ENDOVASCULAR AND SURGICAL TECHNIQUES

Endovascular Repair of an Inflammatory Aortic Aneurysm

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Introduction

Inflammatory aortic aneurysms are characterised by a dense peri-aortic fibrous reaction and a rise in systemic inflammatory mediators. A case of endovascular repair of inflammatory aneurysm is reported with successful aneurysm exclusion and a reduction in systemic markers.

Case Report

A 60-year-old man presented with a 3 month history of abdominal pain, back pain and 10 kg weight loss. Abdominal examination suggested an abdominal aneurysm. A contrast enhanced CT scan demonstrated a 7.3 cm aneurysm surrounded by a rim of soft tissue consistent with a peri-aneurysmal inflammatory response. The left ureter was involved in the inflammatory process with resultant hydronephrosis. Preoperative blood analysis revealed a plasma viscosity of 2.05 (normal <1.73) a C-Reactive protein (CRP) of 3.6 (normal <1.0), and a positive antinuclear antibody titre of 1:64.

The aneurysm was repaired endovascularly using a tapered prosthesis consisting of a Palmaz balloon expandable stent sutured to a pre-expanded PTFE graft as previously described. The patient made an uneventful postoperative recovery and was discharged home on the 7th day. The patient was reviewed at 6 weeks, 6 months and 1 year. CT scanning confirmed aneurysm exclusion, with reduction in AP diameter to 6.7 cm at 1 year. The left hydronephrosis persisted and a double J stent was inserted. At 1 year the plasma viscosity was 1.64 and CRP <1 which confirmed the return to normal levels. The antinuclear antibody titre remained positive at 1:64.

Discussion

Elective conventional surgery for inflammatory AAA is technically demanding and is associated with increased mortality when compared to non-inflammatory AAA. The potential benefits of endovascular repair are therefore likely to be greater in this group of patients, and these preliminary findings suggest that the systemic inflammatory response may be reduced by endoluminal aneurysm exclusion.

The aetiology of inflammatory AAA remains unknown. A bacterial agent is thought to be an unlikely cause. The hypothesis that the aneurysm causes lymph channel obstruction and subsequent lymph extravasation inducing secondary fibrosis is somewhat refuted by this case, in which the inflammatory markers return to normal without a dramatic reduction in the aneurysm size after surgery. It has also been proposed that chronic trauma from aneurysm wall motion may cause fibrotic thickening of surrounding tissues, endovascular aneurysm repair would potentially attenuate this trauma and may explain the reduction in the inflammatory markers. Another theory suggests that increased inflammation and fibrosis occurs around atherosclerotic plaques where the media has been disrupted. Endovascular AAA repair excludes flow from the aneurysm sac and would potentially arrest this process.
Endovascular AAA repair may throw new light on the aetiology of inflammatory aneurysms.

References


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