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SHORT REPORT

Recurrent Abdominal Aortic Aneurysm Secondary to Type II Endoleak

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Introduction

Prevention of aneurysm rupture is the primary aim of the elective treatment of abdominal aortic aneurysm (AAA). The concept of aneurysm recurrence following successful surgical repair is a relatively new concept that has accompanied the development of endovascular repair of AAA (EVAR). Following open repair the chance of aneurysm recurrence was thought to be minimal, except at a later stage when aneurysmal dilatation of aorta can occur above or below the interposition graft. Endovascular aneurysm repair (EVAR) is however complicated by endoleaks and device migration, which can cause persistent perfusion of the aneurysm sac, leading to recurrent AAA. Thus early enthusiasm for endovascular aneurysm repair (EVAR) has been marred by sobering reports of aneurysm sac expansion and rupture despite apparently successful initial treatment.[1] These catastrophes have largely been attributed to graft migration and endoleak.[2,3] Thus regular follow up of patients is needed and EVAR is still in experimental stage. The employment of adjuvant percutaneous radiological techniques such as coil embolisation of a patent inferior mesenteric artery or lumbar vessels, either prior to, or during deployment of an endovascular device, have been utilized in an attempt to reduce the incidence of this complication. However, despite regular follow-up of EVAR cases, interval endoleaks do occur. Many of these can be treated by further endovascular or open surgical intervention. Despite this fatal AAA ruptures still occur after endovascular treatment.

We report a case 18 months post EVAR that, in spite of multiple endovascular interventions aimed at eradicating a type II endoleak arising from persistent back bleed from a lumbar vessel, presented in the interval period as symptomatic aneurysm necessitating urgent laparotomy and open repair of a type II endoleak. In the presence of an intact endovascular graft with good proximal and distal seal, the endoleak was treated without cross clamping the aorta (Figs. 1–4).

Case History

An 83-year-old gentleman had a 6.4 cm AAA discovered during investigation for dyspepsia. He underwent elective EVAR with pre-operative left internal iliac embolisation and a customized Zenith AAA stent used (Wm Cook, Europe, Denmark). The bifurcated device was deployed from the right groin and an articulated iliac limb from left groin. The suprarenal part of the stent was adequately fixed with both renal arteries seen to be patent. A good seal of the right common iliac and left external iliac arteries was produced with no evidence of endoleak at the end of the procedure. A post-procedure CT prior to hospital discharge showed a type II endoleak from a left lumbar artery, which persisted in another CT angiogram done a month later. The lumbar feeding vessel subsequently embolised via the right iliac artery.

A CT after this procedure showed a persistent type II endoleak presumed to be arising from lumbar collaterals, although no clear feeding vessel was

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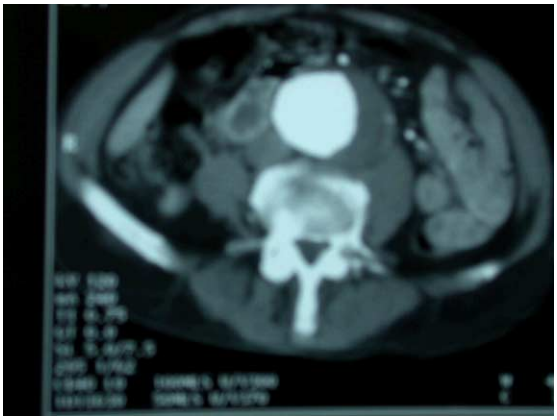

Fig. 1. Preop CT showing 6.4 cm aneurysm sac.

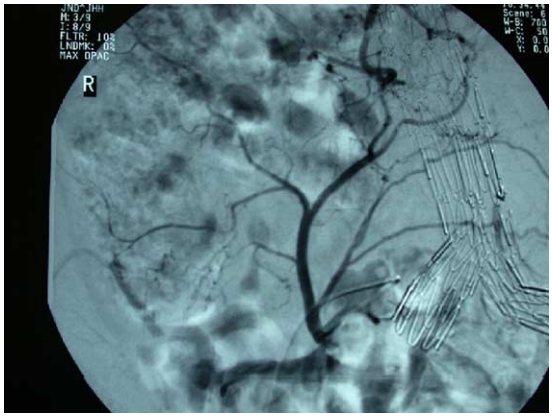

Fig. 3. Embolisation of left lumbar artery.

identified. The sac still measured 6.5 cm so it was decided to embolise the aneurysmal sac. CT guided lumbar puncture of the sac was undertaken and 7.5 ml of thrombin was injected slowly. The pressure measurement at that time showed a reduction of sac pressure from 90/77 to 20/15. Six month follow up CT showed a persisting, but reduced endoleak. At that time the size of the sac measured 6.6 cm.

