Aorto-enteric Fistula After Endovascular Abdominal Aortic Aneurysm Repair: Case Report and Review

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Abstract  Objective: To report a case and to review previous publications regarding the rare complication of aorto-enteric fistula following endovascular aortic aneurysm repair.
Methods: We report the case of a stent-graft infection secondary to an aorto-enteric fistula 14 months after uncomplicated endovascular treatment of an infra-renal aortic aneurysm.
Results: The surgical treatment involved the removal of the infected graft and in situ aortic replacement by cryopreserved allograft. There have been no major complications noted during the 2-month follow-up after surgery.
Conclusions: An aortojejunal fistula is a possible long-term complication of endovascular treatment of abdominal aortic aneurysm. An explantation of the infected graft and aortic replacement by a cryopreserved allograft is a valuable surgical treatment.

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Endovascular repair is an efficient and safe technique for abdominal aortic aneurysm (AAA) treatment. The classical complications after endovascular management are endoleaks, graft migration, aortic rupture, aortic thrombosis and endotension. Infections and aortoduodenal fistulas have been rarely reported.

We report the case of a late stent-graft infection secondary to an aorto-enteric fistula 14 months after uncomplicated endovascular treatment of an infra-renal aortic aneurysm.

Case Report

In October 2007, a 67-year-old man, with an AAA, previously treated by endovascular stent-graft implantation in August 2006, was admitted as an emergency case at our institution complaining of fever and lumbar pain. His past medical history included severe coronary artery disease, obesity, a gastric ulcer and a deep vein thrombosis. A computed tomography (CT) scan, performed for thoracic pain in April 2006, demonstrated an infra-renal AAA which
was subsequently repaired with a bifurcated aortic stent-graft (28 × 12 × 12 mm (Cook aortic intervention)) without initial complication. The follow-up CT scans performed at 3, 6 and 12 months after the procedure demonstrated a decrease in the size of the aneurysm sac without endoleak, migration or kinking of the stent graft (Fig. 1A).

At the time of emergency admission, a CT scan showed an increase in the size of the AAA along with inflammatory changes (Fig. 1B and C). Effacement of the peri-graft fat planes and retroperitoneal fluid accumulation were also demonstrated. There was no evidence of endoleak, migration or fracture of the stent graft. A CT scan with oral contrast showed a close contact between the bowel and the aneurysm and air in the aneurysmal sac (Fig. 1D and E). A CT-guided aspiration yielded purulent material. Cultures of the aspirate revealed *Bacteroides fragilis* (an enteric organism). Endoscopy was performed, but findings were unremarkable.

A diagnosis of endovascular stent-graft infection secondary to an aorto-enteric fistula was suspected. Laparatomy confirmed a close adhesion between the aneurysmal sac and the first part of the jejunum (Fig. 2A). After supraceliac control of the aorta, the aneurysmal sac was opened (Fig. 2B). It was full of purulent material that was collected for bacteriological examination. The explantation of the stent graft was difficult due to the suprarenal barb fixation at the level of the ostia of the superior mesenteric artery and of the renal arteries. All the Dacron was removed, but a part of the proximal bare stent had to be left in place at the level of the ostia of the superior mesenteric artery and the right renal artery. Ulceration of the wall of the first part of the jejunum was seen without finding any active fistula (Fig. 2C). Aorto-iliac continuity was restored by an *in situ* composite cryopreserved allograft, using a thoracic aorta and an aorto-iliac bifurcation (Fig. 2D). The region was drained and the greater omentum was interposed between the jejunal and aortic walls. A discharge gastrostomy and a feeding jejunostomy were performed. The patient was also treated by intravenous antibiotic therapy (amoxicillin, clavulanic acid and metronidazole) for 21 days. The postoperative course was uncomplicated, and the patient was discharged home after 2 weeks. The follow-up has been uneventful for 2 months after surgery. A CT scan demonstrated a decrease of the aneurysmal sac size and the absence of endoleak or inflammatory signs (Fig. 3).

