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ORIGINAL ARTICLE

Management of Type II Endoleaks by Embolisation after Endovascular Abdominal Aortic Aneurysm Repair: Retrospective Review of Patient Data

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ABSTRACT

Objective: *To review the success and complication rate of transarterial and translumbar embolisation of type II endoleaks after endovascular aneurysm repair (EVAR) of abdominal aortic aneurysms.*

Methods: *We conducted a review of post-EVAR type II endoleaks treated by interventional radiology from June 2016 to December 2017.*

Results: *A total of 17 embolisations for type II endoleaks in 11 patients were identified. Two patients had >1 interventions for recurrent endoleaks. Type IIA endoleaks occurred in seven patients. Three patients had type IIB endoleaks from the inferior mesenteric artery (IMA) and lumbar artery (LA). The last patient had endoleaks from multiple LAs. In cases where the IMA was the culprit (n = 6), endovascular access was achieved via the superior mesenteric artery via the arc of Riolan, followed by embolisation of the IMA, and, in some cases, the aneurysmal sac. When the LA was responsible (n = 8), it was accessed via the ipsilateral internal iliac artery and iliolumbar artery. Direct puncture of the aneurysmal sac was performed on five occasions in a single patient with a 13-cm aneurysm sac. The procedural success rate was 100%. The clinical success rate was 72%, 'satisfactory' as defined by stable sac size. No procedure-related complication was identified.*

Conclusion: *Transarterial or translumbar embolisation remains an effective treatment option for post-EVAR type II endoleaks.*

Key Words: *Aorta, abdominal; Aortic aneurysm, abdominal; Blood vessel prosthesis implantation; Embolization, therapeutic; Mesenteric artery, superior*

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中文摘要

血管腔內腹主動脈瘤修復後栓塞治療II型內漏：患者數據回顧

錢凱、梁肇庭、梁錦榮、簡偉權

目的：釐回顧血管腔內主動脈瘤修復（EVAR）腹主動脈瘤後行經動脈和經腰椎栓塞術治療II型內漏的成功率和併發症發生率。

方法：回顧2016年6月至2017年12月期間，以介入放射治療EVAR後II型內漏的病例。

結果：11例患者中共有17次II型內漏栓塞。兩名患者因內漏復發進行了超過1次介入治療。7名患者出現IIA型內漏。3名患者的腸繫膜下動脈和腰動脈發生IIB型內漏。最後一名患者多個腰動脈出現內漏。如果內漏因腸繫膜下動脈而起（n = 6），可通過腸繫膜上動脈的Riolan動脈弓行血管內通路，然後栓塞腸繫膜下動脈，而且在某些情況下須栓塞動脈瘤囊。如果內漏因腰動脈返流（n = 8），可通過同側髂內動脈和髂腰動脈行血管內通路。一例有13厘米動脈瘤囊的患者，進行了五次動脈瘤囊直接穿刺。手術成功率為100%。臨床成功率（指囊大小穩定）也令人滿意，達72%。無與手術相關的併發症。

結論：經動脈或經腰椎栓塞術是治療EVAR後II型內漏的有效選擇。

INTRODUCTION

Since its introduction, endovascular aneurysm repair (EVAR) has evolved rapidly and revolutionised the treatment of abdominal aortic aneurysm. With the advancement of technique and stent-graft design, more and more anatomically challenging abdominal aortic aneurysms can now be treated with EVAR. Compared with open repair, studies have shown that EVAR has a lower perioperative morbidity and mortality rate.[1-3]

Endoleaks, a complication unique to EVAR, are unfortunately common. They are estimated to involve 20% to 25% of post-EVAR patients.[4] Endoleak is defined as evidence of persistent blood flow into the aneurysmal sac, and is classified as types I to IV (Table 1).[5] Types I and III need urgent treatment. Type IV is almost always transient and does not require treatment. The management of type II endoleaks is variable. Persistent type II endoleaks can lead to continuous exposure of the aneurysm sac to arterial pressures and may increase the risk of delayed rupture of the aneurysm, particularly if there is associated sac enlargement. Conversely, many type II endoleaks resolve by themselves, rendering conservative management an option. Therefore, patients receiving EVAR require long-term imaging surveillance.[5]

With increasing experience at many institutions worldwide, interventional radiology is the modality of choice in managing type II endoleaks, using a variety of embolic agents, either alone or in combination. It serves as a versatile and less invasive therapeutic alternative, compared to open ligation. In this article, we aimed to review the success and complication rate of transarterial (TA) and translumbar (TL) embolisations of type II endoleaks.

