mg/dl; cholesterol 527 mg/dl; triglycerides 3,804 mg/dl; normal kidney function and normal liver function tests; amylase 600 U/l and lipase above 3,000 U/L. An upper abdominal ultrasound revealed a normal pancreas, no abdominal free fluid, and no evidence of gallstones. The patient was admitted to the intermediate care unit with the diagnosis of acute pancreatitis secondary to hypertriglyceridemia, with no organ failure according to the modified Marshall scale.

Initial management was hydration, analgesia, and fasting. During the first week, the patient presented with abdominal pain that was difficult to control with opiates and with persistent systemic inflammatory response syndrome characterized by tachycardia and leukocytosis. On the fifth day, due to the lack of clinical improvement, an abdominal computed axial tomography (CAT) scan without contrast was carried out that revealed a necrosis-free pancreas, peripancreatic fluid collections and fluid collections in both paracolic gutters, and thickening at the level of the mesocolon. The patient continued fasting up to the seventh day, when it was decided to begin enteral nutrition through a nasogastric tube, which she tolerated adequately. During the second week, the patient presented with febrile peaks. Blood cultures were negative, the pain improved, and she tolerated the enteral diet. On the 14th day of hospitalization, the patient presented with right flank pain, important abdominal distension, absence of peristalsis, vomiting of undigested food, and clear signs of shock, for which she was transferred to the intensive care unit. A contrast-enhanced CAT scan (fig. 1) revealed necrosis of the peripancreatic fat and left anterior pararenal space. Gas can be observed in the mesocolon.

Figure 1 Contrast-enhanced CAT scan documenting necrosis of the peripancreatic fat and the left anterior pararenal space. Gas can be observed in the mesocolon.

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necrosis of the pancreas and transverse colon (fig. 2), and multiple abscesses in the transverse colon. Total colectomy and ileostomy were performed. The patient was released from the intensive care unit on day 30. On day 40 she presented with a gastrocutaneous fistula that was surgically repaired. The patient subsequently presented with good clinical progression and was taken to the general hospital ward on day 45. She was released from the hospital, completely asymptomatic, on day 60.

Local complications in acute pancreatitis are: acute peripancreatic collections, pancreatic pseudocyst, acute necrotic collection, and walled-off pancreatic necrosis. 1 Necrotizing pancreatitis presents in 10-20% of cases with a high mortality rate of 15-20%. 2

Colon complications are one of the rare local complications, presenting in 1% of the patients that develop acute pancreatitis, and they include bleeding, fistula, necrosis, or perforation. 3-6

The colon has been reported to be affected in up to 15% 2,8 of the patients with severe pancreatitis and is associated with a mortality rate of 54%. 7 Diagnosis is made at a mean of 25 days from the onset of clinical symptoms. 7 The pathophysiology of colon involvement is thought to be due to the extension of pancreatic enzymes and necrosis as main causes. 9

The diagnosis of colon involvement in patients with acute pancreatitis is generally a clinical challenge. It is suspected when there is no clinical improvement and imaging studies show colon pathology, but it is mainly a perioperative diagnosis. The treatment of choice continues to be surgical resection of the necrotic tissue. 10

Gastrointestinal involvement in acute pancreatitis that is undiagnosed, or whose management is delayed, results in a high mortality rate. Therefore, early disease recognition and early surgical treatment are of the utmost importance.

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Conflict of interest

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References


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