On-line Case Report

Superior mesenteric artery syndrome in association with an abdominal aortic aneurysm

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ABSTRACT

Superior mesenteric artery (SMA) syndrome, also known as Wilkie syndrome, is a rare condition characterised by compression of the third part of the duodenum against the aorta by the SMA. This can cause symptomatic duodenal obstruction. It is rarely associated with an abdominal aortic aneurysm (AAA). We report the case of a male patient presenting with an AAA and recurrent upper gastrointestinal symptoms. SMA syndrome was diagnosed with imaging and was then successfully treated by aneurysm repair. This is the smallest AAA associated with SMA syndrome in the literature. We propose an anatomical mechanism for SMA syndrome in this case.

Keywords: Superior mesenteric artery syndrome – Abdominal aortic aneurysm – Wilkie syndrome

Case report

A 65-year-old man presented with epigastric pain and early satiety. He was investigated for a hepatobiliary cause of this pain with abdominal ultrasound. This showed an abdominal aortic aneurysm (AAA) measuring 4.7 cm in maximum diameter, which was thought to be incidental and unrelated to his epigastric pain. The aneurysm was managed with regular surveillance and was found to be enlarging only slowly. He subsequently presented acutely with further episodes of epigastric pain and vomiting with a history of weight loss. On both occasions, there was no clinical suggestion of aneurysm leak. Examination showed he was thin (body mass index 20.2 kg/m²). The abdomen was soft and non-tender and there was an expansile epigastric mass. All lower limb pulses were palpable. Routine blood tests, including full

blood count, liver function tests, C-reactive protein and amylase, were unremarkable. On the second presentation, abdominal computed tomography (CT) was performed (Fig. 1). This showed the aneurysm was 5.7 cm in diameter. The first and second parts of the duodenum were dilated and the superior mesenteric artery (SMA) was heavily calcified. The sagittal reconstruction showed the aneurysm neck was angulated anteriorly and the aortomesenteric angle was only 10°. This suggested compression of the duodenum between the aneurysm and a calcified SMA. A barium meal confirmed this, showing pooling of contrast in the second part of the duodenum.

Aneurysm repair was indicated on the basis of aneurysm size and was performed through a transperitoneal approach. At laparotomy, the neck of the aneurysm was angulated anteriorly and the SMA was hard and densely calcified. These findings were consistent with the CT image. The distal aorta was relatively soft and friable. There was no inflammation, no peritoneal bands, no malrotation and the duodenum was

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Figure 1 CT abdomen. The AAA and a heavily calcified SMA are compressing the third part of the duodenum.

not involved in fibrous adhesions. The aneurysm was replaced with a Dacron tube graft. The postoperative course was uneventful and the patient was discharged home on the sixth postoperative day. On follow-up at 3 months, his gastrointestinal symptoms had completely resolved.

Discussion

SMA syndrome is characterised by symptomatic compression of the duodenum between the SMA and aorta. Many causes of SMA syndrome have been reported,¹ including an aortomesenteric angle of less than 25°. However, only four cases have been reported in association with an AAA: (i) in association with an 8-cm AAA;² (ii) a dissecting AAA with the duodenum involved in fibrous adhesions;³ (iii) postoperative adhesions around a 7-cm AAA following previous partial gastrectomy;⁴ and (iv) in a patient with an AAA and prolonged supine position following a rib fracture.⁵ In this report, we describe a patient with the smallest AAA in the literature to be associated with SMA syndrome. A narrow aortomesenteric angle was demonstrated on CT. Two factors may have contributed to this: the neck of the

aneurysm was noted radiologically and at laparotomy to be angled anteriorly; and the patient had a low BMI. These factors are the likely anatomical explanation in this case. SMA syndrome should be distinguished from other mechanisms of extrinsic duodenal compression associated with an AAA, which is known as aortoduodenal syndrome. This was excluded in our patient as there was no evidence of a peritoneal band, inflammatory adhesions or retroperitoneal fibrosis involving the duodenum. Repair of the AAA was sufficient to widen the aortomesenteric angle and thus relieve the SMA syndrome. SMA syndrome may also be treated by duodenojejunostomy, other bypass procedures and SMA transposition.

Conclusions

SMA syndrome is a very rare complication of an AAA. It has been reported with larger AAAs but here we describe, to our knowledge, the smallest AAA to be associated with SMA syndrome in the literature. It is important to recognise this syndrome as a complication of an AAA. Repair of the aneurysm should be considered more urgently than would be necessary based on its size alone, especially if the duodenal obstruction is severely symptomatic.

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