Aortoenteric fistula: clinical case presentation

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ABSTRACT

Aortoenteric fistula (AEF) is a rare but life-threatening condition characterized by abnormal communication between the aorta and any part of the intestinal tract. It has been suggested that primary AEF arises from an abdominal aortic aneurysm, and secondary is caused by an infection, usually consistent with aortic graft. We present a case report of secondary AEF presented with severe bleeding from the gastrointestinal (GI) tract followed by aortic graft infection.

Keywords: Aortoenteric fistula, gastrointestinal bleeding, abdominal aortic aneurysm, aortic graft

INTRODUCTION

There are many reasons for bleeding from the GI tract, and it is essential not to miss an uncommon cause such as AEF. It is a pathological communication between the aorta and any part of an intestinal tract (1). AEF is an uncommon but life-threatening condition with an incidence rate of 1.6 - 4% (2). This pathology was described for the first time by Sir Ashley Cooper in 1818 (3). AEF is associated with diagnostic challenges - it requires careful attention to a patient's history and relies on clinical acumen (4).

There are two different types of AEF – primary and secondary, depending on their etiology. Primary AEFs commonly arise from an abdominal aortic aneurysm (AAA), and secondary is a complication of reconstructive surgery of an AAA (2 - 6).

The immediate diagnosis and urgent surgery is the only way to save a patient. Otherwise, the mortality of untreated pathology reaches almost 100% (1).

We report a rare case of a secondary AEF followed by abdominal aortic graft infection, presented with GI bleeding. Our purpose is to raise awareness of this catastrophic condition.

CASE REPORT

A 72-year-old man presented to our hospital to the emergency department with general weakness, vomiting of blood, and black tarry stools for the last 24 hours. On the day of admission, his vitals were normal. His past medical history included primary arterial hypertension and heart failure. Six years earlier, the patient was diagnosed with an AAA and undergone treatment with an aortic graft. The initial examination was unremarkable. The digital rectal examination revealed melena. Initial laboratory tests showed haemoglobin level 117 g/l, white blood cells (WBC) count 7,5 x 109/l, platelet count 217 x 109/l, prothrombin time 33,4 seconds, international normalized ratio (INR) 1,14, C – reactive protein (CRB) 5 mg/l, creatinine 85 µmol/l and urea 14,91 mmol/l. Electrolytes were normal.

In the emergency room, esophagogastroduodenoscopy (EGDS) was performed immediately due to melena. EGDS showed bleeding from the lower part of the duodenum. However, there was no possibility to stop the bleeding during the examination.

Later the patient became hemodynamically unstable (blood pressure 70/30 mmHg)

. Repeated laboratory investigation showed a hemoglobin level decrease 97 g/l, INR 1,28, CRB 5 mg/l. In the department of intensive care, two units of packed red blood cells, and two units of fresh frozen plasma were transfused.

Due to the history of AAA repair, computer tomography aortography (CTA) was performed urgently for a potential life-threatening secondary AEF. CTA revealed adhesion between the aortic graft distal part, near the anastomosis, and



Figure 1. CTA coronal view- contrast media extravasation in the duodenum



Figure 2. CTA sagittal view- communication between aorta and duodenum



Figure 3. CTA axial view- contrast media extravasation in the duodenum



Figure 4. CT with contrast media after AEF closing surgery - air in the aortic graft, perigraft infiltration

duodenum. There was enhanced blood in the duodenum, indicating communication between the aorta and the intestinal tract (Figure – 1, 2, 3). CTA undoubtedly helped facilitate the diagnosis of AEF.

The patient was shifted to another hospital for further treatment of the vascular surgery unit. During the surgery, a suppurative aortic graft and 1 cm defect in the duodenum were found. The graft was resected, and axillobifemoral bypass surgery was performed, the AEF was occluded. Unfortunately, after some days, the patient had the following complication – sepsis caused by E.coli occurred, which was correctly treated, and the patient remained alive.

DISCUSSION

AEFs are divided into two types - primary and secondary. According to statistics, the incidence rate of secondary AEF is approximately 2,5 times more common than primary (3, 4, 6).

