

Early Type II Endoleak Complication and Open Conversion Post Endovascular Aneurysm Repair of Ruptured Abdominal Aortic Aneurysm

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ABSTRACT

Background: Endovascular Aneurysm Repair (EVAR) has been a long-stay surgical option to treat abdominal aortic aneurysms (AAA), both contained and ruptured. Endoleaks, defined as persistent flow in the aneurysm sac extrinsic to the endograft, is the most common complication. Type II endoleak (T2EL) results from collateral aortic branches (mainly lumbar arteries, inferior mesenteric artery) flowing retrogradely into the sac. It is often considered the Achilles' heel to EVAR due to the controversies in its timing of diagnosis and management. It is generally accepted that T2EL are slow growing, could expand the aneurysm sac, and carry a small but significant risk of aneurysm rupture.

Case Presentation: This case reports an early post-EVAR complication due to T2EL.

A 66-year-old man was brought to casualty hypotensive, minimally responsive with a rigid and tender abdomen. CT Angiography revealed a ruptured AAA contained within the retro peritoneum. The patient subsequently underwent an uncomplicated EVAR procedure. However, one-hour post procedure in ICU, he was found to be suddenly in haemorrhagic shock. Open conversion via exploratory laparotomy revealed 5L of blood within the peritoneum. Aneurysm sac was opened and four strong bleeding lumbar arteries were identified and oversewn to control the bleeding. It has hypothesised that vigorous T2EL from lumbar back-bleed, through the ruptured aneurysm sac led to ongoing bleeding into the contained retroperitoneum and subsequently secondary "rupture" into the peritoneal cavity.

Conclusion: This case showed that T2EL are not all slow growing and innocuous. One should consider T2EL as a cause of a patient who is acutely deteriorating post-EVAR. Early CT-angiographic imaging post procedure may be indicated in certain groups.

Keywords

Endoleak, EVAR, Ruptured AAA, Type II Endoleak.

Introduction

Abdominal aortic aneurysm (AAA) is an important vascular disease with a prevalence of 4-8% in men aged 60 years and over [1]. This disease is significant due to the major morbidity and mortality involved with a rupture (rAAA), and hence surgical candidates with AAA size greater than 5.5cm are recommended repair via open or endovascular approach [2]. Endovascular

aneurysm repair (EVAR) involves the placement of modular stent graft delivered via access from commonly the femoral arteries to line the aorta, to maintain arterial continuity and to exclude the aneurysm sac from the systemic circulation.

Endoleak is often seen as the Achilles' Heel of EVAR, with type II endoleak being seen as the most perplexing and complex. Type II endoleak (T2EL) is defined by the backflow of blood from aortic collaterals into the aneurysm sac, despite graft deployment. There is continuous perfusion of the aneurysm sac (most commonly from

the inferior mesenteric artery and the lumbar arteries), hence the aneurysm sac is not fully excluded from the systemic circulation [3]. T2EL is not uncommon after EVAR, with a reported incidence of 10.2% from a large scale, multi-centre study by Sidloff et al. [4]. However, there is still debate on the significance of T2EL, the need and the timing for intervention once the endoleak is identified [5]. T2EL are categorised as early (occurring within 30 days of initial procedure), late (occurring after one year) and persistent (duration for longer than 6 months) [3].

A number of risk factors for the development and persistence of T2EL have been reported in the literature, including the presence of multiple collateral vessels – at least 1 patent hypogastric and 4 patent lumbar arteries, at least one of which is greater than 0.2mm [6]. Additionally, patent inferior mesenteric artery, anti-platelet medication and EVAR performed for ruptured AAA have also been identified to contribute to T2EL [7].

There are a variety of intervention strategies divided into percutaneous and open surgical. Percutaneous solutions involve trans-arterial or trans-lumbar embolization. Trans-arterial approach accesses the endoleak via retrograde vascular arcades, whereas trans-lumbar embolization accesses the aneurysm sac and the respective nidus directly [8]. Surgical options include open surgery to reach the aneurysm sac and to ligate the culprit vessels under direct vision, or alternatively via laparoscopic approaches [9]. These interventions are performed to prevent the aneurysm sac from continual expansion, and subsequent aneurysm rupture.

