## CONTINUING MEDICAL EDUCATION ΣΥΝΕΧΙΖΟΜΕΝΗ ΙΑΤΡΙΚΗ ΕΚΠΑΙΔΕΥΣΗ

## Vascular Diseases Quiz - Case 3

A 59 year old male was admitted to our Hospital due to fever and repeated episodes of hematemesis and melena. Past medical history was remarkable for horseshoe kidney and an abdominal aortic aneurysm (AAA) that was repaired successfully with open surgery 15 months before. Laboratory investigation revealed

microcytic anemia with Ht 29%, Hb 9.1 g/dL, WBCs  $19.7 \times 10^9$ /L, platelets  $200 \times 10^9$ /L, C-reactive protein 59.6 mg/L, erythrocyte sedimentation rate (ESR) 100 mm/hr, blood urea nitrogen 39 mg/dL and creatinine 1.2 mg/dL. Colonoscopy was not notable, however upper gastrointestinal endoscopy revealed a small mucosal ulceration in the distal duodenum. A contrast enhanced CT scan demonstrated perigraft inflammatory tissue around the proximal portion of the aortic graft without gas bubbles (fig. 1).

## Comment

Based on patient's clinical signs, medical history, laboratory and imaging investigation, a secondary aortoenteric fistula due to AAA surgical repair was suspected and therefore the patient was transferred to the operating room for exploratory laparotomy. At surgical exploration a diffuse inflammatory process of the periaortic retroperitoneal tissues was found, while the duodenum was strictly adherent to the aorta. Further dissection revealed a small aortoduodenal fistula between the 3rd portion of the duodenum and the anterior proximal wall of the aortic graft. Despite increased technical difficulties owing to the horseshoe kidney, the fistula was resected, the aortic graft was excised and the aortic stump was ligated distal to the renal arteries. Distal circulation was restored with an axillobifemoral bypass graft, while the duodenal defect was repaired appropriately. The patient had a complicated postoperative course with an acute myocardial infarction and thrombosis of the left femoro-femoral bypass graft that led to an amputation above the knee.

Secondary aortoenteric fistula (AEF) should always be suspected in a patient with a history of aortic reconstructive procedure with prosthetic graft that presents with gastrointestinal bleeding. Such a complication used to be frequent enough in the past, occurring in up to 10% after surgical aortic reconstruction. However with improvement in operative techniques, the incidence of secondary AEF has declined to 0.4–2.4%. The pathogenesis of secondary AEF includes duodenal erosion secondary to inflammatory foreign body response of the adjacent pulsating noncompliant prosthesis. Subsequently, bacteria and digestive enzymes imbathe the graft, resulting in graft infection, retroperitoneal inflammation, and finally formation of an AEF.

The time interval between the initial aortic reconstruction and the onset of gastrointestinal bleeding is usually 2 to 4 years after graft placement, however it may range from 2 days to 15 years. The classic triad of symptoms associated with AEF includes gastrointestinal bleeding, sepsis and abdominal pain. Gastrointestinal hemorrhage including hematemesis, hematochezia or melena represents the initial symptom in more than 70% of AEFs. Chronic anemia is also common. Most often, patients present with a "herald bleed", manifesting as limited, hemodynamically insignificant bleeding episodes over several hours to days, since the aortoenteric communication intermittently opens and seals with blood clot.

Massive gastrointestinal hemorrhage is inevitable with a great variance of time interval. In 90% of the patients however, there is

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**Figure 1.** Contrast-enhanced abdominal CT scan demonstrating perigraft inflammatory tissue around the proximal portion of the aortic graft (black arrow). Note the horseshoe kidney (white arrow).

sufficient time for a complete diagnostic workup before the major hemorrhage occurs.

Basic diagnostic workup in case of suspected AEF in hemodynamically stable patients includes upper gastrointestinal endoscopy and contrast-enhanced CT scan. Esophagogastroduodenoscopy may reveal external compression of the duodenum, punctuate mucosal ulcerations and bleeding from the distal duodenum wall, while in 30% of secondary AEFs, the aortic graft can also be seen. CT findings indicative of AEF include perigraft fluid collection, gas bubbles, leakage of oral contrast medium, pseudoaneurysm formation at the proximal anastomosis and thickening of adjacent bowel wall. Although our patient suffered form an AEF, none of the above signs was apparent, but perigraft inflammatory tissue was demonstrated on CT scan. Consequently, it should be highlighted that absence of the typical pathognomonic signs at CT scan does not preclude the possibility of AEF and only exploratory laparotomy can definitely exclude or confirm the existence of an AEF.

AEF is associated with significant morbidity and mortality despite appropriate treatment. The latter is traditionally surgical and aims to control the hemorrhage, eradicate the associated infection, repair the bowel defect and restore the distal circulation.

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