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REVIEW/STATE OF THE ART

Thoracic Endovascular Aortic Repair for the Treatment of Aortic Dissection: Post-operative Imaging, Complications and Secondary Interventions

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Abstract Thoracic endovascular aortic repair (TEVAR) has become an accepted alternative to surgery for the treatment of aortic dissection (AD). Lifelong surveillance is obligatory following TEVAR to monitor the aortic morphology and detect associated complications. This is particularly important in AD where coverage of the primary intimal tear is necessary in achieving thrombosis and regression of the false lumen. A variety of imaging techniques may be used in assessing the technical success, outcome and complications, which may necessitate re-intervention. Of these, computed tomography angiography offers a fast, accessible and sensitive imaging modality and is established as the default surveillance tool. The purpose of this article is to review the imaging modalities, post-procedural appearances including complications and re-intervention strategies following TEVAR for AD.

Keywords Aortic dissection · Endovascular treatment · Complications · TEVAR · CTA

Introduction

Acute aortic syndrome encompasses a heterogeneous spectrum of life-threatening conditions including penetrating atherosclerotic ulcer, intramural aortic haematoma and aortic dissection (AD). Of these, AD carries the worst prognosis with significant early and late morbidity and mortality. AD occurs due to an intima–media tear, most commonly at the proximal thoracic aorta, creating a secondary channel, the false lumen (FL), which allows blood flow into the aortic wall. The FL usually propagates distally, but can also extend proximally to the aortic root. Compression of the normal vessel lumen [true lumen (TL)] by the FL can compromise blood flow to vital organs. The Stanford classification divides ADs into two groups, 'A' where the ascending aorta is involved, and 'B', which involves the descending thoracic aorta [distal to the left subclavian artery (LSA)]. Whilst the conventional treatment for Stanford type B AD is medical, intervention is indicated for cases complicated by recurrent chest pain, hypertension refractory to therapy, malperfusion, early aortic expansion (>4 mm) and aortic aneurysmal dilation (>5.5 cm) [1–3].

Thoracic endovascular aortic repair (TEVAR) was originally introduced as a minimally invasive treatment for thoracic aortic aneurysms. Since the first case report of TEVAR in ADs by Dake [4], it has undergone a dramatic expansion and become accepted as the treatment of choice for complicated type B AD [5] and is also utilised in other acute aortic syndrome disorders [6].

Unlike endovascular repair of aortic aneurysms, there are no randomised controlled studies comparing TEVAR with open surgery. The current literature for endovascular management of thoracic aortic pathology is heterogeneous consisting of case series or registry data based on various pathologies and different presentations. Nevertheless, TEVAR has been proven safe and efficacious for AD, with a significantly lower length of hospitalisation, reduced morbidity and perioperative mortality, and satisfactory short- and mid-term (up to 5 years) results [7–13]. However, there is an increased need for re-intervention when

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compared to open surgery and long-term outcomes are yet to be evaluated, particularly in the younger population [14].

The clinical success and outcome of TEVAR for AD based on lesion exclusion without post-treatment complication (e.g. persistent false lumen perfusion (FLP), aortic size increase and endograft migration) requires close and regular follow-up.

Imaging Following TEVAR

The main imaging techniques used in evaluating TEVAR cases are computed tomography angiography (CTA), magnetic resonance angiography (MRA) and catheter angiography. Of these, advancements in CT technology have propelled it to being the default modality. Given the increasing number of patients with AD treated with endovascular techniques, it is important not only for interventional radiologists, but all radiologists to be familiar with the normal post-TEVAR appearances, the commonly encountered complications and main re-intervention strategies.

CTA is the default imaging modality for the evaluation of the thoracoabdominal aorta before and after endograft repair [15]. Advantages include widespread availability, fast scan time, high-resolution volumetric acquisition enabling detailed multiplanar imaging and the ability to detect other relevant aortic pathology [16, 17]. Importantly, CTA offers high soft tissue resolution essential in evaluating the aortic diameter, true and FL size, sac thrombus and calcification [18]. Disadvantages include radiation dose burden and use of iodinated contrast medium.

