

CASE REPORT

A novel approach to the management of a ruptured Type II endoleak following endovascular repair of an internal iliac artery aneurysm

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ABSTRACT. Endovascular repair of isolated iliac artery aneurysms is an established safe and effective management option. Type II endoleak is a potential complication, but rarely results in significant morbidity or mortality. We report a case of a patient who presented with a ruptured internal iliac artery aneurysm secondary to a Type II endoleak. To our knowledge this and the following method of managing this have not been previously reported. Established methods of managing endoleaks, such as intravascular transfemoral embolisation and open or laparoscopic ligation, were not possible. Therefore, we resorted to a novel approach to this type of aneurysm and successfully performed a transcuteaneous direct puncture and embolisation of the superior gluteal artery.

Received 15 November 2010
Revised 31 January 2011
Accepted 7 February 2011

DOI: 10.1259/bjr/42137038

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Isolated iliac artery aneurysms (IAAs) are rare, with an incidence rate as low as 0.03% in large autopsy series [1]. These aneurysms represent approximately 2–7% of all intra-abdominal aneurysms [2, 3]. Iliac artery dilatation is regarded as aneurismal if it is >25 mm in diameter [4] and repair should be considered for aneurysms greater than 3–4 cm [3, 5]. Over the past decade, endovascular iliac aneurysm repair has been established as a safe and effective alternative to open repair in patients with appropriate anatomy [3, 5, 6]. A Type II endoleak is one of the complications of endovascular repair and arises due to the persistence of pressurised flow in iliolumbar and sacral arteries. Type II endoleaks are found in up to 20% of cases following endovascular repair [7, 8]. Although in most cases Type II endoleaks are considered benign [9], intervention becomes necessary when there is significant expansion of the sac [8] and, subsequently, increased risk of rupture. We report a case of a ruptured IAA secondary to a Type II endoleak and describe a percutaneous technique to embolise the endoleak. To the best of our knowledge, neither has been previously described in relation to an IAA.

Case report

An 84-year-old female presented with a reduced level of consciousness, acute onset abdominal pain and vaginal bleeding for several weeks. Initial assessment revealed that the patient was confused and disorientated, with a Glasgow coma score of 13. The patient was hypotensive (91/49), tachycardic (110) and pyrexial (37.9°C). Heart

sounds were normal, the chest was clear and an abdominal examination revealed tenderness and guarding mostly in the left lower quadrant. Initial investigations revealed haemoglobin (Hb) of 7.7 g dL⁻¹, white cell count (WCC) 9.9 cell L⁻¹, C-reactive protein (CRP) 188, urea 20.7 mmol L⁻¹ and creatinine 255 mmol L⁻¹. Urine dipstick analysis was positive for leukocytes, nitrites, protein and blood. The patient was resuscitated with intravenous (IV) fluids and IV piperacillin and tazobactam was commenced to treat the urinary tract infection. Subsequent urine culture revealed an *Enterobacter* infection.

The patient's medical history included bilateral internal iliac artery aneurysms (IIAAs), chronic renal failure with a left-sided hydronephrosis and ureteric stent *in situ*, hypertension and ischaemic heart disease. 2 years previously an occlusion of the left IIAA was performed with an Amplatzer plug (AGA Medical, Plymouth, MN) followed by endovascular placement of a covered stent

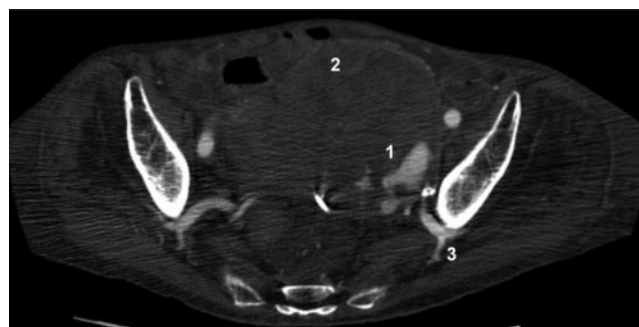


Figure 1. CT angiogram showing rupture of the left internal iliac artery aneurysms with (1) contrast filling the sac, (2) a large pelvic haematoma and (3) a Type II endoleak via the superior gluteal artery.

