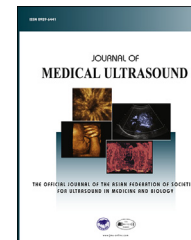


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CASE REPORT

Point-of-Care Ultrasound in Necrotizing Acute Pancreatitis Complicated by Perforated Ileum Due to Nonocclusive Mesenteric Ischemia

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Abstract Necrotizing acute pancreatitis is the most severe form of pancreatitis, and it is a potentially life-threatening condition. Its diagnosis and severity are based on radiological signs. Although computed tomography is the most used imaging tool, ultrasound can be a quick and useful technique in emergency and intensive care scenarios. The use of abdominal ultrasound is generally limited to ruling out cholecystitis. Bowel gas can limit the accuracy of pancreatic imaging. When the pancreas is visualized, ultrasound can reveal pancreatic enlargement, echotextural changes, and peripancreatic fluid. We present a patient with necrotizing pancreatitis who developed peritonitis due to ileal perforation, where the use of ultrasound as a bedside imaging technique was very useful.

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Introduction

Acute necrotizing pancreatitis is characterized by necrosis in and around the pancreas. Necrosis complicates 20–30% of patients with acute pancreatitis and has been associated with high morbidity (34–95%) and mortality (2–39%) rates [1]. Mortality within the first 2 weeks of onset is most often

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due to systemic inflammatory response syndrome, which is an exaggerated inflammatory response associated with systemic organ dysfunction, immunosuppression, and organ failure.

The diagnosis and severity assessment of acute necrotizing pancreatitis are largely based on imaging findings. Computed tomography (CT) is the main imaging procedure in pancreatitis. The use of ultrasound in acute pancreatitis is limited. The pancreas appears enlarged and hypoechoic on ultrasound. Ultrasound may be useful in demonstrating the presence of nonliquefied materials inside of a collection and/or peripancreatic fluid, and in diagnosing gallstones. In addition, necrosis and extrapancreatic inflammation are not well identified by ultrasound. Despite these facts, point-of-care ultrasound can play an important role in the management of acute pancreatitis. First, it can be performed daily without moving the patient from the intensive care unit to another facility. Second, we can detect some complications that occur in these patients early because of the free fluid in the peritoneal cavity and other changes produced during the disease course. Finally, it can be useful for prompt recognition of peritonitis, as we present below.

Case Report

An 82-year-old man with a 10-year history of arterial hypertension, taking enalapril 20 mg daily, was admitted to our hospital because of acute pancreatitis. Initially, oral intake was restricted, and intravenous fluids and narcotic analgesic were administered. CT revealed inflammation of the pancreas consistent with acute pancreatitis, with normal gallbladder and no evidence of dilatation or obstruction of the biliary tract. The patient developed severe respiratory distress, so he was intubated and transferred to the intensive care unit. Thoracic X-ray under mechanical ventilation showed pulmonary edema with bilateral pleural effusions. The abdomen was soft and tender to palpation. The first ultrasound performed showed peritoneal effusion and peristalsis (Figure 1). Over the following 48 hours, condition of the patient worsened; he developed acute kidney failure and hypotension, needing inotropic support. The intra-abdominal pressure increased

to 22 mmHg despite medical treatment. Temperature, leukocytes, and C-reactive protein increased, so cultures were carried out and empiric antibiotic was started (meropenem). Bedside ultrasound showed a larger peritoneal effusion with multiple septations, peristalsis was abolished, and intestinal loops were dilated with thickened walls (Figures 2 and 3). CT revealed necrotizing pancreatitis (>50% necrosis and Grade C of Balthazar score, with a total of eight points), free fluid in the peritoneal cavity, and dilated intestinal loops, but no clear signs of ischemia or perforation (Figures 4A and 4B). Open laparotomy showed 150 cm of infarcted and multiperforated ileum, and a segmentary resection was performed. The patient had to undergo surgery three times more because of abdominal collections. Finally, after a difficult weaning, he was extubated and restarted oral intake.

Discussion

Our patient developed severe necrotizing pancreatitis with persistent organ failure. The mortality rate increases up to 17% and complications occur more frequently.

Acute pancreatitis could be a severe disease with local or systemic complications. Necrotizing pancreatitis is related to a high probability of organ dysfunction. Associated inflammatory changes and collections may displace and compress adjacent organs. Obstruction of the stomach or bowel and hydronephrosis has been described as a mass effect caused by nearby collections and inflamed fat. Hollow viscus perforation is another possible complication, with the colon, stomach, and duodenum being most frequently affected. The effect of enzymes in the pancreatic collections, as well as the state of hypoperfusion and generalized inflammation (inflammatory changes may also secondarily cause bowel wall thickening, mural hyperenhancement, and adjacent fat stranding) associated with severe pancreatitis may explain intestinal perforations.

Patients with severe gastrointestinal tract obstruction or a large abdominopelvic fluid collection are at a risk of abdominal compartment syndrome. Increased intra-abdominal pressure results in organ ischemia and further



Figure 1 First day in ICU, fluid free in abdomen, normal movement in bowel.



Figure 2 Peritoneal effusion with multiple septations.

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