METHODS

We performed a retrospective review of all post-EVAR type II endoleaks treated by interventional radiology at our institution from June 2016 to December 2017, retrieving patient demographics and clinical data from the electronic medical record. All endoleaks were diagnosed on surveillance triphasic computed tomography (CT). Imaging findings (sac size, type of endoleak, feeding vessels on both CT and the angiogram), procedural

Table 1. Classification of endoleaks.

Type I	Endoleaks at the opposing ends of the graft
Ia	Proximal
Ib	Distal
Ic	Iliac limb
Type II	Endoleak due to retrograde flow from vessels
IIa	Single vessel
IIb	Two or more vessels
Type III	Endoleak due to mechanical failure of the stent graft
IIIa	Leak at the junction between the modular components
IIIb	Leak due to fracture or perforation of the stent graft
Type IV	Endoleak due to porosity of the graft

records (approach, angiographic findings, embolic agent[s] used), complications, follow-up interval, imaging modalities used on follow-up, and the decision for re-intervention were reviewed. Either interval sac enlargement or persistent endoleak (>6 months after EVAR) was taken as an indication for treatment after review and consensus between vascular surgeons and interventional radiologists. We used either a TA or TL/direct sac puncture approach at our institution.

Transarterial Approach

This approach relies on the presence of an anastomosis between two different vascular territories. It is crucial for the interventional radiologist to review previous imaging studies (most commonly a CT angiogram [CTA]) and to determine the culprit vessel, technical feasibility (presence and anatomy of the anastomosis) and potential difficulties of the procedure (e.g., vessel tortuosity or stenosis). We performed the embolisation in our angiography suite, mostly utilising a transfemoral, and, occasionally, a transbrachial approach, based on the vascular anatomy.

A 5-Fr vascular sheath is commonly used for the transfemoral approach. A screening aortogram (Figure 1) would first be performed if the preceding CTA could not exclude a type I or III endoleak. Then the aneurysmal sac is navigated, into the culprit feeding vessel using a combination of catheter, guidewire, microcatheter, and microguidewire. The inferior mesenteric artery (IMA) can be accessed via the arc of Riolan, if present. It is a collateral vessel that can be seen (Figures 2 and 3) arching over the left abdomen between the middle colic branch of the superior mesenteric artery (SMA) and the IMA, creating an SMA/IMA anastomosis. A guiding sheath is first brought into the proximal SMA, followed by a 4-Fr Cobra catheter (Cordis, the Netherlands) at the middle colic branch of SMA and a microcatheter into the IMA and aneurysm sac, establishing triaxial access. Intra-arterial injection of nitroglycerin or verapamil may be used to overcome vasospasm during navigation.

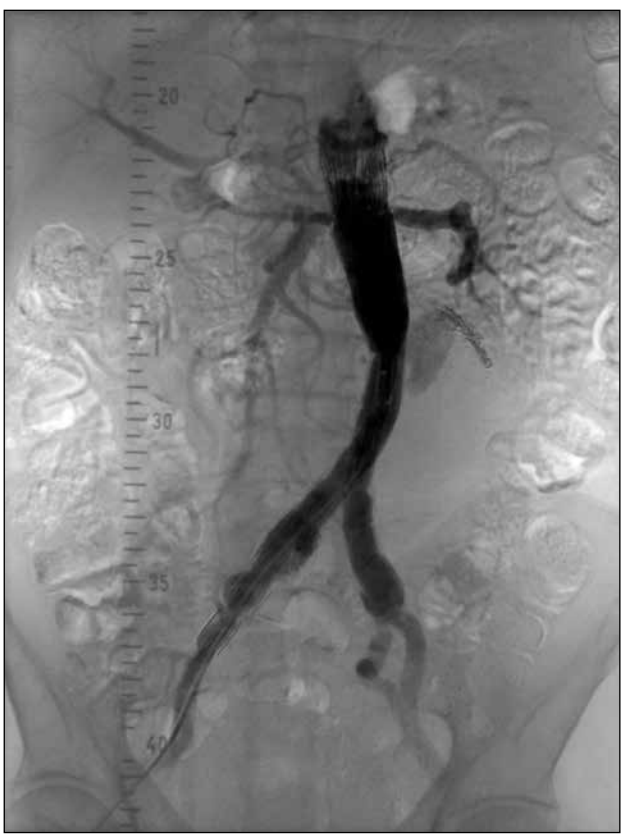

Figure 1. Aortogram performed prior to embolisation to exclude type I and III endoleaks after an inconclusive computed tomography angiogram. No type I or type III endoleak was identified in this aortogram.