There is currently ongoing research involved in the identification, surveillance as well as indicators for surgery for patients with T2EL post EVAR. Studies from Lo [10], Brown [3] and Walker [11] have shown that a proportion of T2EL can resolve spontaneously, hypothesized due to the reasonable chance of spontaneous sac thrombosis preventing further flow. Additionally, it is shown that these transient T2ELs were not associated with late adverse outcomes [12]. It is reported that only a small proportion (5%) of patients post EVAR develops persistent T2EL of longer than 6 months [10]. Due to the lack of consensus evidence relating progression of T2EL and adverse outcomes, patients are often kept under frequent surveillance, with only a small proportion (23.4%) actually receiving intervention [11]. Intervention strategies (percutaneous or surgical) are often centre based, with sac size and sac expansion (>10mm) being the key factor for intervention [3]. These procedures are often performed years after the initial EVAR procedure. However, there are growing evidence to suggest increased adverse complications with T2EL. More recently, Seike et al. showed a statistically significant correlation in cumulative incidence rates between persistent T2EL and adverse events including sac dimension increase >5mm, rupture and AAA mortality (13).

This case report goes against the understanding of late T2EL risks, highlighting a patient with an early, rather than late T2EL post EVAR for a ruptured AAA. He required same day open surgical conversion for management of acute life threatening endoleak complications.

Case Presentation

A 66-year-old man was brought in by ambulance with acute central abdominal pain, hypotension, diaphoresis and confusion. His medical history was significant for hypertension on triple anti-hypertensives, gout, hyperlipidaemia, obstructive sleep apnoea and depression. He was an ex-smoker with no significant surgical history. On initial assessment with the paramedics, he was hypotensive with blood pressure 80/35mmHg, weak pulse with rate of 80 beats per minute, tachypnoeic to 50 breaths per minute and GCS 12. He was unable to mount a tachycardic response in the setting of being on a beta-blocker. His examination revealed clear lungs, regular heart rate and electrocardiogram showing no evidence of acute ischaemia. His abdomen was reported to be firm with generalised tenderness to palpation and he was quickly transported to the emergency department with concerns of an intra-abdominal bleed.

Laboratory results in the emergency department revealed haemoglobin of 9.6g/dL, pH 7.14, Lactate 10.2mmol/L and a base excess of -10.6mEq/L, consistent with haemorrhagic shock. The patient was partially responsive to resuscitation, with 4 units of packed red blood cells given throughout the initial resuscitation efforts pre-operatively. Urgent CT imaging reviewed an acute rupture of a 68mm by 61mm fusiform infrarenal abdominal aortic aneurysm. The rupture was contained in the retroperitoneal space, more marked on the left side, with small active extravasation indicative of small ongoing bleed (Figure 1). The patient was subsequently taken for definitive intervention.



Figure 1: Axial CT Angiogram showing ruptured abdominal aortic aneurysm, with extensive contained retroperitoneal haemorrhage, white arrow marking minute contrast blush of at the level of rupture indicative of small ongoing bleed.

Course of Treatment

The patient underwent an EVAR using a bifurcated stent graft with bilateral iliac stents (Zenith Alpha). The left iliac limb was extended as the aneurysm involved the bifurcation extending into the origin of the left common iliac artery. Completion angiogram

confirmed satisfactory deployment of stent graft, exclusion of aneurysm, patent renal and internal iliac arteries, and no presence of an endoleak (Figure 2). The patient was subsequently taken to the intensive care unit (ICU) for post procedural care.



Figure 2: Digital Subtraction Angiography post EVAR repair showing endograft in satisfactory position, with no endoleak.

The patient rapidly deteriorated in ICU despite optimal support one hour post operatively. His arterial blood gas showed a decreasing haemoglobin of 6.5g/dL and worsening acidosis with pH 7.05, Lactate 8.5mmol/L and base excess -16.9mEq/L. The patient underwent immediate open conversion, where 5L of blood was evacuated from the peritoneal cavity and a breach in the retroperitoneum was identified on the left side. The aneurysm sac was opened, the stent graft found to be appropriately positioned with arterial continuity. The cause of his deterioration was found to be due to four bleeding lumbar arteries, these arteries were oversewn at their ostia to control the bleeding. The stent graft was left intact, the aneurysm sac and retroperitoneum were subsequently closed, and the patient left with an open abdomen due to concerns of bowel ischaemia secondary to risk of abdominal compartment syndrome from the hemoperitoneum. He had two re-look procedures on post-open conversion days 1 and 3, with nil convincing evidence of bowel ischaemia or necrosis, subsequently the abdominal wound was closed.

