

Letters to the Editor

Predictable bad evolution of an acute pancreatitis?

Key words: Nissen. Acute pancreatitis. Abdominal pressure.

Dear Editor,

We present a 60-year-old woman with a previous cholecystectomy, operated of a parahepatic hernia in 2008 and reoperated with a Nissen fundoplication because of its reappearance in 2010. Ten months after this last intervention, the patient went to the emergency department because of sudden abdominal pain. The pain radiated from the epigastrium to both iliac quadrants and her back. In the physical examination she had no fever or dyspnea, she was conscious, with a normal cognitive and hemodynamic status. Abdomen was very distended overall in epigastrium and upper right quadrant and although there was no sign of abdominal guarding, no intestinal sound was audible. Blood tests showed hyperamylasemia (1,005 UI/mL). Abdominal X ray showed an evident dilatation of stomach and small bowel. The first 48 hours after admission there was bad pain control even with increasing doses of analgesics and peristalsis did not improve although amylase levels were normalized. A nasogastric tube was placed with no success. An abdominal TC with intravenous contrast showed an unstructured uncinat process and pancreatic head with peripancreatic fluid and hypercaptation of peripancreatic fat tissue, also great dilatation of stomach and an abdominal wall linea alba small hernia containing fat tissue and a small bowel loop with no sign of ischemia. There were also bilateral pleural effusion and atelectasis of posterior segments of both inferior lung lobes. Because of respiratory and renal functions worsening

the patient was admitted to the intensive care unit the 4th day needing intubation and hemofiltration. Intraabdominal pressure was measured (22 water cm) and a laparotomy was decided. In the intervention the bowel dilatation was confirmed, it extended to the ileum and there, there was a complete obstruction because of an adherence causing a 5-10 cm ileal segment ischemic necrosis. The ischemic segment was resected and an anastomosis was performed. A nasogastric tube for aspiration was placed with endoscopy. The patient had a good evolution and was discharged from the unit the 7th day.

Discussion

This case has made us consider if Nissen surgery could reduce the abdominal compliance to the pressure increase induced by a pancreatitis, favouring compression and intestinal ischemia, or even if acute pancreatitis could have been caused because of a similar mechanism (high intraabdominal pressure) induced by an intestinal obstruction. This kind of surgery has previously been related with intestinal obstruction and hernia development in adults and children and the necessity of reintervention (1-7). There have also been published clinical cases relating this surgery with pancreatitis, pancreatitis with a bad prognosis, gastric dilatation or intestinal obstructions (8-10). Fundoplication could then be causing these complications or their bad evolution. For all of this we think, as other authors have said, that the antecedent of a Nissen surgery should be taken into account when evaluating the evolution of a patient.

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