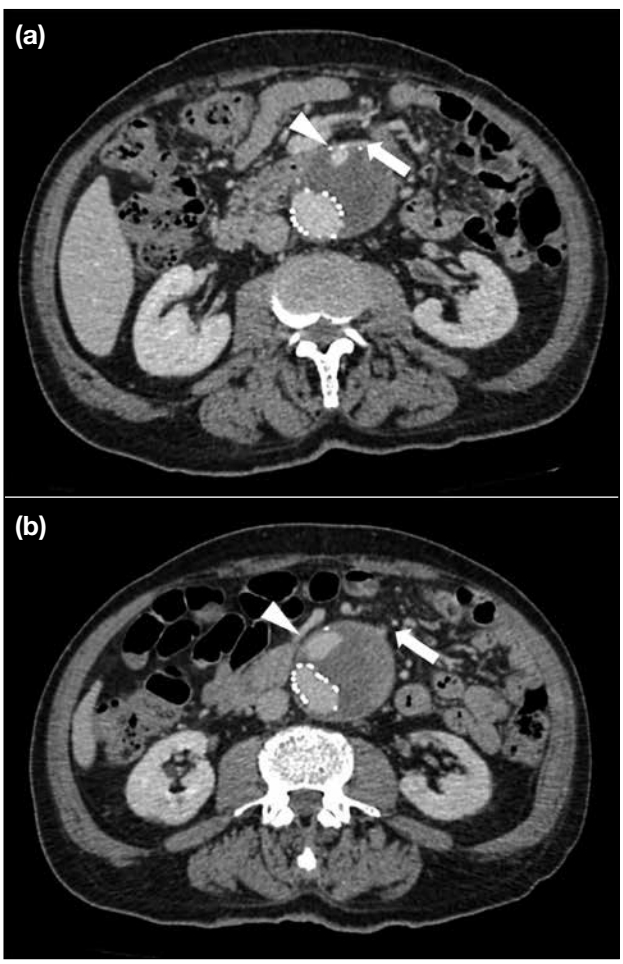

Figure 2. Type II endoleak supplied by the inferior mesenteric artery. (a) and (b) show contrast computed tomography in arterial phase at two levels, demonstrating the endoleak (arrowheads) from the inferior mesenteric artery (arrows).

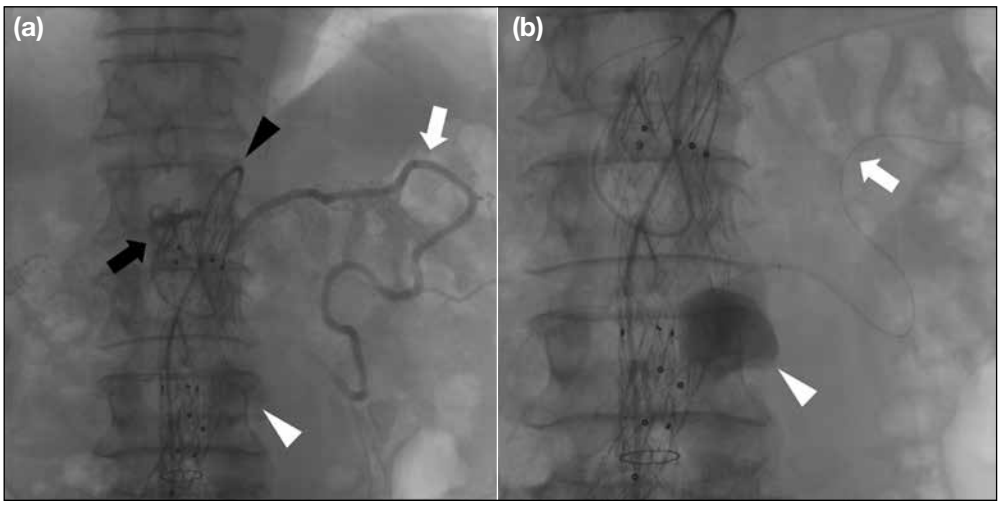

Figure 3. Arc of Riolan. (a) Angiogram with microcatheter navigated into the middle colic artery (black arrowhead) of the superior mesenteric artery (black arrow), outlining the arc of Riolan (white arrows in a and b). (b) Angiogram with microcatheter in the arc of Riolan showing endoleak from the inferior mesenteric artery (white arrowheads in a and b).

