SHORT REPORT

Non Invasive Treatment of Peri-aortic Inflammation after Endovascular Graft

L.W. Brouw,1 C.T.C. van Weerelt,2 C. van Guldener,3 G.P.J. Geenen4 and L. van der Laan2*

Departments of 1Pulmonary medicine, 2Surgery, 3Internal medicine, and 4Radiology, Amphia Hospital Breda, The Netherlands

Introduction. Peri-aortitis is a rare and severe complication of endovascular treatment.

Report. Two cases of peri-aortic inflammation are reported after receiving an abdominal endoprosthesis as treatment for abdominal aneurysm. Both patients were successfully treated with high doses of prednisone and the grafts were left in situ.

Discussion. High doses of prednisone may be the treatment of choice for post endovascular peri-aortitis.

Keywords: Peri-aortic inflammation; Endovascular graft; Abdominal aneurysm; Retroperitoneal fibrosis; Peri-aortitis.

Introduction

Endovascular repair of abdominal aortic aneurysm (AAA) is a relatively modern technique for which the complications and long term results are still being understood. Randomised trials have shown a lower mortality and lower perioperative complications in selected patients as compared to open surgery. More evidence of superiority to open repair and cost effectiveness is still needed, especially long term results.

New problems with the endovascular technique are emerging including breaking of the attachment hooks, migration of the graft, incomplete exclusion of the aneurysm, graft tearing, perforation or folding, endo leaks, fistulae, late rupture, graft thrombosis and graft infection.1 The need for secondary interventions after endovascular repair with conversion to open repair is associated with a worse outcome if the graft could not be preserved.2

Peri-aortitis is a rare and severe complication of endovascular therapy.

Treatment options include operative replacement of the stent or non invasive medical therapy but there is no consensus. For this reason we reported our experience with the non invasive medical therapy of this complication.

Report

Patient 1 is a 60 year old male who received an endovascular prosthesis for an infra-renal AAA (Fig. 1). Pre-operative findings were unremarkable. Three years after this procedure he presented with severe abdominal pain radiating to the back, malaise and fever. Physical examination showed a perspiring man in pain with abdominal guarding.

Laboratory test included a sedimentation rate of 102 mm/sec, C-reactive protein of 213 mg/l and creatinine of 132 μmol/l. CT-scan was performed to evaluate the abdominal aorta which showed a thickened aortic wall with peri-aortic oedema (Fig. 2). No other pathology was found. The diagnosis of peri-aortitis was made. Six blood cultures were taken to make sure no bacterial infection was causing these problems. The patient received high doses corticosteroids (see discussion) after which the symptoms resolved. A follow up CT scan showed improvement (Fig. 3). The patient recovered with preservation of his graft.

*Corresponding author. L. van der Laan, Department of Surgery, Amphia Hospital Breda, Molengracht 21, 4800 RK Breda, The Netherlands.
E-mail address: lvanderlaan@amphia.nl
Patient 2 is 72 year old male who received an endovascular graft for an AAA (Fig. 4). Pre-operative findings were unremarkable. Three months after being discharged the patient developed back pain and fatigue.

Laboratory test showed a sedimentation $>120 \text{ mm/sec}$, a C-reactive protein of 132 mg/l and creatinine of 125 $\mu$mol/l. A CT scan showed an inflammatory reaction around the bifurcation of the aorta (Fig. 5). Blood cultures remained negative. A leukocyte scan was performed, which showed mild leukocyte accumulation at the location of the aorta bifurcation.

This patient was also diagnosed with peri-aortitis. After reasonably excluding infection this patient received high doses corticosteroids. The patient recovered in just two days (Fig. 6).

Corticosteroids were continued and the patient is still being followed as an outpatient.

**Discussion**

Peri-aortitis after placing of an endoprosthesis is a relatively unknown complication of this new procedure. Peri-aortitis is characterised by a chronic specific retro-peritoneal inflammation.3 There are similarities with retroperitoneal fibrosis. Presenting symptoms are pain in the back, flanks and/or abdomen, malaise and weight loss. The urine flow can be obstructed.3
Laboratory findings show increased sedimentation and other inflammatory proteins. The diagnosis can be made by ultrasound, but often a CT-scan or MRI is necessary.

Differential diagnoses include chronic inflammation from an intra-abdominal focus, surgical trauma, autoimmune response, drugs or malignancy (mainly lymphoma). If there is doubt about the diagnosis a biopsy can be informative. In our cases we chose not to perform a biopsy. Some medications are associated with retroperitoneal fibrosis (methysergide, methyldopa, amphetamines, cocaine and B-blockers), but none so far with peri-aortitis.

It is not known what causes the inflammation, but we presume the graft can trigger an inflammatory response. Treatment should focus on suppressing the inflammation. Prednisone (40–60 mg/day) for 6 weeks, followed by 5–10 mg for 6 months to a year results in improvement of the symptoms in most cases of retroperitoneal fibrosis, inflammatory AAA and peri-aortitis. Steroids have detrimental effects on the immune response which needs to be considered before treatment. Cases of urinal obstruction associated with peri-aortitis have been published for which a J-stent or nephrostomy or surgery may be necessary.

In other causes of retroperitoneal inflammation favourable results from tamoxifen and medroxyprogesterone have been reported. The role of tamoxifen in the treatment of peri-aortitis is unknown. In our cases, corticosteroids where used successfully without the use of tamoxifen.

In conclusion, two patients with peri-aortitis after endovascular AAA repair were successfully treated with non-invasive therapy consisting of high dose corticosteroids.

References


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