A complete CT evaluation includes an unenhanced scan of the chest, abdomen and pelvis, followed by an electrocardiographic (ECG)-gated arterial phase contrast-enhanced scan [19]. However, a single arterial phase acquisition is usually sufficient for surveillance, assuming availability of non-contrast images from a previous examination. Where available, a dual-energy CT scan can provide a virtual non-contrast reconstruction from a single post-contrast acquisition. Furthermore, although ECG gating further improves diagnostic sensitivity, it is mainly indicated if there is uncertainty due to cardiac motion and pulsation artefact or ambiguity regarding a Stanford type A dissection. For the latter, ECG gating enables accurate assessment of the sinuses of Valsalva, the valve cusps, the aortic annulus and the coronary arteries as well as the location and extent of a proximal dissection flap [20, 21]. Finally, delayed phase post-contrast acquisition is occasionally required to detect low flow leaks; however, MRA maybe more sensitive in this regard [22, 23].

MRA offers inherent advantages including lack of ionising radiation and iodinated contrast medium. However it has not replaced CTA due to several limitations including higher cost, longer acquisition time, lower special resolution, reduced reliability in distinguishing calcification, limited visualisation of metallic stent struts and incompatibility with stainless steel endografts [24]. However, with increasing availability, there is a potential growing role for MRA, particularly in younger patients, where the consequences of lifelong surveillance in terms of contrast-induced nephropathy (CIN) and cumulative radiation dose are considered [25]. With regards to the latter, Zoli et al. [26] calculated that a TEVAR procedure including a preoperative CTA, three follow-up CTAs in the 1st year and yearly evaluation thereafter increases the risk of leukaemia or solid tumours by more than 2.7 % within 15 years. From a renal perspective, the recommended guidelines for renal protection should be followed in all cases undergoing CTA, particularly in patients with a glomerular filtration rate (GFR) <45 ml/min which is considered a risk threshold for iodinated contrast and carries a 15 % risk of CIN after a single contrast load for CTA [27]. Whilst gadolinium-based contrast agents are less nephrotoxic compared with iodinated contrast, caution should be exercised in cases with GFR <30 ml/min due to risk of nephrogenic systemic fibrosis [28].

Plain radiographs are seldom used for TEVAR assessment due to evident limitations. Catheter angiography is not used for surveillance, but is a useful tool for problem solving; mostly utilised when non-invasive modalities cannot adequately characterise a known or suspected endoleak or FLP. Targeted angiograms via a flush catheter and selective vessel injections can help assess the origin, flow direction and extent of these. In addition, simultaneous flat-panel rotational CT on the angiography table can further enhance diagnostic sensitivity in complex cases.

Imaging Surveillance Protocols

The clinical success and outcome of TEVAR requires close and regular follow-up.

With little data available are on long-term outcomes, our understanding of the frequency and significance of various adverse findings is evolving and so are the surveillance methods and strategies used.

At our institution, CTA is the default surveillance imaging tool, obtained at suspended full inspiration following the administration of 100 mls non-ionic contrast medium 300 mgI/ml at 5 mls/s via a power injector into an 18G venous access catheter in the antecubital vein, with 40 mls saline chaser at 5 mls/s, with scan delay determined by bolus tracking over a region of interest over the thoracic or abdominal aorta.

Our post-TEVAR surveillance protocol includes a CTA before discharge post-stenting, at 6 weeks, 6 and 12 months and annually thereafter. In the presence of an adverse finding, scans are performed at more frequent intervals to assess

whether the adverse finding is persistent or progressive mandating the need for re-intervention. Non-contrast CT is used in cases of renal impairment or contrast allergy. MRI and chest radiographs are seldom used.

