Endograft preservation during aortoenteric fistula management after EVAR

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INTRODUCTION

Aortoenteric fistula (AEF) is a relatively rare condition with high morbidity and mortality.[1] It is defined as a pathologic communication between the aorta and the gastrointestinal tract, usually the third and fourth segment of the duodenum due to their close proximity.[1] AEF can be classified into primary, were they occur de novo, with an annual incidence of 0.007 per million and secondary, following aortic reconstructive surgery and an annual incidence of 0.6-2%.[2] The occurrence of secondary AEF is found to be higher in patients following open abdominal aortic aneurysm (AAA) repair,[2] whereas only few cases of AEF after endovascular aneurysm repair (EVAR) have been reported in the existing literature.[3] The clinical manifestation with the classic triad of gastrointestinal bleeding, abdominal pain and palpable mass is found in only 11% of patients.[3] On the other hand, herald bleeding and clinical signs of sepsis are more common with computed tomography angiography (CTA) being the necessary modality for diagnosis and evaluation with variable sensitivity (40-90%) and specificity (33-100%).[2]

This is a single center experience of two patients who developed AEF post EVAR and treated with open repair without removing the stent graft.

CASE REPORT

The first case is a 69-year-old man, with a past medical history of diabetes, dyslipidemia and an episode of cerebral ischemic attack. He presented to the Emergency Department of his district hospital with symptoms of vomiting and fever for the past 5 days and an episode of loss of consciousness. He had undergone EVAR (Excluder device, GORE), 23 days ago, for an infrarenal (5.4cm max diameter) AAA (Figure 1).

On clinical examination, he had mild abdominal tenderness and a body temperature of 39°C, with normal vital signs. His initial blood tests revealed a baseline anemia (Hgb 8.3 gr/dl), and elevated inflammation markers (WBC 13.8x10^3/μL, Neutr. 81.7%, CRP 189.1 mg/dl). Due to his recent EVAR, the patient was referred and transferred to our hospital. A CTA was performed which showed soft tissue thickening and edema around the aneurysmal aortic wall along with presence of air inside the aortic sac and extravasation of intravenous contrast medium into the bowel lumen, verifying the diagnosis of AEF (Figure 1).

Given the above findings, open surgical intervention was decided. Under general anesthesia and midline abdominal incision, an AEF was identified between the second part of the duodenum and the anterior wall of the aneurysmal sac. After surgical debridement, the duodenum defect was sutured by layers. The aneurysmal sac was opened with no signs of graft migration or endoleak. After thorough ablution of the graft and the abdomen, the aneurysm sac was partially closed and part of the omentum was mobilized and placed inside the aortic sac in close proximity with the stent graft. The patient resuscitated well and transferred to standard surgical care unit where he had an uneventful recovery. Postoperatively, parenteral nutrition was provided until the 8th postoperative day. Intraoperative cultures came back positive for Enterococcus faecalis and faecium, so he was treated with targeted antibiotic therapy. Patient was discharged on the 11th postoperative day under long-term antibiotics intake (Ciprofloxacin 500 mg twice daily). In the 6-month-follow up, the patient was in a good general condition, inflammation markers were back to normal and on CTA, abdominal aorta had a maximum diameter of 3.4cm, with no evidence of endoleak or inflammation (Figure 2). Taking into account clinical, radiological findings and lab tests, antibiotics were discontinued. One year follow up with CTA had similar findings (Figure 2) and the patient was asymptomatic.

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ISSN 2732-7175 / 2021 Hellenic Society of Vascular and Endovascular Surgery Published by Rotonda Publications