The patient had ongoing vasopressor requirements, with persistent acidosis refractory to treatment, delirium and worsening abdominal distention. Serial CT imaging showed a segment of proximal sigmoid colon with mural thickening, decreased enhancement and intramural gas locules concerning for ischaemia (Figure 3). He underwent a Hartmann's procedure on day 13 with short segment sigmoid colon resection and an end colostomy formation.

The patient had a protracted ICU and hospital stay due to significant morbidity, including acute kidney injury requiring short term haemodialysis, ventilator associated pneumonia,

persistent ileus requiring total parenteral nutrition for 22 days and prolonged delirium. His condition stabilised after 26 days in ICU, convalesced for a further 12 days on the surgical ward and was eventually discharged. Outpatient follow-up with the patient showed a remarkable recovery, with no cognitive deficits and independence with mobility and activities of daily living.



Figure 3: Axial CT Abdomen, arrow pointing to site of mural thickening, hypo-enhancement and gas within bowel wall.

Discussion

This case highlights the rare circumstances where T2EL can not only lead to chronic progressive concerns, but can also cause early complications leading to a worsening of an originally contained aneurysm rupture. As detailed earlier, persistent T2EL leading to potential aneurysm sac size change and complications are usually slow progressing. These patients are monitored for month to years, and eventually intervened upon after careful consideration and planning. Various studies have shown the mean timeframe of open conversion for T2EL repair to be 5.7 years [14] and 5.5 years [15]. Early open conversions after the initial EVAR procedure are usually performed for other complications such as graft infection, renal artery coverage and type I endoleak rather than type II [14].

The 66-year-old man in this case report likely had his initial aneurysm rupture contained within the retroperitoneum towards the left side (Figure 4). This containment was aided by the concept of hypotensive haemostasis, where the low blood pressure avoided disruption of the unstable clot in the retroperitoneum, and hence avoided exsanguination. However, after the EVAR procedure was completed, arterial continuity restored, patient optimised and blood products replaced, the briskly back bleeding lumbar arteries led to disruption of the clot containment. Subsequently, the rising pressure in the retroperitoneal cavity led to a peritoneal breach on the left side, and henceforth the secondary rupture and uncontained haemorrhage into the peritoneal cavity leading to the critical instability found in the ICU.



Figure 4: Axial CT Angiogram further showing retroperitoneal contained rupture with large amount of retroperitoneal haemorrhage, predominantly on the left.

Despite the fact that rAAA has been identified as a positive risk factor for the development of T2EL [7], there is still a clear gap in the literature about understanding the potential underlying contributing factors. Boniakowski et al. conducted the first multicentre retrospective study directly looking into the link between rAAA and T2EL in 2016 from a 13-year database. They found 16 out of 56 patients with T2EL after EVAR for rAAA, 0 patients with delayed aneurysm rupture and only 5 patients requiring intervention [16]. The only statistically significant variable associated with the development of T2EL in their retrospective study was elevated body mass index (34.7 vs 28.6). Their study investigated late and persistent T2EL after EVAR procedures. However, this case report demonstrates that early T2EL post EVAR also has the potential to carry significant adverse events. This suggests that there is clear room for further research to investigate for risk factors of both late, as well as early onset T2EL after EVAR for ruptured AAA. Furthermore, this will lead to better understanding of the progression of T2EL, help identify high-risk groups earlier and better mitigate its adverse effects.

The higher risk factor of rAAA leading to T2EL could potentially alter the follow-up algorithms. Currently, The Society for Vascular Surgery practice guidelines suggest post EVAR surveillance protocol with CT imaging initially at 1 month, 6 months and 12 months, then yearly thereafter [17]. Perhaps higher risk patients such as rAAA EVAR patient would benefit with post procedural or pre-discharge CT assessment. This may help to stratify their risks and plan follow-up protocols ahead of time, depending if T2EL was immediately detected post procedurally.

Conclusion

Endoleaks, particularly type 2 continues to remain the Achilles' Heel of endovascular AAA repair. The growing evidence of T2EL's potential detriment should raise concerns. In lieu with the case report's discussion of T2EL post EVAR causing early

complications with significant morbidities, more robust research is needed in the field of rAAA and T2EL, in order to identify further risk factors to aid the understanding of the pathophysiology of this complication. This would aid in more prudent rAAA EVAR patient selection, help set surveillance protocol for high-risk groups, as well as create uniform evidence-based guidelines for T2EL intervention in positive patient groups.

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