Further attempts at embolisation of the inferior mesenteric and left lumbar arteries were undertaken: using a 5 French sidewinder 1 catheter placed in the SMA, selective catheterization of the marginal artery was performed with a micro catheter which was taken around to the IMA which was then embolised with a solitary 4 mm diameter coils. Thereafter the catheter was passed down the iliac system and micro catheter was introduced selectively from the internal iliac to the collateral feeding vessels into a lumbar artery.

Despite all this, a repeat CT showed a persistent type II endoleak and it was decided to undertake surgical ligation of the lumbar vessel. However, the patient presented as an emergency with increasing abdominal pain radiating to the back, prior to elective surgical admission. An urgent CT angiogram showed a persistent leak into the aneurysm sac from a lumbar artery. Delayed scans showed an extensive amount of contrast within the aneurysm sac and the left psoas muscle was prominent, suggesting the possibility of a contained rupture.

An emergency open repair was therefore carried out. At operation the aneurysm sac was opened and the leaking lumbar artery oversewn from within the aneurysm sac. The endovascular device was noted to be intact and securely fixed proximally and distally. It was therefore left *in situ* and the sac closed over it. Six week follow-up showed no problems and the patient was well (Figs. 5 and 6).

Discussion

Aneurysm expansion may be regarded as an indicator of failure of aneurysm exclusion following endografting.[4] Although there is good evidence to implicate both type I and III endoleaks and migration in the

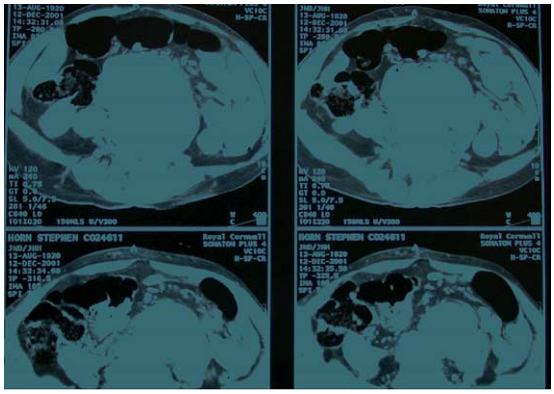

Fig. 2. CT images 1-month postop showing type II endoleak.

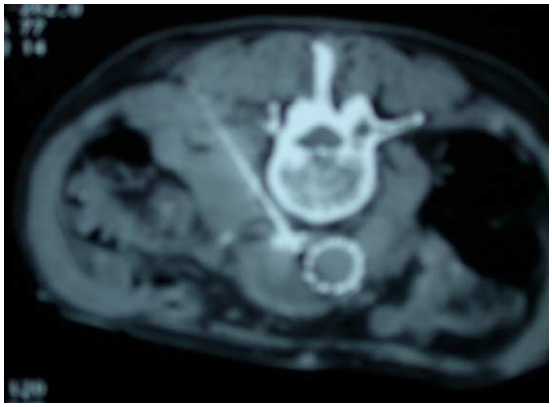

Fig. 4. CT guided injection of thrombin into the aneurismal sac.

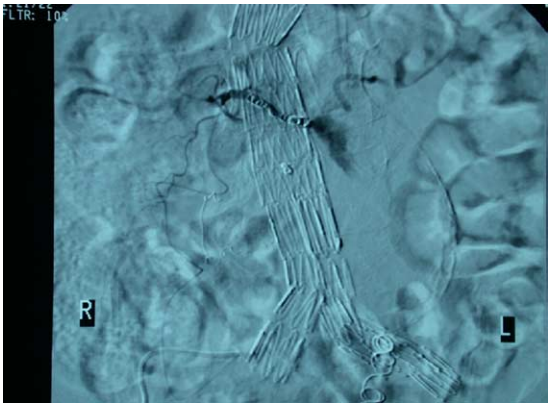

Fig. 5. Repeat attempt at embolisation of left lumbar artery along with its collaterals.