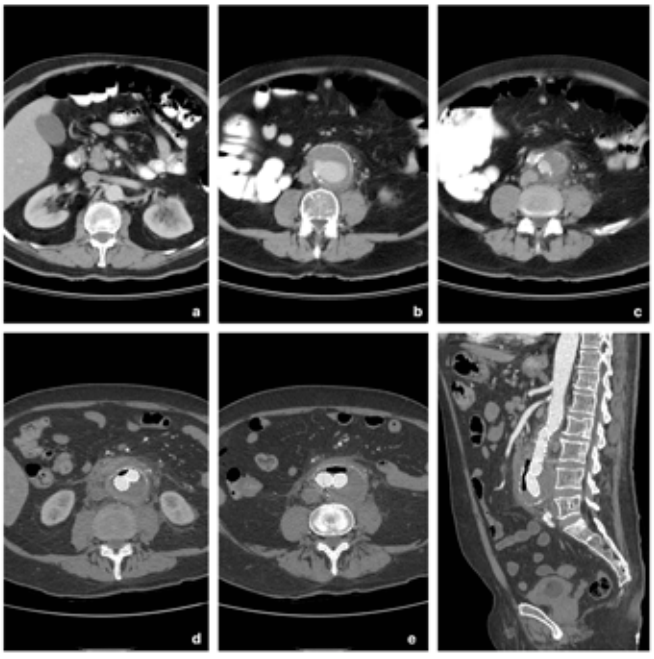

Figure 1. 1st case, a,b,c: Preoperative CTA d,e,f: CTA 23 days postoperative

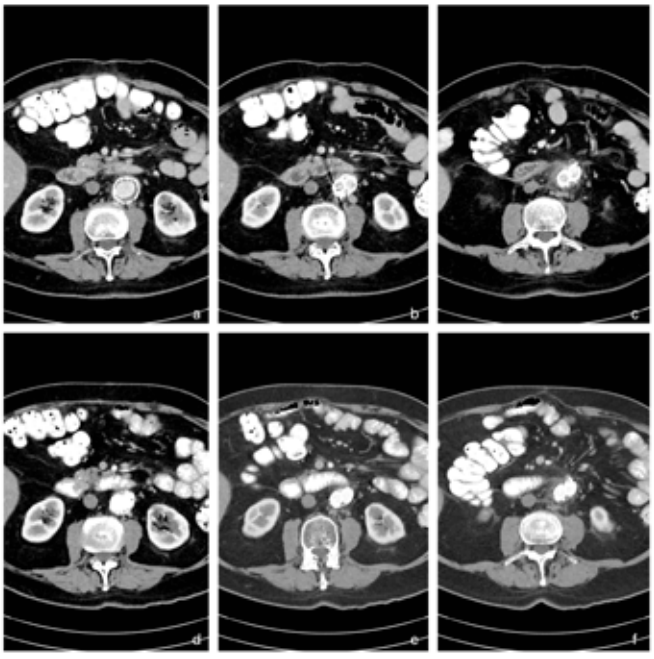

Figure 2. 1st case a,b,c : 6 months follow up CTA d,e,f: 12 months follow up CTA

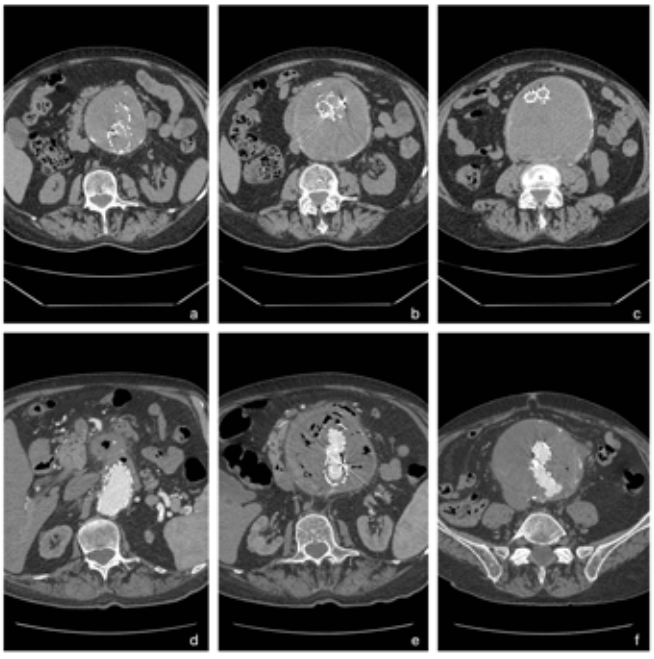

Figure 3. 2nd case a,b,c: CT 8 months prior to onset of symptoms d,e,f: CTA indicating AEF

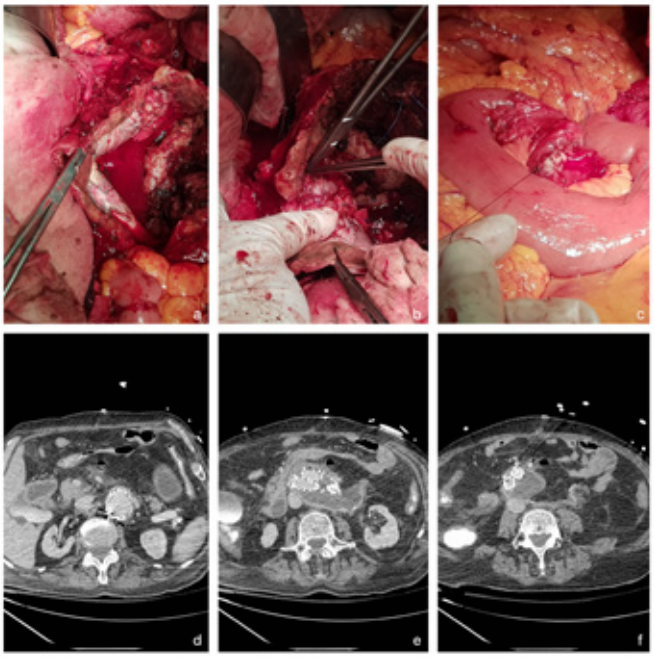

Figure 4. 2nd case, a: Endograft inside the aortic sac, b: Fixating sutures of aortic neck and stent graft, c: Duodenum repair d,e,f: CTA 10th postoperative day

The second case is a 77-year-old man, with a past medical history of hypertension, dyslipidemia and obstructive pulmonary disease who had undergone EVAR due to a ruptured AAA on 2008 (Talent device, Medtronic). On 2014 due to graft migration and type Ia endoleak, an Endurant (Medtronic) stent graft was implanted. He was referred to our hospital with symptoms of mild lumbar pain and episodes of fever for the past 15 days. His blood tests on admission showed abnormal inflammation markers (WBC 10,7 10^3 /μL, Neutrophiles 82.3%, CRP 223 U/L) and renal impairment (Cr. 2.26 mg/dL). His CTA revealed a 14.4 cm infrarenal AAA with Ib endoleak from the left iliac limb, air in the aortic sac, and signs of communication between the duodenum and aortic sac (Figure 3).

