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CASE REPORT

Aneurysm Sac Enlargement After Conventional Inflammatory Aneurysm Repair with a Polytetrafluoroethylene Aortobiiliac Graft

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Introduction

Collections of serous fluid around prosthetic grafts can be caused by infection or ultrafiltration of serum. Making the distinction is often troublesome. Perigraft seroma without proven infection has been described in several reports, especially in grafts for haemodialysis and grafts in extra-anatomic positions.[1,2] After conventional abdominal aortic aneurysm (AAA) repair by interposition of an artificial graft, the aneurysm sac will usually be closed around the graft to prevent adhesions between graft and bowel. Postoperative symptomatic enlargement of the sac has recently been described by Williams[3] in one case similar to our own.

Case Report

A 58-year-old man without special risk factors underwent elective repair of a 7.5 cm inflammatory AAA with a stretch aortobiiliac ePTFE prosthesis. The diagnosis of inflammation was made by CT enhancement of the thick aneurysmal wall and the typical intraoperative adhesions. Preoperatively the left obstructed kidney had been drained by an ureteral-catheter. The dense adhesions of the proximal small bowel to the aneurysm wall were not taken down and the neck of the aneurysm was prepared in the suprarenal position by mobilising the left renal vein. The prosthesis was sutured to the neck of the infrarenal aorta and to both common iliac arteries. The aneurysm wall was closed around the prosthetic graft as usual. Antibiotics were given as a single dose prophylactically. The AAA repair was straight forward and the patient's recovery was uneventful.

Routine Duplex 3 months postoperatively showed an aneurysmal sac with a diameter of 4 cm and a patent graft. The left obstructed kidney remained painful and demonstrated no function by scintigraphic examination. The patient had no signs of infection, but a repeat CT scan demonstrated further enlargement of the aneurysm sac with a diameter of 5.5 cm and a wall-thickness of about 1 cm. A large fluid collection surrounded the arterial prosthesis. Calcification at the rim of the cystic mass was interpreted to be the former aneurysm wall, covering the prosthetic graft. The aneurysm wall continously showed inflammatory signs by enhancing contrast. There was no fever or leucocytosis and a decision was made to treat the patient conservatively.

Two months later the patient was experiencing abdominal discomfort with right upper abdominal pain. Ultrasound demonstrated subacute calculous cholecystitis and a severly inflamed gallbladder was removed by open cholecystectomy using a subcostal incision. The recovery was uneventful, but during the following months the patient developed progressive discomfort with mid-abdominal pain and vomiting. A plain abdominal X-ray demonstrated small bowel obstruction. Laboratory tests were normal, including creatinine level, white blood cell count, CRP and cholsterol. Repeat CT revealed further enlargement of

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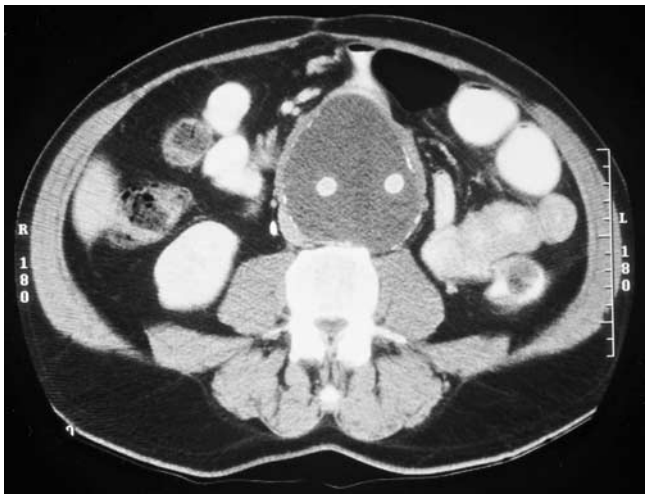

Fig. 1. CT scan 12 months after primary operation demonstrates an inflamed aneurysm sac with a diameter of 10 cm and a patent graft.

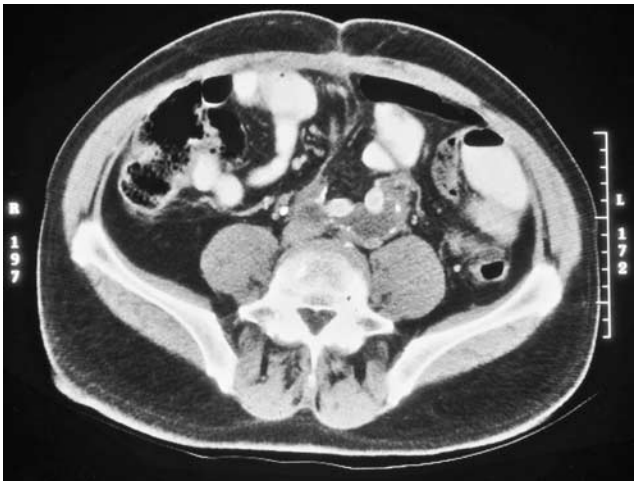

Fig. 2. CT scan 4 months after partial aneurysm sac resection and omental flap covering. There is minor fluid accumulation and the graft is patent.

the inflamed AAA sac to a diameter of 10 cm (Fig. 1).