**Discussion**

Secondary aorto-enteric fistula is a rare but well-known complication after open AAA repair, occurring in 0.4–1.6% of cases. The incidence of aorto-enteric fistulas after endovascular aneurysm repair is poorly defined, with only a few cases reported in the recent medical literature (Table 1). To our knowledge, only 15 cases have been described since 1998.

**Clinical presentation**

Diagnosis of secondary aorto-enteric fistula can be challenging. Classically, the patient complains of abdominal or back pain, nausea, vomiting and gastrointestinal bleeding. Most often, the clinical presentation is less typical, in keeping with infection, and the patient is admitted with fever, weight loss or septic shock, with no gastrointestinal haemorrhage. Sometimes, symptoms arise from septic embolism from an infected graft with non-specific events, such as septic arthritis, that make diagnosis more difficult.

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**Figure 1** Preoperative CT scan. (A) Six months after EVAR: decrease in the aneurysmal sac size. (B and C) Fourteen months after EVAR: increase in aneurysmal sac size and inflammatory changes (white arrow): effacement of the peri-graft fat plane and retroperitoneal fluid accumulation. (D) Fourteen months after EVAR: adhesion between the aneurysmal sac and jejunum (yellow arrow). (E) Fourteen months after EVAR: air in aneurysmal sac (yellow arrow).
Figure 2  Perioperative view. (A) Adhesion between the aneurysmal sac and the first portion of the jejunum (white arrow). (B) Endovascular stent graft after aneurysmal sac opening. (C) Ulcerations of the wall of the jejunum (white arrow) and of the aneurysm (yellow arrow) after dissection. (D) In situ cryopreserved allograft.

Figure 3  Postoperative CT scan. Decrease of the aneurysmal sac size.
Furthermore, a CT scan confirms diagnosis in only 33–80% of cases, and endoscopy, rarely performed in the absence of gastrointestinal bleeding, does not exclude diagnosis if no fistula is seen. Most often, diagnosis is made on surgical exploration. In our case, the patient was admitted with a septic syndrome and no abdominal symptoms were reported. Diagnosis was suspected on CT scan and confirmed by surgical operation. Endoscopy did not contribute to the diagnosis. In previous cases reported, aorto-enteric fistulas occurred from 4 to 58 months after aneurysm repair.

Aetiology

Diagnosis is also made more difficult due to the multiple mechanisms leading to formation of aorto-enteric fistula. The most frequently reported mechanism is aortic wall erosion by a stent. In two instances, stent migration and kinking were implicated. In the first case, reported by Hausegger et al., a migrated and kinked stent graft had eroded the aneurysmal wall and caused secondary aorto-enteric fistula. In a second case, migration of the stent graft caused by enlargement of the upper neck of the aneurysm led to secondary fracture of the metallic structure and kinking of the stent graft that was associated with formation of aorto-enteric fistula. A number of further cases of aorto-enteric fistulas have been associated with coil embolisation of endoleaks. Alankar et al. reported a case of aorto-enteric fistula associated with rupture of the aneurysm secondary to a type I endoleak. In this case, endoleak was associated with an increase in the size of the aneurysm that led to an erosion of the wall of the duodenum. In the other two cases, Elkouri et al. and

### Table 1 Aorto-enteric fistulas after endovascular repair of aortic aneurysm in literature. EVAR: endovascular aneurysm repair; AEF: aorto-enteric fistula.

