

'Coffee ground vomiting': an unusual cause of a common presentation

Introduction

This article describes an unusual presentation of aortoduodenal syndrome secondary to an inflammatory abdominal aortic aneurysm in a 70-year-old man who presented with 'coffee ground vomiting' and mild abdominal pain.

Discussion

Osler first described duodenal obstruction secondary to an abdominal aortic aneurysm, or 'aortoduodenal syndrome' in 1905. Although over 100 years have passed since then, fewer than 40 cases have been reported in the literature (Cahill et al, 2009). It is a rare condition with an elusive causative mechanism.

Inflammatory abdominal aortic aneurysm was first described by Walker et al in 1972. It accounts for 3–10% of all cases of abdominal aortic aneurysm and predominantly affects elderly men. Inflammatory abdominal aortic aneurysm is thought to occur 5–10 years earlier than atherosclerotic abdominal aortic aneurysm (Gans et al, 1993). Smoking and male gender are strongly linked with the incidence of inflammatory abdominal aortic aneurysm, more so than in atherosclerotic abdominal aortic aneurysm (Crawford et al, 1985). This case describes a diagnosis of aortoduodenal syndrome secondary to inflammatory abdominal aortic aneurysm.

Inflammatory abdominal aortic aneurysm is a distinct variant of atherosclerotic abdominal aortic aneurysm. Abdominal or back pain, fever and weight loss are the presenting complaint in 93% of cases. In

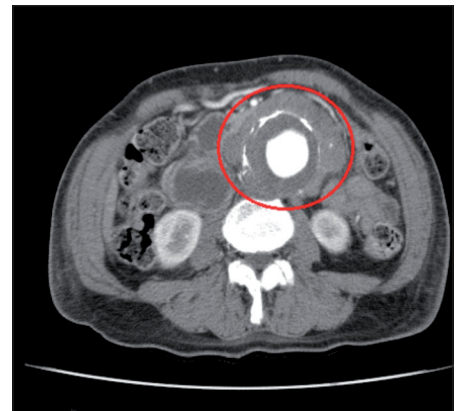
contrast, atherosclerotic abdominal aortic aneurysm is usually asymptomatic unless it has ruptured. A patient with an abdominal aortic aneurysm which has ruptured retroperitoneally presents with back pain, hypotension and pulsatile abdominal mass, whereas a patient with an abdominal aortic aneurysm which has ruptured intraperitoneally usually is fatal before arrival at hospital. An inflammatory abdominal aortic aneurysm, unlike an atherosclerotic abdominal aortic aneurysm, presents with raised inflammatory markers such as C-reactive protein and erythrocyte sedimentation rate (Bajardi et al, 2009). The mainstay of therapy in aneurysms greater than 5.5 cm in diameter is surgical or radiological repair, although corticosteroids and immunosuppressive therapy may also have a role in cases of inflammatory abdominal aortic aneurysm.

Conclusions

Aortoduodenal syndrome is a rare disorder. The exact causative mechanism is yet

to be elucidated although direct compression of the duodenum by the aneurysm against the abdominal wall or superior mesenteric artery has been postulated. Vomiting and a pulsatile abdominal mass are the most common presenting symptoms; abdominal pain, weight loss and electrolyte imbalances may also occur. Computed tomography scan followed

Figure 1. Abdominal aortic aneurysm with inflammatory component (circled in red).



Case Report

A 70-year-old man presented with a 3-day history of multiple episodes of 'coffee ground vomiting', mild abdominal pain, hiccups and low-grade fever. He denied previous similar events. He was on a course of flucloxacillin for bilateral leg cellulitis and had a previous medical history of well-controlled hypertension on lisinopril. He denied ischaemic heart disease, diabetes or hyperlipidaemia. He was an ex-smoker who had a moderate alcohol intake and was otherwise fit and well.

It was documented by the admitting team that he had a soft abdomen with generalized mild tenderness with no organomegaly or palpable expansile masses. His vital signs were unremarkable. His admission bloods showed markedly raised C-reactive protein level (45.1 mg/litre) and raised serum urea (17.7 mmol/litre) and creatinine (116 µmol/litre) levels. His full blood count, liver biochemistry and electrolytes were all within normal ranges. His Blatchford score was 7/15. His gastroscopy was only significant for severe oesophagitis (Los Angeles classification Grade D). Full assessment of the stomach was not possible because there was a large amount of fluid and food residue and a computed tomography scan was performed to elicit the cause of the obstruction. Duodenal obstruction as a result of entrapment between the superior mesenteric artery and an abdominal aortic aneurysm was demonstrated, and a 7.8 cm abdominal aortic aneurysm with inflammatory component causing duodenal compression and displacement of the pancreas was diagnosed (Figure 1).