aetiology of sac rupture following endografting, this has not been the case with type II endoleaks.[2,5]

The natural history of the type II endoleak is undetermined. These endoleaks have been considered benign, especially when compared with type I endoleaks.[9] Spontaneous thrombosis is reported in up to two thirds of type II endoleaks.[6] Despite these observations, there is emerging evidence to suggest that not all type II endoleaks follow a benign course. Schrurink et al demonstrated no correlation between size of the endoleak and pressure transmission.[7] Secondly, *in vivo* pressure recordings of type II endoleak channels have recorded systemic pressure and pulsative waveforms. This pressure was transmitted to the aneurysm sac itself.[8,10] Further, even thrombosed type II endoleaks may be capable of raising intra-sac pressure[11] and there have been reports which have noted an increasing aneurysm sac size with these lesions.[12]

Studies of abdominal aneurysms treated by surgical ligation and bypass have revealed a 2% aneurysm sac patency despite intra-operative ligation of collateral vessels.[13] Almost a quarter of patients with persistent

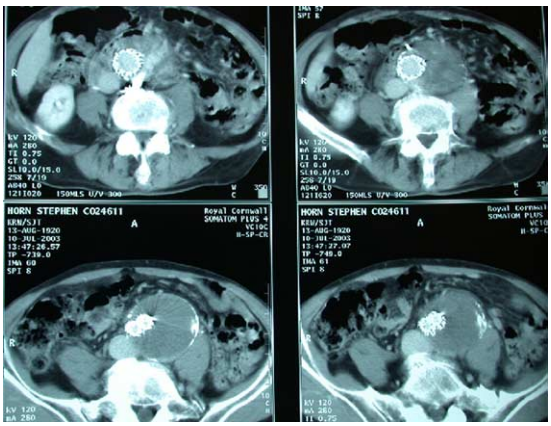

Fig. 6. Persistent endoleak and acute presentation? Rupture.

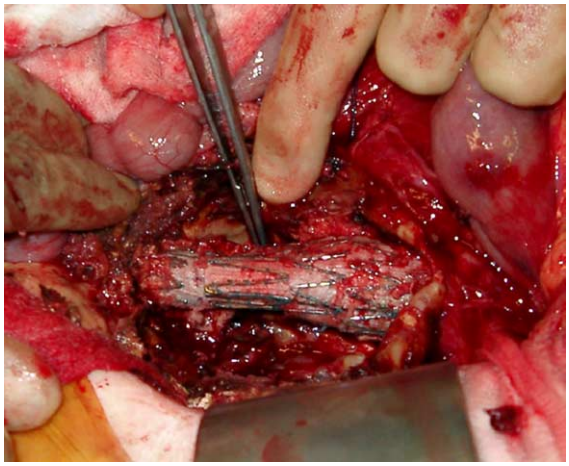

Fig. 7. Operative finding of intact stent graft, thrombus within the sac, and the pointer in the feeding lumbar vessel occluding the back flow.

collaterals subsequently presented with sac rupture.[13] Mortality following secondary conversion to open repair after EVAR were significant in elderly patients, who had a lower body weight and had a higher prevalence of chronic obstructive pulmonary disease.[3] The EUROSTAR collaboratory registry out of 1871 patients enrolled 11 patients had secondary conversions of which 6 were for rupture and 5 were for persistent endoleaks.[3]

It has been suggested that patients with endovascular grafts have a favorable outcome if they rupture in comparison to rupture without treatment.[14] In this case the endoleak persisted and became acutely symptomatic despite multiple attempts to occlude the feeding lumbar vessel, including direct sac embolisation. This confirms the danger of untreated type II endoleaks associated with aneurysm sac enlargement. Only complete occlusion of endoleaks results in decrease in the size of the aneurysm sac. Early interventional treatment of type II endoleaks is recommended because of endotension and the risk of rupture.[15] Although, in the main, type II endoleaks behave in a benign fashion, those which are associated with aneurysm sac expansion should be treated in order to prevent rupture (Fig. 7).

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