If the culprit vessel is a lumbar artery (LA) [Figure 4], it might be accessed using an ipsilateral transfemoral approach. After gaining access, we place a 5-Fr guiding sheath (Flexor Ansel Guiding Sheath; Cook Medical [IN], US) in the ipsilateral internal iliac artery (Figure 5). Oblique projections can help in delineating the vascular anatomy and better visualisation of the iliolumbar artery. The iliolumbar artery is subsequently catheterised using a microcatheter with the aid of a microguidewire (1.5-2.7-Fr microcatheter, 0.008-0.018-inch microguidewire).

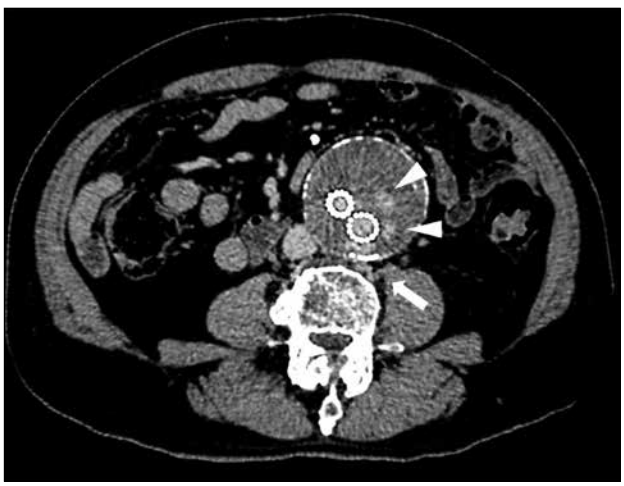

Figure 4. Type II endoleak (arrowheads) from a left lumbar artery (arrow).

Once advanced into the aneurysm sac, contrast is injected via the microcatheter (saccogram), to confirm the tip position of the microcatheter and evaluate the intra-sac flow dynamics (e.g., net flow rate, outflow vessels, and filling defects). The aneurysm sac is then embolised, followed by the feeding vessel(s), and confirmation of stasis (Figure 6).

Translumbar or Direct Sac Puncture Approach

The retroperitoneal aneurysm sac can also be assessed with the patient lying prone, under either fluoroscopy, cone-beam CT, or multidetector CT guidance. At our centre, we use a combination of these methods, with additional reference to prior CTA findings. The radiopaque stent-graft, adjacent bony structures, and any prior radiopaque embolisation material offer good radiological landmarks on fluoroscopy. An 18-gauge trocar-type needle is used for puncturing the aneurysm sac, at the side where the aneurysm sac is most readily accessible (usually the left) through the flank region, and is angled anteromedially (Figure 7). The needle is advanced under fluoroscopic guidance in orthogonal planes, or under CT guidance, until back bleeding is encountered. Contrast is then injected into the sac to confirm needle tip position, followed by embolisation and a saccogram for confirming stasis. Some reports suggest measuring the pressure within the aneurysm sac and documenting the loss of the arterial waveform within the sac as additional evidence of successful embolisation.[4,6] Such measures were not adopted at our centre.