The Aims of TEVAR

Evaluation of the normal and adverse imaging findings following TEVAR is dependent on an understanding of what TEVAR aims to achieve. The ultimate aim of TEVAR is to prevent aortic disease progression or rupture. In AD this is achieved primarily by exclusion of the main communication between the true and FLs in order to enable thrombosis of the FL and re-expansion of the collapsed TL. Successful FL thrombosis is typically associated with an increase in TL size, concomitant regression of the FL and overall reduction or stabilisation of the aortic dimension, which should all be assessed on follow-up imaging [29, 30] (Fig. 1A, Table 1). Conversely, persistent FLP is associated with an increase in the FL size and aortic diameter (Fig. 1B). In a study of 41 TEVAR cases for acute type B

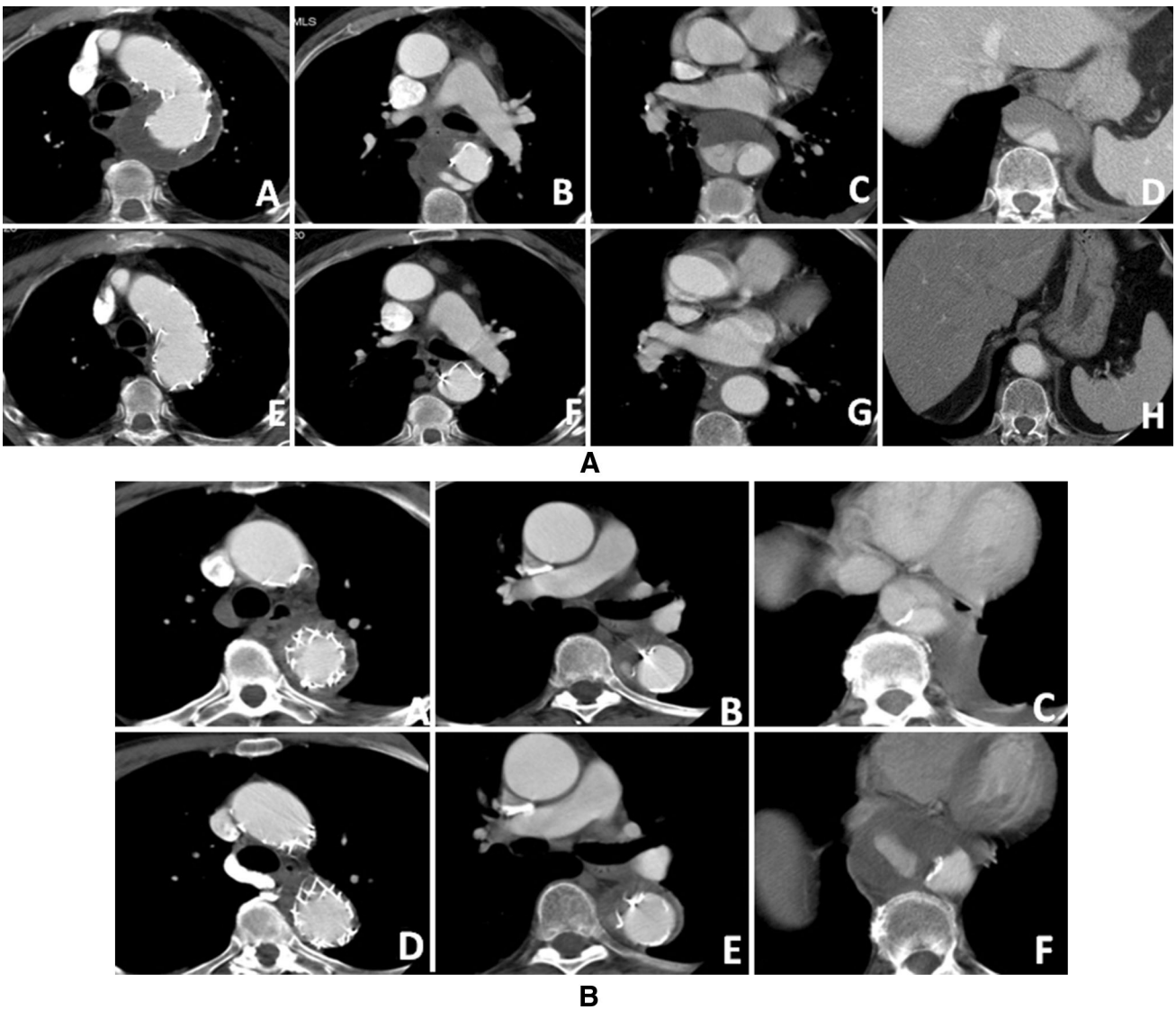


Fig. 1 **A** Optimal TEVAR result in a patient with chronic aortic dissection (CAD). Axial CTA images at the level of the aortic arch (**A**, **E**), main pulmonary artery (**B**, **F**), right atrium (**C**, **G**) and diaphragm (**D**, **H**), 1 week (*top row*) and 3 years (*bottom row*) following TEVAR for type B aortic dissection. There is near-complete thrombosis and exclusion of the FL at 1 week. After 3 years, the FL diameter has significantly diminished, the TL diameter has increased and the aortic dimensions have reduced. **B** Persistent false lumen perfusion following TEVAR for CAD. Axial CTA images at the level of the aortic arch (**A**, **D**), right pulmonary artery (**B**, **E**), and ventricles (**C**, **F**), 1 week (*top row*) and 2 years (*bottom row*) following TEVAR for type B aortic dissection. There is persistent FL perfusion at 1 week. After 2 years, the FL diameter has increased, the true lumen diameter is unchanged and the aortic diameter has increased significantly

Table 1 Questions to address on follow-up imaging

Questions to address on follow-up imaging
Is there persistent false lumen perfusion?
If so, what is the cause?
Is there a type A dissection
Has the thoracic aorta increased in size?
Has the abdominal aorta increased in size?
Are the endografts intact?
Is there stent-graft collapse/separation/fabric tear

