Gastric outlet obstruction caused by a giant gastroduodenal artery aneurysm: a case report

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Gastric outlet (GO) obstruction in an adult is usually caused by intrinsic gastric or duodenal lesions or pancreatic tumours. This study describes a case of a 77-year-old man who developed GO obstruction due to extrinsic compression from a large gastroduodenal artery aneurysm under rupture. This cause of GO obstruction has never previously been reported in the literature. ***Eur J Gastroenterol Hepatol*** **13:59–61 © 2001 Lippincott Williams & Wilkins**

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Introduction

Visceral (splanchnic) artery aneurysms are uncommon conditions. They may cause vague symptoms, and few warning signs appear before sudden rupture occurs [1]. Gastroduodenal artery aneurysms are rare, most of them are related to pancreatitis and may present with rupture, abdominal pain, GI bleeding or jaundice [1–4], but gastric outlet obstruction has not been associated with them.

Case report

A 77-year-old man developed acute onset of abdominal pain radiating to the mid-scapular region and vomiting, 24 h following a blood transfusion. His past medical history was remarkable for chronic myelomonocytic leukaemia related to anaemia, requiring transfusions, thrombocytopenia and hepatosplenomegaly. There was no history of pancreatitis or cardiovascular disease.

On admission to a medical ward, his blood pressure was 110/80 mmHg and the pulse rate 90 bpm. The abdomen was diffusely tender, but without signs of peritoneal irritation and there was palpable organomegaly. The haemoglobin concentration was 9.0 g/dl, the leucocyte count was 15.8×10^9/l, and the platelet count was 40×10^9/l. The activated partial thromboplastin time (aPTT) and international normalized ratio (INR) were normal. The urea and creatinine were marginally increased, but electrolytes and amylase were normal. The liver function profile was essentially within normal limits, apart from a slightly reduced albumin (3.0 mg/dl) and a slightly increased total bilirubin (1.3 mg/dl). Testing for faecal occult blood was positive (++). An abdominal X-ray was unremarkable and a chest X-ray showed the left hemidiaphragm to be elevated. An emergency ultrasound scan showed a 6.5 cm 'cystic' lesion medial to the second part of the duodenum, a small amount of peritoneal fluid and hepatosplenomegaly.

During the next 2 days the patient continued to vomit, but his pain settled and he remained haemodynamically stable, receiving intravenous fluids and parenteral nutrition. He also received 2 units of red cells because his haemoglobin concentration had dropped to 7.9 g/dl. An upper gastrointestinal endoscopy showed pyloric deformity not allowing scope passage and a barium meal confirmed gastric outlet obstruction due to extrinsic compression with displacement of the pylorus and the duodenum by a large round mass (Fig. 1). Abdominal computed tomography (CT) was performed twice and demonstrated a round mass of 8 cm in diameter, posteriorly to the duodenal loop; this mass was contrast-enhanced and consistent with a large aneurysm of a visceral artery (Fig. 2). His symptoms of gastric outlet obstruction persisted and, on hospital day 5, he was referred to the surgeons and subsequently transferred to a surgical ward. Over the next few days, he remained haemodynamically stable and his haemoglobin concentration was also stable at 9.5 g/dl. His vomiting subsided and he received oral fluids and a semisolid diet.

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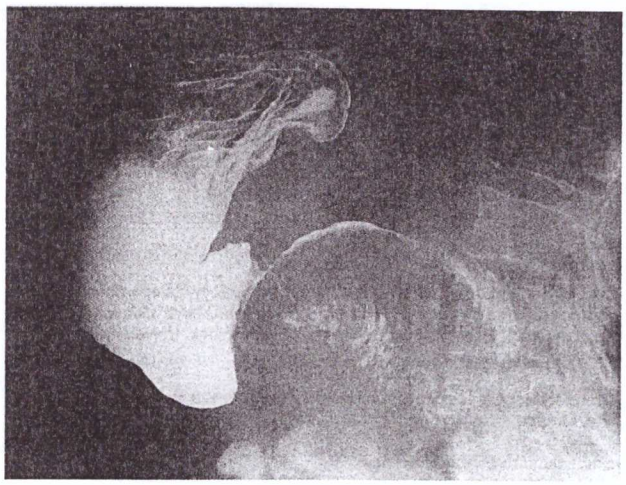
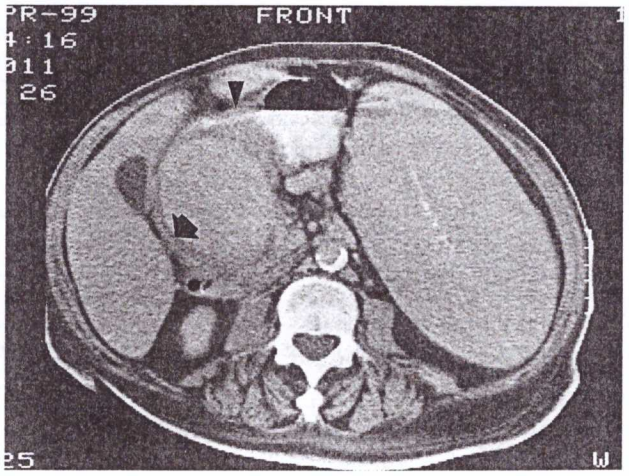
Fig. 1