Following his diagnosis, he was kept nil by mouth and had nasogastric tube decompression. He was started on total parenteral nutrition. Endovascular aneurysm repair was carried out which treated the aneurysm and relieved the obstruction. The operation was uneventful and there were no postoperative complications.

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by either upper gastrointestinal imaging endoscopy or contrast-enhanced upper gastrointestinal imaging should be performed to confirm the diagnosis and rule out alternative causes of duodenal obstruction (Bajardi et al, 2009).

Interestingly this patient did not complain of typical symptoms of small bowel obstruction and was investigated for upper gastrointestinal bleeding. This case demonstrates an unusual cause of a relatively common condition – doctors should be aware of this rare presentation of abdominal aortic aneurysm. **BJHM**

Bajardi G, Pecoraro F, Mirabella D, Bellisi MG

(2009) Inflammatory abdominal aortic aneurysm (IAAA). *Ann Ital Chir* **80**: 171–6
 Cahill K, Roche-Nagle G, MacEaney P, McGreal GI (2009) Upper gastrointestinal obstruction secondary to aortoduodenal syndrome owing to a noninflammatory abdominal aortic aneurysm. *Vascular* **17**: 168–71
 Crawford JL, Stowe CL, Safi HJ, Hallman CH, Crawford ES (1985) Inflammatory aneurysms of the aorta. *J Vasc Surg* **2**: 113–24

Gans RO, Hoorntje SJ, Rauwerda JA, Luth WJ, van Hattum LA, Donker AJ (1993) The inflammatory abdominal aortic aneurysm. Prevalence, clinical features and diagnostic evaluation. *Neth J Med* **43**: 105–15
 Osler W (1905) Aneurysm of the abdominal aorta. *Lancet* **ii**: 1089–905
 Walker DI, Bloor K, Williams G, Gillie I (1972) Inflammatory aneurysms of the abdominal aorta. *Br J Surg* **59**: 609–14

LEARNING POINTS

- Inflammatory aortic aneurysms are uncommon.
- This case highlights an unusual presentation with small bowel obstruction that was initially misdiagnosed as an upper gastrointestinal bleed.
- Early diagnosis with contrast imaging is key to treatment.

IMAGES IN MEDICINE

A 'chunky' pulmonary angiogram

A 42-year-old man presented to the emergency department with shortness of breath and mild chest pain following a syncopal episode earlier that morning.

On examination a loud systolic murmur throughout the precordium but no radiation to carotids was noted. Bloods showed a troponin level of <20 ng/ml (normal) and a D dimer level of 1.32 ng/ml (elevated). A provisional diagnosis of cardiac syncope as a result of symptomatic aortic stenosis was made with a need to exclude pulmonary embolus. A 12-lead electrocardiogram showed inferolateral T-wave inversion, a biphasic T wave in V1, with no left ventricular hypertrophy by voltage criteria. A

computed tomography pulmonary angiogram (Figures 1a and b) showed no pulmonary embolism but interventricular septum thickness of 27 mm with posterolateral wall up to 19 mm. The left ventricular wall was thickened but not dilated, consistent with hypertrophic cardiomyopathy.

Transthoracic echocardiogram showed interventricular septum hypertrophy of 2.3 cm with dynamic left ventricular systolic function and impaired left ventricular diastolic function. Left ventricular outflow tract gradient was 41 mmHg at rest increasing to 71 mmHg with valsalva indicating

dynamic outflow tract gradient confirming hypertrophic obstructive cardiomyopathy.

The computed tomography pulmonary angiogram is increasingly used to detect pulmonary embolism in patients with unexplained breathlessness or chest pain. Clinically useful data about the heart are often produced which can give important clues to the cause of presenting symptoms and signs (McKie et al, 2005). **BJHM**

McKie SJ, Hardwick DJ, Reid JH, Murchison JT (2005) Features of cardiac disease demonstrated on CT pulmonary angiography. *Clin Radiol* **60**: 31–8

Figure 1. a. Computed tomography pulmonary angiogram showing thickened cardiac interventricular septum and posterolateral wall suggestive of hypertrophic cardiomyopathy. b. Left ventricle is thickened but not dilated, suggesting hypertrophic cardiomyopathy.



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