Spontaneous rupture of a pancreatic pseudocyst

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DESCRIPTION
This is a case of a 50-year-old woman with medical history significant for chronic pancreatitis secondary to alcohol abuse who initially presented with a 3-week history of progressively worsening epigastric pain. The patient described the pain as sharp, radiating to the back, worsened with intake of both solid foods and liquids and relieved with self-induced emesis. On admission, the patient was hypertensive, but other vitals were within normal limits. The abdomen was soft; however, there is a firm palpable mass extending from the epigastric to the right and left upper quadrants. Labs were remarkable for hypokalaemia with potassium (K) 3 meq/L (normal range 3.5–4.9 meq/L), mildly elevated lipase 132 U/L (7–60 U/L) and an elevated alkaline phosphatase of 164 U/L (41–108 U/L). CT of the abdomen with contrast (figures 1 and 2) showed a large cystic structure measuring 12.9×21.2 cm directly adjacent to the body of the pancreas and a fluid collection with enhancing rim measuring 4.8×6.6 cm near the pancreatic tail with subsequent displacement of the stomach. Gastroenterology recommended cyst-gastrostomy at a tertiary care centre. However, surgery recommended surgical cyst-gastrostomy in the facility. The patient was made nil per os and started on total parenteral nutrition. Patient declined pain medications. Four days after admission, the patient started complaining of sudden worsening of the abdominal pain. Abdomen examination remained unchanged from admission and no interval imaging studies were performed. The following day, the patient’s abdomen became diffusely rigid and she developed tachycardia and hypotension. Labs were remarkable for leucocytosis with rise in white cell count to 41.5×10⁹/L (4.0–12.0×10⁹/L), acute kidney injury with rise in creatinine from 0.36 to 1.85 mg/dL (0.60–1.40 mg/dL), hyperkalaemia with K 6.5 meq/L and elevation of lactate to 3.5 mmol/L (0.2–1.8 mmol/L). The patient was then started on piperacillin–tazobactam for empiric coverage. An emergent CT of the abdomen showed a decrease in the size of the pancreatic pseudocyst to 7.3×13.6 cm and a large amount of ascites suggesting rupture of the pseudocyst into the abdominal cavity (figure 3). Patient was then taken for emergent exploratory laparotomy. On entering the abdomen, there was a large amount of ascitic fluid which was sucked out using a pool-sucking device. About 3.5 L of abdominal fluid (light brownish, clear, fluid) was aspirated. There were some inflammatory flakes on the small bowel which were removed. There were inflammatory exudates in the pelvis and the suprahepatic region which were all washed out. There was a perforation noted in the pseudocyst about 3 cm just below the greater curvature of the stomach and between the transverse colon. The cavity could be easily seen and explored with the suction cannula. The fluid in the pseudocyst cavity was emptied. The colon was

Figure 1  CT of the abdomen (axial) with contrast showing a large cystic structure measuring 12.9×21.2 cm directly adjacent to the body of the pancreas.

Figure 2  CT of the abdomen (coronal) with contrast showing a fluid collection with enhancing rim or thickened wall measuring 4.8×6.6 cm near the pancreatic tail.

Figure 3  CT of the abdomen with contrast showing a decrease in the size of the pancreatic pseudocyst to 7.3×13.6 cm and a large amount of ascites suggesting rupture of the pseudocyst into the abdominal cavity.
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Figure 3  CT scan of the abdomen showing a decrease in the size of the pancreatic pseudocyst to 7.3x13.6 cm and a large amount of ascites suggesting rupture of the pseudocyst into the abdominal cavity.

Pancreatic pseudocysts are localised fluid collections as a result of acute or chronic pancreatitis. These structures are surrounded by fibrous and granulation tissue and contain fluid rich in amylase and other pancreatic enzymes. The pseudocysts appear to arise from an insult to the pancreatic duct leading to extravasation of pancreatic secretions. Pseudocysts occur in 25% of patients with chronic pancreatitis and have been observed more commonly in alcohol aetiology. Large pseudocysts can cause compression of adjacent structures leading to various complications and will warrant aggressive management. In a longitudinal study by Mehta et al, cyst >7.5 cm or >250 mL needs either surgical or endoscopic intervention.1 Spontaneous rupture of pancreatic pseudocysts has been reported in <5% of cases. Approximately, half of these empty into the free peritoneal or pleural cavity, portal or splenic vein; the other half into an adjacent hollow viscus including the oesophagus, stomach, duodenum, small intestine, colon and renal pelvis.

Pseudocyst rupture into the hollow gastrointestinal tract may lead to spontaneous regression; however, rupture into the intra-abdominal cavity may result in peritonitis. Even though the exact mechanism of pancreatic pseudocyst rupture is unknown, several factors contribute to an increased risk: progressive digestion of the cyst wall by proteolytic enzymes activated by entero-kinase, increased intra-abdominal pressure and minor abdominal trauma. The presence of high concentration of enzymes such as lipase, amylase and other proteolytic enzymes can lead to invasion into adjacent structures.2 3 Alternatively, erosion into an adjacent vessel may cause intravascular thrombosis.2 4

Learning points

- Pancreatic pseudocyst rupture is a rare complication that may warrant immediate surgical intervention if it causes signs of peritonitis.
- A sudden increase in abdominal pain with or without signs of peritonitis warrants further investigation to rule out possible pseudocyst rupture.
- Pancreatic pseudocysts which rupture into hollow organs may resolve spontaneously without causing any complications.

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REFERENCES