Because of continued symptoms the patient was reoperated. A very tense pulsatile white coloured aneurysm wall was covered by a distended loop of proximal small bowel stretched out and fixed to the aneurysm sac. The afferent loop to this bowel segment was distended explaining the patient's symptoms. The pulsating mass was punctured and a large volume of straw-coloured, slightly turbid fluid under high pressure was removed. It was not possible to detach the bowel from the aneurysmal wall. The bowel segment had to be resected, together with the anterior wall of the aneurysmal sac. The wall thickness of the sac was 1 cm. The graft was found unattached to any tissues except the anastomoses.

The surface of the graft was covered with patchy white material that was not slimy. This material was removed from the graft and proved to be fibrin without any inflammatory cells on histology. The stiffness of the residual aneurysm wall prevented the direct closure of the sac. An omental pedicle was brought through the mesocolon transversum to cover the entire graft. The aneurysm sac was left open. The recovery was uneventful. All cultures were negative.

The CT scan after 4 months demonstrated minor fluid accumulations and a patent graft (Fig. 2). The patient remained well and is working full time. Duplex scan after 8 months shows no further fluid accumulation.

Discussion

The behaviour of the aneurysmal sac after endoluminal repair attracts increasing interest. Symptomatic aneurysm sac enlargement after conventional open repair is rare. Only one case is reported in the literature.[3] In this case the sac was growing after conventional graft replacement of a non inflammatory aneurysm with an ePTFE aortobiiliac prostheses. A huge perigraft seroma developed without proven infection. Intraoperatively identical anatomical details as described in our patient were found, with the exception of dense adhesions to the small bowel. The aneurysmal wall was partially resected and closed around the prosthesis leaving a central "window". To prevent adhesions sealing the communication between the cyst and the abdominal cavity, an ePTFE "membrane" was sutured to the retroperitoneum and a central donut hole was sutured to the opening left in the aneurysm sac. No perigraft seroma reoccurred until 10 months postoperatively. In our patient the draining of the perigraft seroma into the peritoneal cavity was not possible due to dense inflammatory adhesions of the aneurysmal sac to the small bowel that had to be resected because of stenosis.

The idea of draining a perigraft seroma to the peritoneal cavity was described by Lowery *et al*., 1987.[4] They successfully created a window from a subcutaneous perigraft seroma of an axillofemoral graft into the peritoneum. The most promising procedure to relief perigraft seromas seems to be the removal of the graft and cyst membrane with placement of a new graft of material other than the original along a different anatomic route.[1,2] In case of intra-abdominal reconstructions this "aggressive" procedure means excision of the graft followed by an extra-anatomical axillobifemoral graft. This approach is without doubt justified in infected grafts.

In this case it seemed essential to determine whether the failure of incorporation of the ePTFE graft was due to biofilm infection. Towne *et al*.[5] characterise

biofilm infections as "an absence of systemic sepsis, a fluid-filled cavity surrounding the graft, a draining sinus tract and microorganisms that must be removed from the prosthesis for bacterial cultures. Polymorphonuclear leucocytes (PMN) but not often bacteria are seen on cellular analysis of aspirated fluid." The patient could have had such an infection, but all cultures for *S. epidermatis* within the perigraft collection and aneurysm wall were negative and no PMNs were found within the seroma.

However, there is a strong evidence both clinically and experimentally that ultrafiltration may occur without infection. The aetiology is not fully understood. Sladen *et al*.[6] described an inhibitor of fibroblast growth identified in two patients with perigraft seroma. Normal ingrowth of fibrous tissue resumed after plasmapheresis, leaving the patients clinically stable for up to 3 and 5 years after treatment.

A comparison of the described "endotension" within an excluded aneurysm sac by a perigraft seroma with "endotension" after endovascular repair is not possible because of different pathophysiological mechanisms. At no time did our patient demonstrate any signs of infection; however, he developed a subileus due to an increasing fluid collection within an inflammatory aneurysm sac. If ultrafiltration can occur, and there is ample evidence it can, the presence or absence of acute inflammatory cells in the perigraft fluid may allow us to distinguish between patients who may or may not require graft excision.

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