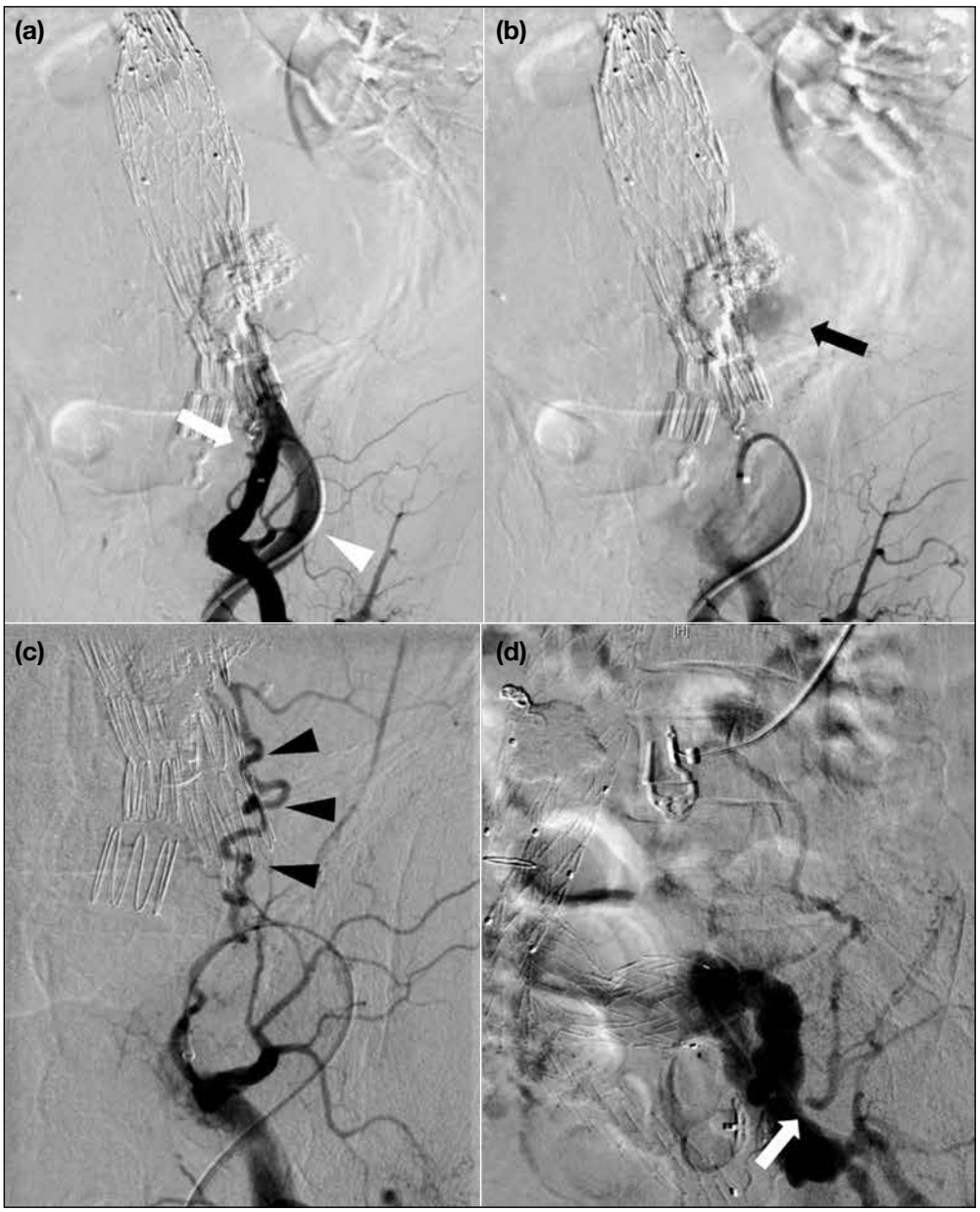

Figure 5. Transarterial embolisation via the iliolumbar artery. (a) Angiogram with catheter navigating from left external iliac artery (white arrowhead) into the proximal part of the ipsilateral iliac artery (white arrow). (b) Subsequent image showing evidence of endoleak (black arrow). (c) Angiogram demonstrating the tortuous left iliolumbar artery (black arrowheads), after a microcatheter is advanced into the proximal part. (d) Angiogram showing the iliolumbar artery in another patient. Sometimes the iliolumbar artery has unfavourable anatomy, with an acute take-off angle and stenosis (white arrow).

Embolic Agents

We commonly use liquid embolic agents (n-butyl cyanoacrylate [NBCA], 13/17 of our cases), or ethylene vinyl alcohol copolymer (Onyx; ev3, Irvine [CA], US) [2/17]. In one case we used bovine-based thrombin (THROMBIN-JMI; Pfizer Inc, New York [NY], US). Ideally, the aim is for complete, permanent sac exclusion and controlled reflux into the most distal segment of the feeding vessels, without causing non-target embolisation into the IMA or LA. In practice, we considered a substantial reduction of intra-sac flow acceptable as a procedural endpoint, particularly in aneurysm sacs with high net outflow rates, after weighing the risks of non-target embolisation. Additional coils were usually placed at the feeding vessel(s). We mixed NBCA with ethiodized oil (Lipiodol; Laboratoire Guerbet, Aulnay-Sous-Bois, France) in the range of 12.5% to 25%. The addition of ethiodized oil increases the radiodensity of the mixture, allowing its visualisation during procedures. By varying its proportion, it also enables the adjustment of polymerisation time of NBCA in an inverse fashion. The total volume of NBCA used varied substantially depending on the sac size, ranging from 0.5 to 52 mL in our review. Some suggest putting coils into the

