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LESSON OF THE MONTH

Rupture of an Abdominal Aortic Aneurysm Secondary to Type II Endoleak

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Introduction

Prevention of aneurysm rupture is the primary aim of treatment of abdominal aortic aneurysm (AAA). Early enthusiasm for endovascular aneurysm repair (EVAR) has been tempered by sobering reports of aneurysm sac rupture, despite apparently successful initial treatment.[1] These catastrophes have largely been attributed to graft migration and endoleak.[2,3]

Endoleak is a term that describes "the inability to obtain or maintain a secure seal between the aortic wall and a transluminally implanted intra-aneurysmal graft".[4] Type I, or attachment site endoleak, causes continued filling of the AAA leading to increased pressure and eventual rupture.[5,6]

Type II, or retrograde endoleak, was first described in 1998.[7] The significance of type II endoleak is largely unknown and its natural history has generally been thought to be benign.[8] There was no increase in aneurysm diameter during an 18 month follow-up of type II endoleak in one study.[9] Despite this, there has been one reported case of predicted aneurysm sac rupture due to persistent sac perfusion from an accessory renal artery.[10]

We report a case of fatal aneurysm sac rupture secondary to a known type II endoleak (inferior mesenteric artery).

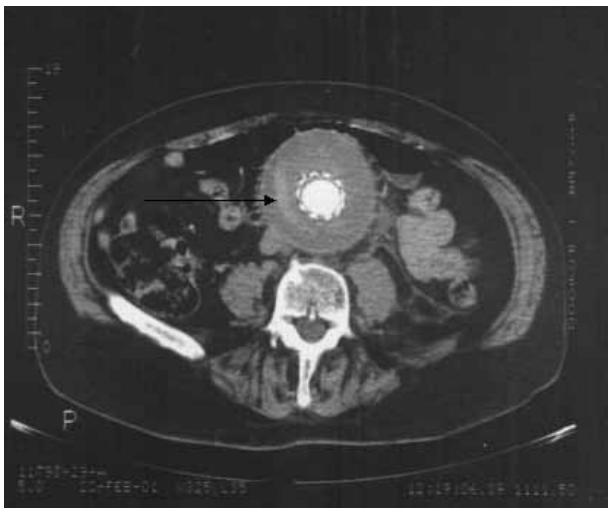

Fig. 1. One year SCTA, endoleak (arrow).

Table 1. Pre-operative aneurysm morphology.

Diameter of supra-renal aorta	22 mm
Angle of aortic neck with aorta	50°
Diameter of aorta at level of renal arteries	22 mm
Diameter of aortic neck (cranio-caudal)	23–34 mm
Diameter of aneurysm (maximum AP)	89 mm
Diameter of right common iliac artery	23 mm
Diameter of left common iliac artery	22 mm
Diameter of right external iliac artery	7 mm
Diameter of left external iliac artery	7–8 mm
Length of aneurysm neck	45 mm
Length of left common iliac artery	41 mm
Length of right common iliac artery	44 mm

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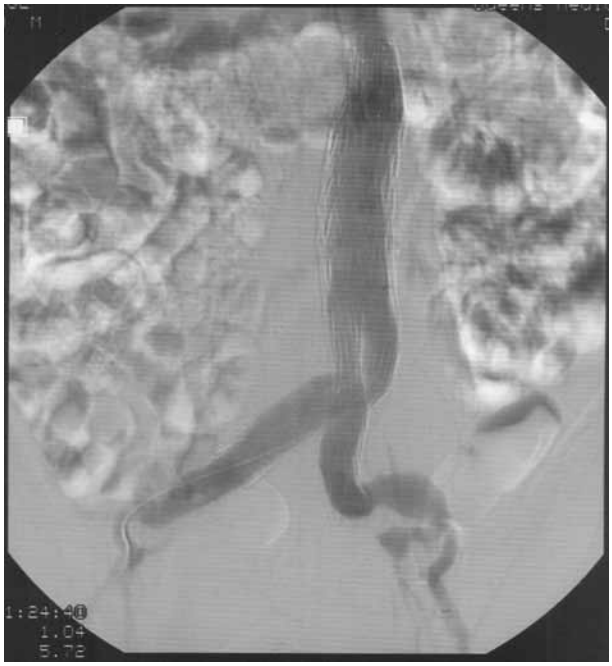

Fig. 2. Normal digital subtraction angiography (1 year).

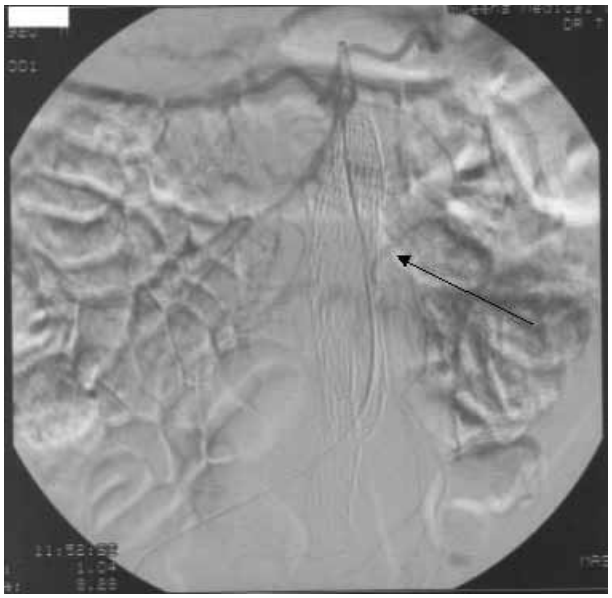

Fig. 3. Selective catheterisation of SMA, patent IMA (arrow).