<table>
<thead>
<tr>
<th>Date</th>
<th>Author</th>
<th>Clinical presentation</th>
<th>Time after EVAR (months)</th>
<th>Aetiology of the AEF</th>
<th>Vascular reconstruction</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 1998</td>
<td>Norgren</td>
<td>Abdominal pain, digestive haemorrhage</td>
<td>17</td>
<td>Peri-aortic inflammatory mass and ruptured graft</td>
<td>Aortobiiliac (PTFE graft)</td>
<td>Alive at 6 months</td>
</tr>
<tr>
<td>2 1999</td>
<td>Hausegger</td>
<td>Abdominal pain, digestive haemorrhage</td>
<td>18</td>
<td>Migrated and kinked stent graft</td>
<td>Aortobiiliac (Dacron graft)</td>
<td>Alive at 6 months</td>
</tr>
<tr>
<td>3 2000</td>
<td>Janne d’Othee</td>
<td>Digestive haemorrhage, infection</td>
<td>22</td>
<td>Migrated and kinked stent graft</td>
<td>Axillobifemoral</td>
<td>Alive at 40 months</td>
</tr>
<tr>
<td>4 2000</td>
<td>Makar</td>
<td>Abdominal pain, fever, digestive haemorrhage</td>
<td>4</td>
<td>Crohn’s disease</td>
<td>None (antibiotic only)</td>
<td>Died</td>
</tr>
<tr>
<td>5 2001</td>
<td>Ohki</td>
<td>Digestive haemorrhage</td>
<td>9</td>
<td>Unknown</td>
<td>None (open conversion)</td>
<td>Died</td>
</tr>
<tr>
<td>6 2001</td>
<td>Ohki</td>
<td>Infection</td>
<td>30</td>
<td>Unknown</td>
<td>None (trans-abdominal drainage)</td>
<td>Died</td>
</tr>
<tr>
<td>7 2001</td>
<td>Parry</td>
<td>Septic arthritis, digestive haemorrhage</td>
<td>6</td>
<td>Peri-aortic inflammatory mass</td>
<td>Aortobiiliac (silver-impregnated Dacron graft)</td>
<td>Alive at 7 months</td>
</tr>
<tr>
<td>8 2002</td>
<td>Kar</td>
<td>Infection</td>
<td>20</td>
<td>Unknown (endotension?)</td>
<td>Aortobiiliac (rifampicin-soaked Dacron graft)</td>
<td>Alive at 1 year</td>
</tr>
<tr>
<td>9 2003</td>
<td>Alankar</td>
<td>Abdominal pain</td>
<td>4</td>
<td>Endoleak</td>
<td>Aortobiiliac (rifampicin-soaked Dacron graft)</td>
<td>Alive at 6 months</td>
</tr>
<tr>
<td>10 2003</td>
<td>Elkouri</td>
<td>Digestive haemorrhage</td>
<td>17</td>
<td>Endoleak coil embolisation</td>
<td>Axillobifemoral</td>
<td>Died at 12 h (septic shock)</td>
</tr>
<tr>
<td>11 2003</td>
<td>Bertges</td>
<td>Infection, vomiting</td>
<td>53</td>
<td>Endoleak coil embolisation</td>
<td>Axillobifemoral</td>
<td>Alive at 1 month</td>
</tr>
<tr>
<td>12 2003</td>
<td>Abou-Zamzam</td>
<td>Infection</td>
<td>11</td>
<td>Unknown (endotension?)</td>
<td>Axillobifemoral</td>
<td>Alive at 4 months</td>
</tr>
<tr>
<td>13 2004</td>
<td>French</td>
<td>Digestive haemorrhage</td>
<td>16</td>
<td>Stent-graft infection</td>
<td>Axillobifemoral</td>
<td>Died at 6 days (multi-organ failure)</td>
</tr>
<tr>
<td>14 2006</td>
<td>Ghosh</td>
<td>Abdominal pain, digestive haemorrhage</td>
<td>9</td>
<td>Stent-graft infection</td>
<td>None</td>
<td>Died</td>
</tr>
<tr>
<td>15 2007</td>
<td>Ruby</td>
<td>Abdominal pain, digestive haemorrhage</td>
<td>58</td>
<td>Unknown (endotension?)</td>
<td>Aortobiiliac (Dacron graft)</td>
<td>Alive at 13 months</td>
</tr>
<tr>
<td>16 2007</td>
<td>Present</td>
<td>Infection</td>
<td>14</td>
<td>Unknown (endotension?)</td>
<td>Aortobiiliac (allograft)</td>
<td>Alive at 2 months</td>
</tr>
</tbody>
</table>
Bertges et al. described aorto-enteric fistulas occurring after coil embolisation of type II endoleak. In these instances, erosion of the aortic and duodenal walls by coils led to fistulisation.