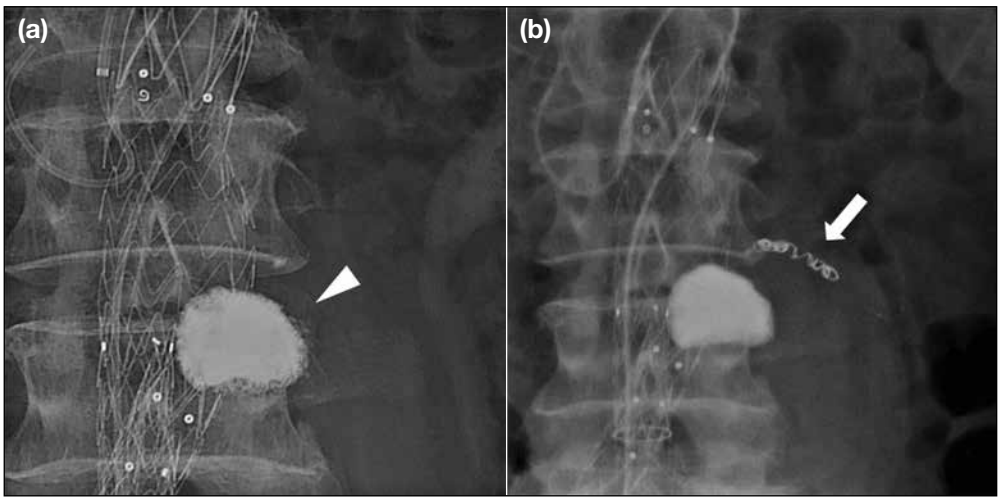

Figure 6. Sac embolisation and coiling of the inferior mesenteric artery. Fluoroscopic images showing (a) complete embolisation of the sac (arrowhead) and (b) the inferior mesenteric artery subsequently embolised by coils (arrow).

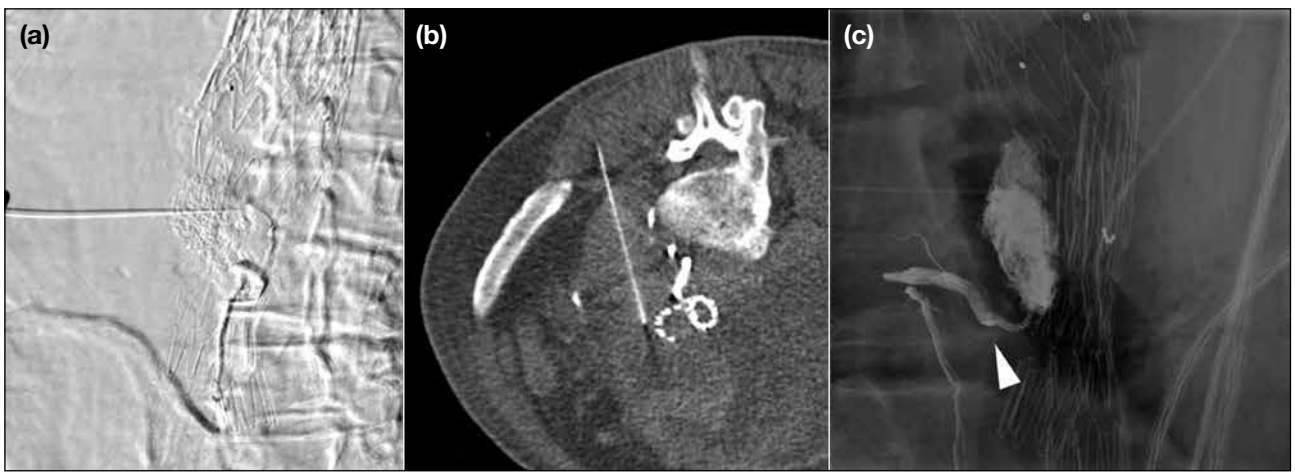

Figure 7. Translumbar approach/direct sac puncture. (a) Anteroposterior fluoroscopic image showing needle inserted into the aneurysm sac. (b) Computed tomography confirms needle in situ. The stent graft should be cautiously avoided. (c) Image post-embolisation using n-butyl cyanoacrylate. There is reflux into the lumbar artery (arrowhead).

aneurysm sac before injection of liquid agent, with the intention of slowing the flow within the aneurysm sac and reducing the risk of non-target embolisation into the inflow vessels. The type of embolic agents used has bearing on subsequent imaging surveillance. In our experience, coils cause significant artefact on CT (Figure 8), which might affect surveillance or assessment in the case of recurrent endoleak.

RESULTS

A total of 17 embolisations for type II endoleaks on 11 patients (10 men, 1 woman) with median age 85 years, (range, 71-92 years) were identified (Table 2). The median aneurysmal sac size was 8.2 cm (range, 4.2-21.8 cm).

