A 43-year-old male with intense epigastric abdominal pain with an episode of hematemesis looked for medical assistance. He was with heart rate of 91 bpm, blood pressure of 150/69 mmHg, temperature of 37.3°C, pale skin and mucous membranes, soft abdomen sensitive at the epigastrium. The laboratory examination results were: leukocytes: 6,080 cells/mm³, hematocrit 43.8%, temperature of 37.3°C, pale skin and mucous membranes, soft abdomen sensitive at the epigastrium. The laboratory examination results were: leukocytes: 6,080 cells/mm³, hematocrit 43.8%, hemoglobin 15.3 gr/dl, C-reactive protein 36.96 mg/dl. Due to the hematemesis, was done an upper GI endoscopy that revealed diffuse gastritis with necrotic foci. The patient’s abdominal pain persisted with peritoneal irritation signs, and follow-up laboratory examinations revealed: leukocytes 9,999 cells/mm³, hematocrit 44.4%, hemoglobin 15.3 gr/dl, C-reactive protein 457.1 mg/dl, amylase 616 U/l, lipase 698 U/l, total bilirubin 2.44 mg/dl, GOT/GPT 64/48 U/l and normal electrolytes. Patient had progressive hemodynamic instability, oliguria, with increased creatinine, requiring high doses noradrenaline.

A diagnosis of AP was made and he was re-evaluated, with diffuse abdominal pain with peritoneal irritation and 34 mmHg intra-abdominal pressure. Severity score of acute pancreatitis was APACHE II 10, PCR 457; Marshall of 4.

AP, gastric necrosis and abdominal compartment syndrome were possible diagnoses; an exploratory laparotomy was performed, foul-smelling bloody fluid was observed in the peritoneal cavity, stomach exhibited at least 95% necrosis from gastroesophageal junction to prepyloric region, greater omentum was completely necrotic, posterior abdominal wall was fused to the body of the pancreas. When a partial opening in epiploic transcavity was made, extensive pancreatic necrosis was revealed. Surgical cleaning was performed without gastric resection due to stomach and pancreas involvement. Postoperative care was administered in the ICU with antibiotics, hydration, parenteral nutrition and continuous insulin delivered via an infusion pump. The patient’s condition worsened in parallel with increases in the inflammatory parameters. An evaluation by hepatobiliary surgery and an abdominal and pelvic contrast using enhanced CT showed a slight increase in volume and the absence of enhancement of the body and tail of the pancreas (Figures 2 A and B) associated with a collection that extended towards the posterior gastric wall (Figure 2 C), which was found to be thickened and unenhanced (Figures 2 A and C). No involvement of the celiac axis (Figure 2 D) or its main branches was detected. A surgical re-exploration was scheduled for the 8th postoperative day. An abdominal angio-CT was performed and related vascular involvement was ruled out (Figure 3). An exploratory laparotomy revealed abundant, foul-smelling necrotizing free fluid; a culture was taken, surgical cleaning of the cavity was performed. A longitudinal partial gastrectomy of the necrotic body was the decision, and the patient was left with a contained laparotomy (Figure 4). Again, management in the ICU was required with mechanical ventilation for 27 days, after which the patient was transferred to the high-dependency unit, where he remained for 43 days before transfer to a ward. During this period, he was submitted to seven surgical cleanings and required a splenectomy and partial necrosectomy of the tail of the pancreas in addition to various antibiotic therapy regimens. The final surgical cleaning occurred 47 days after the initial one, and at this time, the Bogota bag was removed, and the abdominal wall was closed. Additionally, a high-debit pancreatic-digestive fistula was diagnosed and managed with drainage, and acute lobar cholecystitis (biliary sludge) was diagnosed and managed via the performance of a percutaneous cholecystostomy.
Enteric perforations are a rare complication of acute pancreatitis and involve a severe underlying pathology. This involvement usually occurs in cases of severe necrotizing pancreatitis.