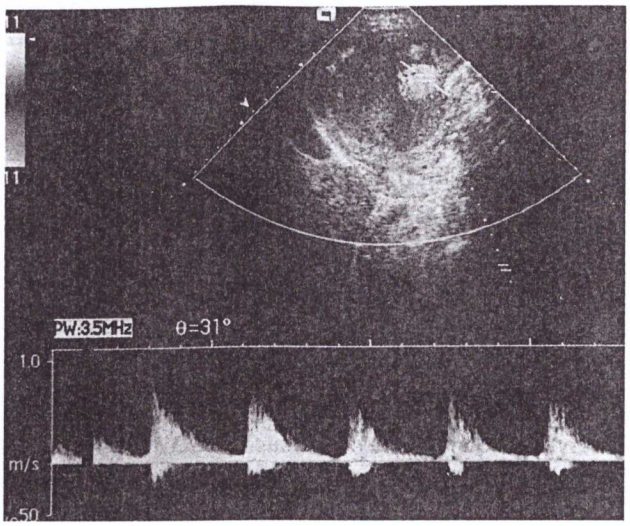
Lateral view of the upper GI tract after a barium meal. Pylorus and duodenum are partially obstructed by a large round mass.

Fig. 2



Contrast-enhanced abdominal CT scan. The aneurysm, with a thin layer of thrombus (thick arrow) adherent to its wall, is demonstrated posteriorly to and compressing the pylorus (arrow head). Splenomegaly is also noted.

Fig. 3



Duplex scanning of the structure reveals circumferential flow in it with a characteristic arterial waveform.

On hospital day 8 (a Friday), a colour flow duplex scan revealed a large vascular structure with arterial flow in it, lying caudally to the hepatic artery but separate from it, and, therefore, consistent with an aneurysm of the gastroduodenal artery (Fig. 3). On the following Monday, the patient was scheduled to have an arteriogram of the visceral arteries and possible embolization of the aneurysm, as that was thought to be the least invasive procedure for this debilitated and significantly thrombocytopenic elderly patient. During the weekend he should have received suitable treatment to improve his blood parameters prior to the procedure. Unfortunately, however, on hospital day 9 he suddenly collapsed and died. An autopsy showed rupture of the aneurysm into the free peritoneal cavity.

Discussion

Aneurysms of the gastroduodenal artery (GDA) are rare, accounting for only 1.5% of around 3000 visceral artery aneurysms documented in the literature [4]. These lesions are more common in men (male to female ratio is nearly 4:1). Periarterial inflammation due to pancreatitis has been reported to be the commonest cause of GDA aneurysms, because the majority evolve as complications of acute or chronic pancreatitis [2–4]. Other causes may be atherosclerosis or polyarteritis nodosa, which has been related to multiple visceral artery aneurysms. In this present case, the patient's aneurysm was atherosclerotic.

The majority of patients with GDA aneurysms are asymptomatic prior to rupture, which can be intra-abdominal, retroperitoneal or into the GI tract. Clinical presentation may include abdominal pain, acute or chronic upper GI bleeding and occasionally jaundice. Gastroduodenal obstruction has never been recognized as a complication of these lesions. To the best of our knowledge, this is the first documented case of GO obstruction secondary to a GDA aneurysm, which was compressing the adjacent duodenum because of its large size. Presumably, the aneurysm had acutely expanded and was under rupture, but no free or contained rupture was demonstrated on the contrast-enhanced CT scan. A case of duodenal stenosis, related to

ruptured tiny aneurysms of GDA branches, was due to haematoma formation and fibrosis between the pancreas and duodenum, and obviously not directly caused by the aneurysms themselves [5].

The prognosis of visceral aneurysms without treatment is poor, because of their high rupture rate [2–4]. In the present case, thrombocytopenia combined with technical problems precluded earlier intervention.

Treatment options for a GDA aneurysm include surgical ligation of entering and exiting arteries from within the aneurysm or percutaneous transcatheter embolization. A small number of embolizations for visceral artery aneurysms have been reported in the literature, mainly for aneurysms related to pancreatitis [6,7]. Embolic agents, such as metallic coils, gelatine sponge or bucrylate (isobutyl 2-cyanoacrylate) [8], should be placed intra-arterially proximally and distally to the aneurysm neck.

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