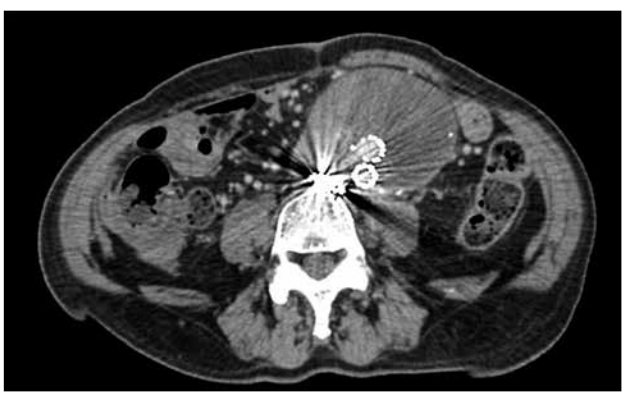

Figure 8. Coil can cause significant artefacts on computed tomography images, limiting subsequent assessment in case of recurrent endoleak.

Table 2. Overview of the patients undergoing embolisation for type II endoleak.

Patient No.	Sex / age, y	Procedure No.	Type	Culprit vessels on CTA	Sac size prior to embolisation, cm	Approach	Target embolised	Embolic agents
1	M / 85	1	IIa	IMA	4.2	TA	sac + IMA	NBCA
2	M / 76	2	IIa	IMA	5.2	TA	sac + IMA	NBCA + coil
3	M / 72	3	IIa	IMA	5.3	TA	sac + IMA	NBCA
4	M / 71	4	IIa	IMA	4.7	TA	sac + IMA	NBCA + coil
5	M / 85	5	IIa	LA	6.6	TA	sac + LA	NBCA
6	M / 89	6	IIa	LA	8.6	TA	sac + LA	NBCA
7	M / 74	7	IIa	LA	8.2	TA	sac + LA	Onyx
8	F / 88	8	IIb	LA ± IMA	8	TA	sac + LA	NBCA + coil
9	M / 83	9	IIb	IMA + LA	7.8	TA	sac + IMA	NBCA + coil
10	M / 77	10	IIb	IMA + LA	7.5	TA	sac + IMA	NBCA + coil
10	M / 78	11	IIb	LA	8.7	TA	LA	Coil
11	M / 92	12	IIa	LA	13	TA	sac + LA	Onyx
11	M / 92	13	IIa	LA	14.5	TL	sac + LA	NBCA
11	M / 92	14	IIb	LA	17.5	TL	sac + LA	NBCA
11	M / 92	15	Probably IIb	N/A	18.2	TL	sac + LA	NBCA
11	M / 92	16	Probably IIb	N/A	20.6	TL	sac	Thrombin
11	M / 92	17	Probably IIb	N/A	21.8	TL	sac	NBCA

Abbreviations: CTA = computed tomography angiogram; IMA = inferior mesenteric artery; LA = lumbar artery; N/A = not directly visualised on CT, probably from LA through fine anastomosis; NBCA = n-butyl cyanoacrylate; TA = transarterial; TL = translumbar.

Type IIa endoleaks were seen in seven patients: four of them with IMA supply and three with LA supply. Two patients had more than one intervention for recurrent endoleaks. Three patients with type IIb endoleaks from the IMA and the LA had embolisations performed on the IMA alone (n = 1), LA alone (n = 1), or both IMA and LA (n = 1). The last patient had endoleaks supplied by varying numbers of LAs over time.

Direct puncture of the aneurysmal sac (TL approach) was performed on five occasions for a single patient (initial sac size 13 cm). Either NBCA (12.5% to 25% concentration) or thrombin (a total of 8000 units, in a dilution of 1000 U/mL) was injected.

The median fluoroscopic time and dose-area product were 30.2 minutes (range, 14.3-66.3 minutes) and 237.0 Gy·cm^2 (range, 27.4-772.6 Gy·cm^2), respectively.

The procedural success rate was 100%, with angiographic evidence of haemostasis or substantial flow reduction within the aneurysm sac achieved in all 11 patients. The clinical success rate was satisfactory (72%), as eight patients had stable sac size during a median follow-up period of 9.5 months (range, 1-27 months). The results in this group of patients were as follows: reduced or completely resolved endoleak in six patients (75%) and persistent endoleak in two patients (25%). Two patients had an increase in sac size (6-mm and 12-mm increase) during a mean follow-up period of 6.3 months (range, 4-8.5 months); one patient received a second embolisation, resulting in a stable sac size but a new endoleak with an indeterminate culprit vessel on CTA while the other was managed conservatively. The eleventh patient with a 13-cm initial sac size had multiple embolisations performed over 5 months because of persistent endoleaks and sac enlargement. No procedural-related complications were noted.