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from the common to the external iliac artery, covering the IIAA origin. This was performed in another centre and was complicated by a Type II endoleak as a result of the Amplatzer plug becoming dislodged. Given the patient's age and comorbidities, any further intervention was felt to have an unacceptably high risk of significant morbidity and mortality and no further intervention was offered at that time. The patient subsequently moved into the catchment area covered by our hospital.

A CT angiogram confirmed bilateral IIAA with a large pelvic haematoma around the left IIAA and flow into the aneurysmal sac via the superior gluteal artery was clearly evident (Figure 1). It became clear that the acute

presentation was the result of a rupture of the left IIAA secondary to the Type II endoleak.

Procedure

With no other approach open to us we attempted to access the endoleak through a transcutaneous approach. The patient was placed prone on the table. Intravenous sedation and pre-procedural antibiotic therapy to cover skin commensals were administered. A routine single-entry arterial needle was used to puncture one of the branches of the superior gluteal artery under duplex

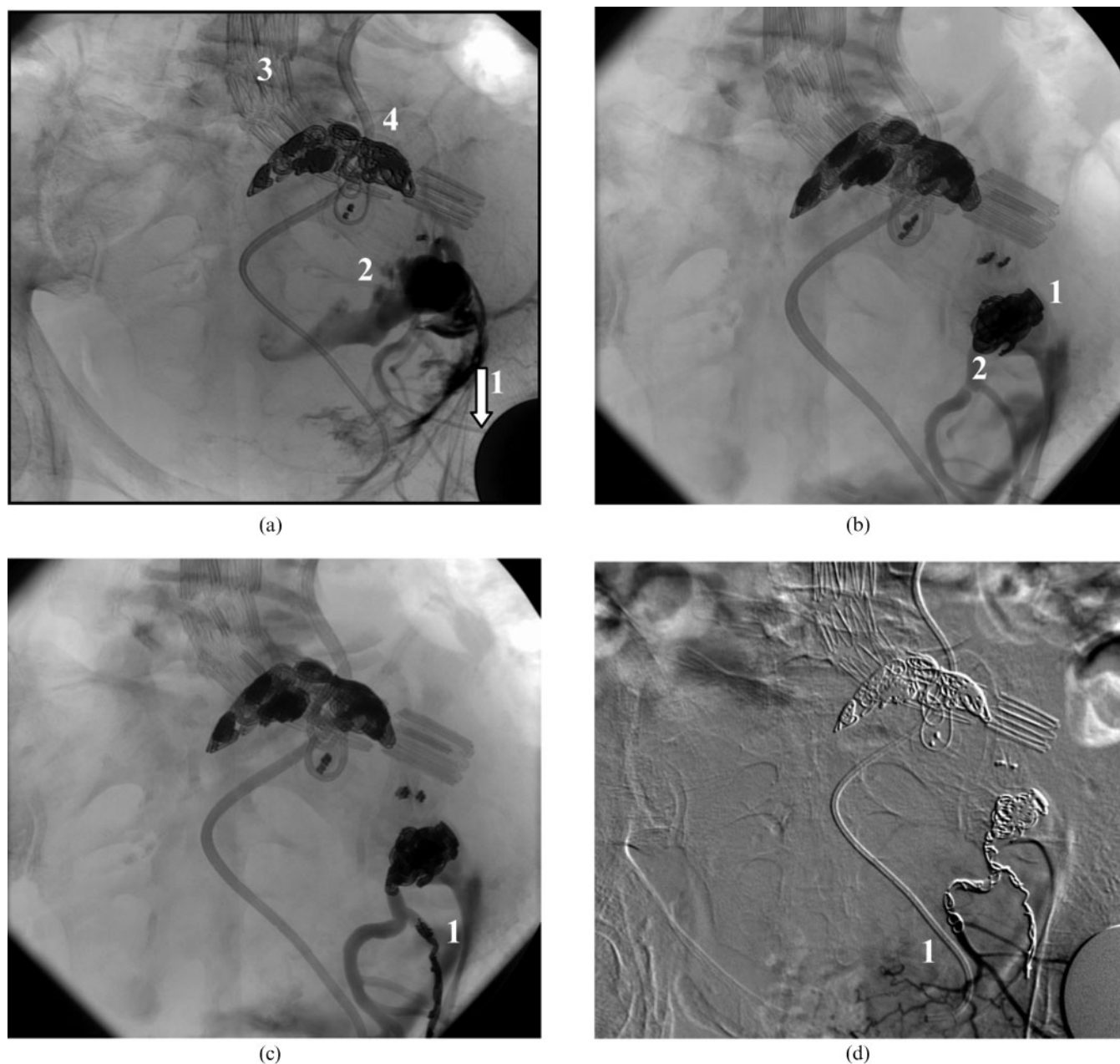


Figure 2. Angiographic images taken during the procedure. (a) Initial contrast injection through (1) the dilator after cannulation of the superior gluteal artery, (2) showing the endoleak into the internal iliac artery aneurysms sac, (3) the previous stent and (4) displaced Amplatzer occluder. (b) Initial coils (1) placed in the aneurysm sac and (2) the superior gluteal artery and its branches. (c) Further coils placed in the (1) left branch of the superior gluteal artery. (d) Completion angiogram after further coils placed in the (1) right branch of the superior gluteal artery with no further endoleak demonstrated.

ultrasound guidance, followed by a J-wire over which a 4 F dilator (Cook Ltd, Hertfordshire, UK) was inserted. Contrast medium was administered, which demonstrated the endoleak filling the aneurysm sac (Figure 2a), corresponding to the CT appearance. 24 microcoils (VortX; Boston Scientific, Natick, MA) of 4, 5, 6, and 7 mm diameter were released distal to the aneurysm via the dilator to embolise the main trunk (Figure 2b). A micro-catheter (Boston Scientific) was used to selectively cannulate and embolise the two branches (Figure 2c,d). A duplex ultrasound demonstrated complete occlusion of the endoleak with no flow in the aneurysm sac.

Discussion