dissection, Kim et al. [31] reported an increase in TL size of 29 % at 30 days, 51 % at 1 year and 80 % at 5 years, with FL regression of 69, 76 and 86 % respectively among 31 patients with no FLP. This compared with no significant change in the TL size at 1 year in the 11 patients with FLP.

Success of TEVAR is contingent on adequate stability at its proximal and distal landing zones to avoid FLP [32]. Similar to EVAR, a proximal landing zone without significant calibre change, calcification, or thrombus is desirable. However, anatomic complexities at the aortic arch including short curved and tapered necks and luminal surface irregularity present a frequent challenge in patients undergoing TEVAR and are the most important reasons for early and late stent-graft treatment failure.

Nomenclature—is the term endoleak appropriate after TEVAR for aortic dissection?

When assessing CTA post-TEVAR for dissection, the term "endoleak" may be confusing. Persistent FLP from inadequate coverage of the proximal fenestration can be referred to as an endoleak—proximal type 1 endoleak. However, persistent FLP from natural fenestrations distal to the endografts should probably not be referred to as a distal endoleak, but simply as persistent FLP from distal fenestrations. FLP due to retrograde flow from intercostal vessels are regarded as type 2 endoleaks by some authors and as FLP due to retrograde intercostal artery perfusion by others. The term type 2 endoleak in this setting is probably appropriate.

Regarding the situation of endograft disconnection or a fabric tear (which in aneurysmal disease would produce a type 3 endoleak); in the setting of AD, the dissection flap is usually still intact, so FLP due to endograft disconnection or a fabric tear does not usually occur. As a result, type 3 endoleaks in AD are very unusual.

Therefore, in the follow-up after TEVAR for AD, the terms "proximal type 1 endoleak" and "type 2 endoleak" can be correctly used similar to patients after TEVAR for aneurysmal disease. The term "distal type 1 endoleak" for FLP from distal fenestrations is not appropriate and the "type 3 endoleak" should be limited to those cases where there is endograft disconnection or a fabric tear PLUS a focal tear in the dissection flap leading to FLP at this location.