DISCUSSION

The clinical course for type II endoleaks can be quite variable. It is usually divided into early (within 6 months after EVAR) and late (>6 months after EVAR). A substantial number of early type II endoleaks will resolve spontaneously, with prevalence at 6 months approximately 10% to 15%.[5] Some patients develop 'de novo' delayed type II endoleaks. For patients with persistent type II endoleaks, the clinical course is also variable: the majority of the patients (50%-70%) have a stable sac size, 25% have a decrease in sac size, and the remaining 25% have an increase.[5] It has been shown that persistent endoleaks result in persistent arterial pressurisation of the aneurysm sac, which could theoretically increase the risk of delayed sac rupture. Whether to adopt conservative management with continuous imaging surveillance or early treatment for these patients is still up to debate and likely varies among local practices,[7,8] but most agree that a significant

increase in sac size (defined as >5 mm in diameter)[5] on interval imaging surveillance should be regarded as an indication for expeditious treatment, with other factors taken into consideration (e.g., pretreatment sac size, presence of culprit vessel[s] on imaging, symptoms, and patient's co-morbidities and preferences).

Different feeding vessels contribute to the formation of type II endoleaks, either alone (type IIa) or in combinations (type IIb). Examples include the IMA (Figure 2), the lower LA, and, less frequently, the median sacral artery or even an accessory renal artery.[9] Risk factors for a type II endoleak includes a patent IMA, number and diameter of patent LAs, and continued angicoagulation.[5] One study showed that the cross-sectional area of the contrast-enhanced aortic lumen at the level of the IMA is positively associated with the development of a type II endoleak.[10]

Previous studies have shown that durable embolisation of type II endoleaks requires embolisation of both the aneurysm sac and the feeding vessels. This approach is analogous to the approach to arteriovenous malformations in the brain: embolisation of the feeding arteries alone without embolising the nidus will lead to persistence or recurrence of the underlying vascular lesion.[11,12] Similarly, embolisation of the aortic aneurysm sac alone has a higher rate of endoleak recurrence.[11,12]

The most common approach for treating endoleaks includes TA and TL routes, as adopted at our institution. The technical challenge in the TA approach mainly lies in the vascular anatomy and the significant length of the vessels that need to be traversed before reaching the aneurysm sac. Vasospasm may occur with prolonged procedure. Embolising a culprit LA is usually more challenging, as the vessel tends to be more tortuous, and it is not uncommon for the iliolumbar artery to have an unfavourable take-off angle from the internal iliac artery (Figure 5d). The transcaval approach is reported in the literature but rarely utilised due to the potentially serious consequences of the complications, such as aortocaval fistula, pulmonary embolism from non-target embolisation, and retroperitoneal bleeding.[4]

Complications of TA and TL embolisations can be categorised mainly into embolic agent-related or approach-related. Overall complication rate ranges from 0% to 12%.[11-14] Nontarget embolisation into proximal parts of the IMA and LA when using a liquid embolic agent is the main concern. Ischaemic sciatic neuropathy after TA embolisation using NBCA and non-target embolisation of IMA branches has been reported.[12] Approach-related complications specific to TL embolisation include: inadvertent injury to the LA and nerve root as the needle traverses the abdominal wall; accidental puncture of the stent graft, resulting in type III endoleak; and retroperitoneal bleeding and haematoma.[11-14] Still, the TL approach is considered a safe approach for managing type II endoleak.

Studies have shown that both TA and TL approaches have comparable clinical success rates, with no significant differences in aneurysm sac growth, persistent endoleak, or complications. TL approach may result in shorter fluoroscopy time and total procedural time.[4,12,14]

There has been no universal definition of 'clinical success'. Some define it as cessation of type II endoleak. In this retrospective study, we defined clinical success as absence of a substantial increase in sac size. Our results are comparable to those in a relatively large retrospective study conducted by Stavropoulos et al.[12]

Limitations of our study include its retrospective nature, the small sample size, and the relatively short and wide range of follow-up times. All TL embolisations were performed in a single patient presenting with a 13-cm aneurysm sac. This limits further statistical analysis and comparison between the TA and TL approaches.

CONCLUSION

In the era of EVAR, both TA and TL embolisations are effective treatment options for type II endoleaks.

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