CASE REPORT

Acute necrotising pancreatitis: a late and fatal complication of pancreaticoduodenal arterial embolisation

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SUMMARY

A 70-year-old man was diagnosed with a massive bleeding duodenal ulcer which was refractory to emergency endoscopic management. Angiogram of the coeliac and superior mesenteric arteries revealed bleeding from the superior and inferior pancreaticoduodenal arteries. Transcatheter arterial embolisation of superior and inferior pancreaticoduodenal arteries along with the gastroduodenal artery was performed. Two weeks later he developed severe necrotising pancreatitis of the pancreatic head probably due to ischaemia, which was managed conservatively. Three months later the patient experienced another episode of pancreatitis which progressed into multiorgan dysfunction and the patient passed away.

BACKGROUND

Acute pancreatitis is a very rare complication of transcatheter arterial embolisation (TAE) of the main arteries supplying the pancreas. Few cases of pancreatitis following TAE of the splenic artery have been reported. This is probably the first case report of necrotising pancreatitis following TAE of superior and inferior pancreaticoduodenal arteries with metal coils and gelfoam.

CASE PRESENTATION

A 70-year-old Caucasian man with no significant medical history presented to the emergency room reporting of an episode of presyncope. His vitals were stable and physical examination was unremarkable. In the emergency room he had an episode of massive haematochezia and haematemesis and his haemoglobin dropped from 11.4 to 6.6 g/dL (reference value 13–14.2 g/dL). He received four units of packed red blood cells. Oesophagogastroduodenoscopy was performed which revealed a large class 1b duodenal bulb ulcer which was oozing blood.¹ Local haemostasis was attempted with epinephrine and coagulation with a gold probe but was unsuccessful. A selective angiogram of the coeliac and superior mesenteric arteries was performed and the patient was found to be bleeding from his superior and inferior pancreaticoduodenal arteries. He subsequently underwent transcatheter arterial embolisation of both arteries as well as the gastroduodenal artery with gelatin foam and arterial coils (figures 1–3). No subsequent bleeding was identified. He was discharged after a hospital stay of 2 weeks.

Figure 1 Selective angiography of the gastroduodenal artery showing duodenal bleeding (black arrow) from superior pancreaticoduodenal artery.

The patient presented to the emergency room 2 weeks later with abdominal pain. He did not have any melaena, haematemesis or haematochezia. Examination revealed a distended, diffusely tender abdomen with guarding and sluggish bowel sounds. An abdominal roentgenogram revealed air fluid levels. Laboratory data revealed an elevated lipase level at 3527 U/L (reference range 22–51 U/L). CT of the abdomen revealed necrosis within the pancreatic head, adjacent fluid collection, omental liquifactive necrosis and pelvic ascites (figures 4 and 5). He was diagnosed with acute necrotic pancreatitis with acute necrotic collection, as per the revised Atlanta classification system,² probably

Figure 2 Angiography of the superior mesenteric artery showing bleeding into the duodenum from inferior pancreaticoduodenal artery (black arrow). Metallic coil placed in gastroduodenal and superior pancreaticoduodenal artery.

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Figure 3 Metallic coils in gastroduodenal, superior artery and inferior pancreaticoduodenal artery with resolution of the duodenal bleed.

secondary to ischaemia from the pancreaticoduodenal artery embolisation, with an Acute Physiology and Chronic Health Evaluation (APACHE) II score of 10. The patient did not have any history of alcohol abuse, cholelithiasis or abdominal trauma and was not on any pancreato-toxic medications. His alanine transaminase, aspartate transaminase, alkaline phosphatase, calcium and triglyceride levels were within normal reference range. CT of the abdomen did not show any signs of cholelithiasis. He was managed conservatively and made a good recovery.

OUTCOME AND FOLLOW-UP

Three months later he again presented to the emergency room with severe epigastric pain. Laboratory data showed an increased amylase level at 1600 IU/L (reference range 30–110 IU/L) and lipase 9033 IU/L. CT of the abdomen revealed large fluid collection between the stomach and pancreas, diffuse inflammation within the pancreatic head and ascites. He was again diagnosed with necrotising pancreatitis with an APACHE³ II score of 17. The patient rapidly progressed into multiorgan dysfunction and passed away.

Figure 4 CT of the abdomen: arrow showing inflammation within the pancreatic head.

Figure 5 CT of the abdomen: arrow showing necrotic pancreas.

DISCUSSION

Endoscopy is considered the first-choice therapy for bleeding duodenal ulcers as it is the gold standard for diagnosis and multiple interventions can be performed to arrest the bleeding.⁴ TAE is the preferred second-line therapy for failed endoscopic intervention as the risks of surgery can be avoided.^{5 6} TAE is generally performed with gelatin foam and arterial coils. The metallic coils act as scaffolding for clot formation. Gelfoam can cause thrombosis by its close contact with platelets.⁷ TAE is generally regarded as a safe procedure with few complications. Wang *et al*⁸ reported a case series of 29 patients managed with TAE with a 93% success rate and no ischaemic complications. Another study with 95 patients showed a success rate of 98% in achieving haemostasis and with bowel ischaemic complications in 4% of the patients.⁹ Few case reports have been published on duodenal infarction¹⁰ and hepatic infarction¹¹ following TAE of the gastroduodenal artery.

Our patient is unique as acute necrotising pancreatitis of the pancreatic head following TAE of pancreaticoduodenal arteries for a bleeding duodenal ulcer has never been reported. The blood supply to the pancreatic head is mainly from the superior pancreaticoduodenal branch of the gastroduodenal artery and inferior pancreaticoduodenal branch of the superior mesenteric artery. The body and the tail are supplied by the dorsal pancreatic artery and caudal pancreatic artery which are branches of the splenic artery. The pancreas is sensitive to ischaemia damage in the laboratory and clinical settings such as cardiopulmonary bypass or haemorrhagic shock.¹² Cases of acute pancreatitis of the tail secondary to occlusion of end arteries and ischaemia following embolisation of the splenic artery have been reported.^{12 13}

Detailed history and laboratory investigations in our patient ruled out other probable causes of pancreatitis such as trauma, alcohol, cholelithiasis, hypertriglyceridaemia, hypercalcaemia or drug-induced pancreatitis. The fact that our patient had necrosis only within the head of the pancreas with sparing of the body and tail points towards ischaemia secondary to the gastroduodenal, superior pancreaticoduodenal artery and inferior pancreaticoduodenal artery embolisation as the probable aetiology. However, there was a time lag of 4 weeks between the procedure and the presentation with pancreatitis. The patient was

having vague abdominal pain during the period which he attributed to peptic ulcer disease and did not seek medical attention. He was probably having mild pancreatitis during that period which could have gradually progressed to acute necrotising pancreatitis. We hypothesise that the metallic coils stopped the duodenal bleeding initially but the thrombosis around the coils had gradually extended leading to complete obstruction of the arteries causing pancreatic ischaemia and infarction leading to necrosis in the pancreatic head.

Learning points

- ▶ Transcatheter arterial embolisation is the preferred second-line therapy for a bleeding duodenal ulcer after endoscopic interventions have failed.
- ▶ Acute pancreatitis is a potential complication of superior and inferior pancreaticoduodenal arterial embolisation.
- ▶ Patients should be informed of this potential complication when obtaining consent for the procedure.

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