Case Report

An 80-year-old man underwent elective EVAR of an 8.9 cm (maximal anteroposterior diameter) AAA. The patient had a history of hypertension and ischaemic heart disease (ASA grade 3). He was not taking warfarin.

The aneurysm morphology revealed a long infrarenal neck (45 mm), angulation was 50° (supra-renal aorta to neck) and the neck was conical (23–34 mm) (Table 1).

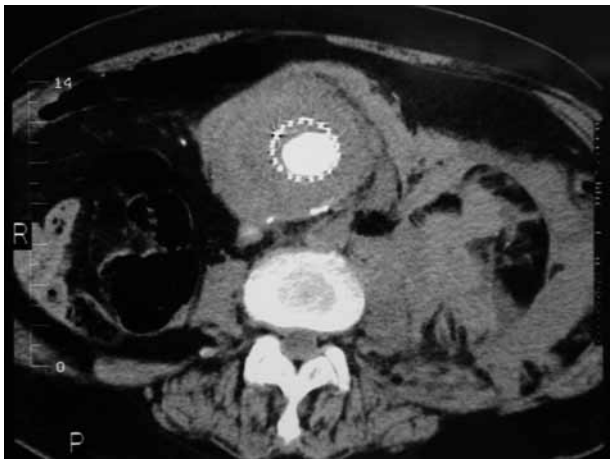

Fig. 4. Ruptured aneurysm sac.

The aneurysm was treated with a Zenith Trifab bifurcated endovascular stent-graft (Cook, Europe). There were no intra-operative complications and completion angiography was satisfactory with no evidence of endoleak. (Operative duration 150 min.)

The patient made an uncomplicated recovery and spiral computerised tomographic angiography (SCTA) at one week was satisfactory. The maximal anteroposterior (AP) aneurysm diameter was 9.1 cm. Follow-up SCTA at eight months showed satisfactory position and alignment of the endograft with no significant angulation or distortion. There was no evidence of endoleak although the aneurysm sac diameter had increased to 10.2 cm. Further SCTA was undertaken at 12 months when the patient was admitted with a short history of abdominal pain (Fig. 1). Radiographic contrast was observed in the 11 cm aneurysm sac (endoleak). Digital subtraction angiography with selective catheterisation of the superior mesenteric artery (SMA) demonstrated a patent inferior mesenteric artery (IMA) (Figs 2 & 3). Contrast was not clearly seen to enter the aneurysm sac and therefore embolisation was not performed. The patient was discharged and further management discussed.

Shortly afterwards, the patient represented with a one hour history of sudden onset, generalised abdominal pain. During SCTA the patient became haemodynamically unstable, rapidly deteriorated and died from hypovolaemic shock. SCTA confirmed aneurysm rupture (Fig. 4). There was no evidence of graft migration and the graft was patent.Subsequent post-mortem examination revealed a left-sided anterolateral aneurysm sac rupture. The stent-graft was well incorporated into the aortic wall both proximally

and in the common iliac arteries distally. The stent-graft body was intact as was the join between the main body and iliac limbs. There was fresh thrombus within the aneurysm sac. The IMA could not be detected due to the distortion produced from the large haematoma.

No other intra-abdominal pathology was found.

Discussion

Aneurysm expansion may be regarded as an indicator of failure of aneurysm exclusion following endografting.[11] Although there is good evidence to implicate both type I and type III endoleak and migration in the causation of sac rupture following endografting, this has not been the case with type II endoleak.[2,5]

The natural history of type II endoleak is undetermined. These endoleaks have previously been considered benign, especially when compared with type I endoleak.[9] Spontaneous thrombosis may be expected in up to two-thirds of type II endoleak.[12] Despite these observations, there is emerging evidence to suggest that not all type II endoleaks follow a benign course. Schurink *et al.* demonstrated no correlation between size of endoleak and pressure transmission.[13] Secondly, *in-vivo* pressure recordings of type II endoleak channels have recorded systemic pressure and pulsatile waveforms. This pressure was transmitted to the aneurysm sac itself.[14,15] Further, even thrombosed type II endoleaks may be capable of raising intra-sac pressure.[16] And there have been reports which have noted an increasing aneurysm sac size with these lesions.[17]

Studies of aneurysms treated by surgical ligation and bypass have revealed a 2% aneurysm sac patency despite intra-operative ligation of collateral vessels. Almost a quarter of the patients with persistent collaterals subsequently presented with sac rupture.[18]

It has been suggested that patients with endovascular grafts have favourable outcome if they rupture in comparison to rupture *de novo.*[19] In this case rapid haemorrhage from an aortic side branch resulted in hypovolaemic shock and death. The stent-graft did not appear to confer any significant advantage with regards to degree of haemorrhage, an observation corroborated by a recent review.[3]

This case confirms the danger of untreated type II endoleak associated with aneurysm sac enlargement. 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.

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