There is little published data on the incidence, management or risk of rupture associated with Type II endoleaks following endovascular treatment of IAAs. Some of the principles of management are inferred from the published data on endovascular repair of abdominal aneurysms. Although most experts in the field consider Type II endoleaks to have a benign course [9], in some cases they assume clinical significance particularly when they remain after six months or appear *de novo* resulting in aneurysm sac expansion. In our case, sac expansion due to continued filling from branch vessels led to rupture, although the overall risk of this is low [5, 9, 10]. Therefore, most authors advocate patients with Type II endoleaks following endovascular repair of an IAA should be managed conservatively unless there is evidence of enlargement of the aneurysm sac [5, 10].

Treatment options include percutaneous transluminal coil embolisation [11], intra-arterial embolisation [12], and—more specifically in the case of IAAs—percutaneous transosseous embolisation [13]. The technique of laparoscopic branch ligation has been suggested as an alternative for the treatment of Type II endoleaks by some experienced investigators [14], particularly in cases of failed embolisation. Open ligation is rarely used and only indicated when all other approaches have failed.

The incidence of Type II endoleak following iliac artery repair is around 15% [5] even with routine coil embolisation of distal vessels pre-stent insertion. Ruptures secondary to Type I endoleak [6] and Type III endoleak [15] following endovascular repair of an IAA have been previously described but, to our knowledge, we are the first to report rupture following a Type II leak. In our case there was no intravascular transfemoral route to embolise the endoleak. Open or laparoscopic ligation was not considered owing to patient age and comorbidities. Therefore, we resorted to a novel approach and successfully performed a transcuteaneous direct puncture and embolisation of the superior gluteal artery. To our knowledge this has never before been described in this type of aneurysm. Surveillance is essential to detect endoleaks early and we adopt a combined approach using CT angiogram and six monthly duplex ultrasound, which has been shown to have a high sensitivity at detecting endoleaks [16, 17]. Overall we feel the best approach to treatment of Type II endoleaks is one that is safe, effective and tailored to the specific circumstances and anatomy of the patient. Although rupture of IAAs is rare, particularly after Type II endoleak, a high degree of

suspicion is essential to correctly diagnose and manage this condition.

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