Imaging Assessment Following TEVAR: Complications and Treatment Strategies

Complications following TEVAR can be divided into procedural, early (<6 months) and late (>6 months) (Table 2). Procedure-related adverse events include access site complications and neurological events including stroke and paraplegia. The latter are rare with a reported incidence of 0.8–1.2 % for stroke and 0.4–0.9 % for paraplegia in two systematic reviews [10, 33]. These complications are recognised clinically and are not discussed further in this article. Early and late complications are further evaluated below.

Early Complications

The most common early complication following TEAR is persistent perfusion of the FL. Retrograde type A dissection (RTAD) is infrequently encountered, but carries a poor outcome.

Table 2 Complications following TEVAR for AD

Procedural	Early (<30 days)	Late (>30 days)
Neurological: stroke, paraplegia	False lumen perfusion	Persistent or late-onset false lumen perfusion
Puncture site: haematoma, pseudoaneurysm	Retrograde type A dissection	Retrograde type A dissection (Usually presents early)
Other: cardiopulmonary complications, bowel ischemia, unintended side branch occlusion		Increase in thoracic and/or aortic size
		Stent-graft migration, collapse, strut fracture, fabric tear
		Recurrence/progression of original disease

Retrograde Type A Dissection

Retrograde extension of the primary tear into the ascending aorta is a serious but rare event seen in 1.3–1.6 % of cases and is commonly an early complication with 13–20 % of cases occurring peri-operatively and a further 38–50 % in the first 30-days post-surgery [34, 35]. This is seen as a new or extension of existing dissection to the ascending aorta or aortic root (Fig. 2). RTAD is more common after TEVAR for AD compared with aortic aneurysm and is associated with oversizing of the endograft, post-deployment balloon dilation of the endograft and possibly the use of endografts with a proximal bare spring and/or barbs. Treatment is by emergency aortic repair [36]. However, there is a high reported mortality rate of 34 % [34].

False Lumen Perfusion

The aim of TEVAR in AD is the complete exclusion of flow into the FL at the main proximal fenestration by coverage of the fenestration by an endograft. The most important predictor of proximal FLP is the existence of a short neck and thus ensuring an adequately long, proximal landing zone which is the main factor in preventing this. At least a 20-mm proximal neck of non-dissected aorta is advocated prior to the initial TEVAR to avoid incomplete sealing of the primary intimal tear [37, 38].

Persistent FLP due to inadequate endograft coverage of the main proximal fenestration is referred to as a proximal type 1 endoleak (Fig. 3). As stated previously, persistent FLP due to natural fenestrations distal to the endografts (usually at the level of the upper abdominal visceral arteries) should not be referred to as a distal type 1 endoleak because the distal FLP is not endograft related. A type II endoleak due to retrograde perfusion of the FL from intercostal arteries is not of major concern in AD as these are not thought to impede remodelling and thrombosis of the FL and usually spontaneously resolve within 6 months [39].

If persistent FLP is due to a proximal type 1 endoleak due to poor endograft coverage at the proximal fenestration, these patients require proximal extension of endograft

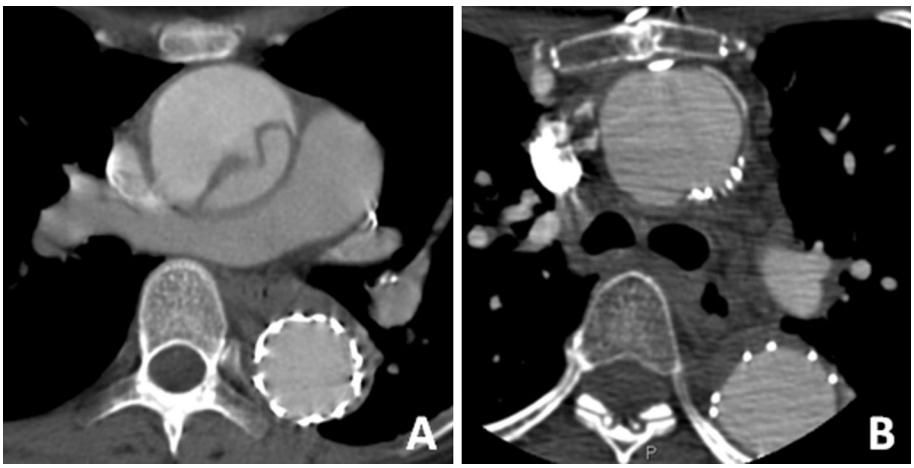


Fig. 2 Retrograde type A dissection. Axial CTA images at the level of the ascending aorta show retrograde type A dissection following TEVAR for a type B dissection (**A**), which was successfully surgically repaired (**B**)