Primary AEFs commonly arise from an AAA of which 85% are atherosclerotic (3, 5, 6), and it occurs when an erosive aortic segment opens into the adjacent gastrointestinal lumen (4). Rare known conditions related to primary AEF are tuberculosis, syphilis, infection, cancer, foreign bodies, and collagen vascular disease (2, 3, 6). Even the case of vertebral osteophyte has also been shown to influence the development of an AEF (7).

Secondary AEF is a complication of reconstructive surgery of an AAA, involving open repair surgery and endovascular treatment, as well as vascular grafts (2, 4). It is more common in patients with a history of open aortic repair comparing with patients after endovascular stent placement. An abnormal communication can develop between the aorta and any part of the intestinal tract. An estimated 80% of secondary AEFs affect the duodenum, mostly the third and fourth parts (the horizontal and ascending duodenum) and the proximal suture line of the aorta (3), just as in our case. The involvement of the other gastrointestinal segments are less frequent; for instance, aortocolonic fistulae occur only 5 to 6% of all cases (4).

MacDougall L. et al., in the article 'Aorto-enteric fistulas: a cause of gastrointestinal bleeding not to be missed,' says that the pathogenesis of this disease has not yet been fully understood (3), but there are two theories. The first theory suggests that fistula formation is caused by repeated mechanical trauma between the pulsating aorta and duodenum, and the second asserts lowgrade infection as the primary event with abscess formation and subsequent erosion through the bowel wall. The second theory is the most likely because the majority of grafts show signs of infection at the time of bleeding, and approximately 85% of cases have blood cultures positive for enteric organisms (5). In our case, there was the graft infection caused by E. coli positive culture, which also applies the second theory.

AEF is characterized by the classical triad: abdominal pain, gastrointestinal blood loss, which can be acute or chronic, and pulsating abdominal mass (1, 8, 9). However, this triad is only found in 11 - 38,5% patients, which makes diagnosis even more challenging (1). Abdominal pain can occur only in 35% of patients, pulsating mass in 25% patients, and the most frequent gastrointestinal bleeding presents in 94% cases, as in our case. In addition to severe bleeding, significant hemodynamic instability often occurs (10). Other symptoms consistent with this pathology may be intermittent back pain, fever, sepsis, weight loss, and syncope (1). Our patient presented with melena, haematemesis, and general weakness.

Commonly used diagnostic methods for AEFs are abdominal CT with intravenous contrast, interventional angiography, and EGDS (3). The detection rates for each of these modalities are 61%, 26%, and 25%, respectively (6). According to Chick JFB et al. in the article 'Aortoenteric fistulae temporization and treatment: lessons learned from a multidisciplinary approach to 3 patients', CT angiography is the first-line imaging modality for the detection of aortoenteric fistula and has a reported sensitivity of 94% and specificity of 85% (8).

Concerning CTA findings, active extravasation of contrast media in the GI tract is reported the most often, any part of intestines are seen in close contact with an AAA or an aortic graft, there is often fat infiltration around the aortic graft, consistent with infection. Just after secondary AEF closing, CT findings such as fluid, ectopic gas, and per graft soft tissue edema can be normally seen (Figure 4). However, 3 - 4 weeks later, any ectopic gas is abnormal means perigraft infection and possibly fistulization to a GI tract. In 2 - 3 months after surgery, the perigraft soft tissue thickening, hematoma, or fluid should be resolved (3).

The main goals of treatment are control of bleeding and revascularization, repair of intestinal defects, and eradication of related infection. In this case, surgical intervention is performed, and antibiotics are supplied (1, 3). The treatment has been improved for many years. Despite numerous surgical techniques, many patients do not survive or may remain weak after surgery. Survival depends on the onset of bleeding severity and how quickly the operation is performed. Mortality rates range from 24 to 45,8% (2), and up to 100% if untreated (3). That is why it is so vital to suspect and diagnose the rare pathology as fast as it is possible.

CONCLUSION

Our case report is a reminder for doctors that secondary AEF should be strongly suspected in all patients with a prior history of aorta repair presenting with GI bleeding. Urgent diagnosis of AEF is vital and may save a patient from the catastrophic outcomes.

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