Inflammation within the wall of the AAA may be a further mechanism underlying fistula formation. In the first case reported in the literature in 1998, Norgren et al. present a case where a non-specific peri-aortic inflammatory mass led to a rupture of the AAA, causing secondary aorto-enteric fistula. Another case of peri-aortic inflammatory mass was described by Parry et al. in 2001; no stent failure was found at surgical exploration, suggesting that local inflammation can lead to fistulisation without involvement of the stent graft. Makar et al. presented a case of Crohn’s disease as a possible cause of aorto-enteric fistula after endovascular AAA repair, without any stent failure.

A further mechanism underlying aorto-enteric fistula may be primary infection of the stent graft, as reported by French et al. in 2003 and by Ghosh et al. in 2006. In some cases, no clear mechanism for fistula formation is apparent. In 2001, two cases were reported by Okhi et al. in a review of complications after endovascular graft repair of AAA, but no details were provided. In the last three cases, no aetiology was found. The hypothesis made by the authors was persistent endotension of the AAA sac, leading to fistulisation without stent failure. In our case, no migration, kinking, fracture or endoleak could explain fistulisation between the AAA and jejunum, and we believe that persistent endotension was involved.

Treatment

The treatment of aorto-enteric fistula complicating endovascular AAA repair is controversial. The explantation of the infected graft and extra-anatomic bypass remains the gold standard. Recent studies suggest that in situ aortic reconstruction using antibiotic-impregnated graft, cryopreserved allograft or autogenous vein is associated with fewer amputation, conduit failure, re-infection and lower rate of early and late mortalities than extra-anatomic bypass, but more data are needed to confirm these results. Surgical treatment must be associated with antibiotic therapy, but no guidelines exist on the exact duration of treatment. In the previous cases described, lower extremity re-vascularisation was performed by axillo-femoral bypass in five cases, and by in situ aortic replacement in six cases (one with polytetrafluoroethylene (PTFE) graft, two with Dacron graft, two with rifampicin-soaked graft and one with silver-impregnated graft). In one case, no surgical treatment was performed, and the patient was treated with intravenous antibiotics only. Management of two patients was not specified. For the last patient, no graft explantation or re-vascularisation was performed as the patient presented with colonic ischaemia and faecal peritonitis. In our case, the patient was successfully treated by explantation of the infected graft and in situ aortic replacement by cryopreserved allograft. Jejunal ulceration protection was provided by omental interposition, gastrostomy and jejunostomy. To our knowledge, this is the first time that a cryopreserved allograft was used for this indication. A cryopreserved allograft was chosen for in situ aortic replacement because it is a safe and effective treatment of major vascular infection, with a better resistance to re-infection compared to prosthetic grafts. The well-known major drawback of this technique is the risk of degeneration of the allograft, leading to complications such as calcification, dilatation or even rupture of the allograft. Immunological reactions seem to be partly responsible for degenerative changes occurring in the allograft wall. There is no evidence that immunosuppressive treatment prevents these long-term complications. Furthermore, immunosuppressive therapy was not reasonable in the setting of severe peri-prosthetic infection. As the incidence of complications is expected to increase with time, follow-up has to be close and lengthy, with regular screening, at least annually, by thoraco-abdominal CT scan or magnetic resonance (MR).

Conclusion

Aorto-enteric fistula is a rare but severe complication occurring after endovascular AAA repair that is difficult to diagnose and treat. Explantation of the infected graft and aortic replacement by cryopreserved allograft is a valuable surgical treatment.

Conflict of Interest

None.

References