An open repair maintaining the stent graft was decided, however prior to that, under local anesthesia and bilateral common femoral approach, two Excluder (Gore) limbs were deployed, treating the endoleak and securing the distal graft fixation. The following day, the patient underwent open repair with similar approach to the first patient. The AEF was dissected with primary closure of the duodenum and the aortic sac was opened. Fixating sutures where placed between the graft and the aortic neck and omentum was placed inside the aortic sac which was partially closed (Figure 4). Patient was transferred to standard surgical care unit where he had a normal postoperative course. However, on the 9th postoperative day he suffered an acute episode of respiratory distress which led to respiratory and cardiac arrest. The patient was resuscitated, intubated and transferred to ICU. A CT thorax abdomen scan (Figure 4) and bronchoscopy was performed which led to diagnosis of aspiration pneumonia. Unfortunately, the patient passed away on the 27th post-op day in ITU with no signs of peritonitis or complications related to his aneurysm.

DISCUSSION

Primary AEF arise in patients with no prior surgery, and it mainly develops due to an atherosclerotic aneurysm of the abdominal aorta or penetrating atherosclerotic ulcer.[4] More rare causes include inflammatory or mycotic aneurysm, radiation, malignancies and trauma.[5] Secondary AEF has been suggested to be the result of perigraft infection and pressure of the graft upon the bowel, that takes place usually during a long post-operative timespan, from the early to the extended post-operative period.[4] Risk factors include emergency surgery for ruptured aneurysm, intra-operative complications such as bowel injury, and continuous aortic pulsation upon the bowel wall due to endoleaks or stent migration.[5] AEF post EVAR is quite rare with only 52 cases reported by 2016.[6]

In the first case presented here, it is possible that this was a primary AEF. Retrospective re-evaluation of the pre-operative CTA scan might indicate an inflammatory AAA, as one can identify periaortic wall thickening and perianeurysmal fibrosis (Figure 1). Moreover, the appearance of the fistula only 23 days after EVAR leads to speculation that erosion of the enteric wall might have started before EVAR. To our knowledge, the shortest interval between EVAR and AEF formation is 1 month (6). Regarding the second case it is worth mentioning that his latest CT scan 8 months prior of the onset of his symptoms, although with no contrast due to his renal impairment, did not reveal any signs of AEF (Figure 3).

Due to its rare occurrence there is no greater consensus for treatment of AEF. Conventional surgical management includes resection of the fistula, graft excision in case of secondary AEF, and revascularization through extra-anatomic bypass or in situ replacement using donor cryopreserved aorta, vein allograft or even synthetic graft with antiseptic properties.[7] All these different treatment modalities are associated with quite increased in-hospital mortality and morbidity. Management of the intestinal part of fistulas has been demonstrated to have acceptable outcomes with simple bowel repair, although resection may be necessary for certain patients.[7] Although an endovascular approach for AEF, have only been considered as a bridging therapy to open repair, in unstable patients with hypovolemic shock and severe sepsis, there have been quite a few reported cases of EVAR as definite treatment with prolonged, targeted antibiotic therapy.[8] According to a recent pooled analysis, EVAR was associated with a significantly reduced in-hospital mortality (7.1%) when compared to open surgery (33.9%), a difference which disappeared 18-24 months postoperatively.[4] The above findings are consistent with previous studies, with sepsis being the most frequent and main cause of recurrence and overall mortality.[9]

In the aforementioned cases, taking into consideration the patients clinical condition and comorbidities, open repair was decided for the resection of the fistula, but without explantation of the graft. A comparison of explantation of endovascular and open grafts due to AEF, showed significant difference in mortality and morbidity (37.5% vs 0% and 50% vs 40% respectively) between these two types of grafts in the short term, 30 days postoperatively.[5] By preserving the endoprothesis, patient can avoid suprarenal aortic cross-clamping and therefore renal ischemia, long operating time and ICU stay. Furthermore, the omentum rapping has proven to be quite protective for re-infection of prosthetic materials.[10]

CONCLUSION

AEF is a relatively rare complication but with high morbidity and mortality. With the increasing use of EVAR, the occurrence of secondary AEFs may increase in the future. Therefore, vascular surgeons should have increased awareness for early diagnosis and treatment. The standard of care for these cases remain open repair and graft explanation. Endograft preservation could be used as a bridging technique or as definitive treatment in high-risk fragile patients.

Acknowledgement: None

Conflict of interest: None

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