Gastric necrosis related to pancreatitis is a rare complication because the perfusion originates from the branches of the celiac axis. The causes of gastric necrosis can be vascular, toxic, inflammatory, mechanical, infectious, autoimmune or idiopathic. In a case report published in 2012, only two cases were associated with acute pancreatitis.

Against this background, any vascular complication around the aorta and the celiac axis must be excluded. In this case, the vascular structures were examined via abdominal CT with contrast in the arterial phase (Figure 2 D) and subsequently with abdominal angi-o-CT, which ruled out pathology of the celiac axis or aorta (Figure 3). Another potential etiology involves the origination of the necrosis from disseminated extravascular coagulation, which would explain why there was no evidence of thrombosis detected by the angi-o-CT.

Another point to emphasize is the rarity of this clinical presentation. In the literature, there is only one case in which a patient with gastric perforation due to pancreatitis initially presented with hematemesis. In another reported case, a perforated gastric ulcer simulated pancreatitis, which emphasizes the importance of imaging to define the etiology.

For this patient, who was in a serious condition that involved multiple organ dysfunction, gastric necrosis in which some viability of the gastric curvature was preserved, and pancreatic and peripancreatic necrosis, we initially decided to perform a partial gastrectomy and pancreatic necrosectomy and planned several cleanings of the cavity during the evolution. This approach could be considered "damage control" for severe pancreatitis. It contrasts with the treatments administered in some reports, which include total gastrectomy, esophago-jejunal anastomosis, left pancreatectomy, cholecystectomy and splenectomy. Subsequent surgeries included a splenectomy for splenic necrosis and pancreatic and peripancreatic necrosectomies. During the evolution, a gastric fistula appeared and subsequently closed spontaneously. To guarantee the closure of this fistula, the feeding jejunostomy was important for nutritional management. Since discharge, endoscopic check-ups have revealed no lesions in the gastric mucosa or stenotic areas.

**REFERENCES**

1. Banks P, Bollen T, Dervenis C, Gooszen H, Johson C, Sarr M, et al. Acute Pancreatitis Classification Working Group. Classification of acute pancreatitis-2012: revision of the Atlanta classification and definitions by international consensus. Gut. 2013;62:102-111.

2. Losada M, Munoz-ozc, Burgos, Silva J. Protocolo de tratamiento de pacientes con pancreatitis aguda. Estudio de cohorte. Rev Chil Eter. 2010;62(6):557-563.

3. Petrov M, Windsor J. Classification of the Severity of Acute Pancreatitis: How Many Categories Make Sense? Am J Gastroenterol. 2010;105:74-76.

4. Johnson C, Abu-Hilal M. Persistent organ failure during the first week as a marker of fatal outcome in acute pancreatitis. Gut. 2004;53:1340-1344.

5. Butler A, Imrie C, Carter C, Evans S, McKay C. Dynamic nature of early organ dysfunction determines outcome in acute pancreatitis. Br J Surg. 2002;89:298-302.

6. Maravi E, Zubia F, Petrov M, Navarro S, Laplaza C, Morales F, et al. SEMICYUC. Recomendaciones para el manejo en cuidados intensivos de la pancreatitis aguda. Med Intensiva. 2013;37(3):163-179.

7. Losada H, Burgos L, Silva J, Acencio L, Arias O, Troncoso A, et al. APACHE II, Pancreatitis and gastric necrosis subsequent to acute pancreatitis. Panreatology. 2012;14:325-7.

8. Scholefield JG, Goodman A, Morgan W. Abdominal wall and gastric infarction in acute pancreatitis. Panreatology. 1988;3:494-6.

9. Nakamura H, Horiyata Y, Abe S, Omari N, Otsuki M. Ruptured aneurysm and gastric perforation associated with acute pancreatitis: a rare cause of hematemesis. Gastrointest Endosc. 2001;53(6):658-60.

10. Kuriko V, Okeno N. Occult perforation of gastric ulcer simulating pancreatitis. Klin Khr. 1974(04):47.