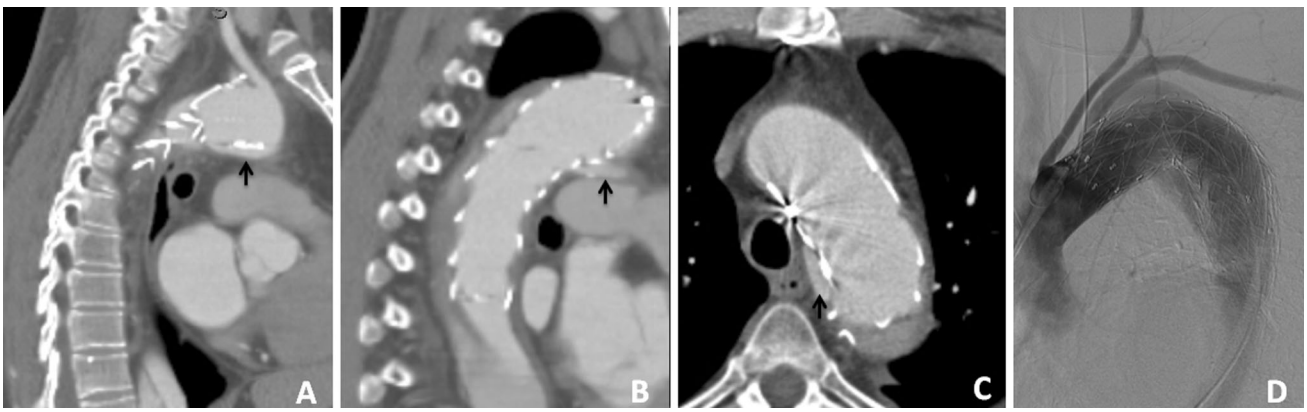


Fig. 3 False lumen perfusion related to proximal attachment site. Sagittal (**A**, **B**) and axial (**C**) CTA images at the level of the aortic arch show persistent FLP from the proximal attachment site (proximal type 1 endoleak) following TEVAR (*arrow*). Catheter angiography with the tip of the flush catheter beyond the proximal endograft shows filling of the FL confirming FL perfusion from the proximal attachment site

coverage. However, if the CTA shows good coverage of the proximal fenestration between the TL and FL, it can be assumed the FL arises from the abdominal fenestrations. Re-intervention is not generally required in this scenario in the absence of increasing aortic size for several reasons. Firstly coverage of the proximal primary intimal tear sufficiently redirects blood flow into the TL to promote FL thrombosis, and FL regression in the medium to long term [15]. Furthermore, these distal tears are usually sufficiently small not to allow significant pressurisation of the TL or aortic rupture compared with the proximal primary tear. Thirdly, the upper abdominal natural visceral fenestrations enable continued perfusion of branch vessels supplied by the FL. Finally, minimising initial overall graft length reduces the risk of paraplegia [40]. However, if thrombosis of the FL does not occur or there is an increase in the aortic size, extension of endograft coverage distally can be performed at a later time.

Late Complications

Late complications of TEVAR include disturbed endograft integrity (collapse or separation), late-onset FLP, and an increase in the size of the thoracic or abdominal aorta. The latter can be due to increase of FLP or progression of aortic disease.

Endograft Collapse

Although collapse of the stent graft has been reported following dissection repair, this is much less common compared with TEVAR for trauma. Endograft collapse can be corrected with a balloon expandable bare-metal stent [e.g. Palmaz stent (Cordis Corp., Miami, FL)] [41].

Endograft Separation

Junctional separation of the endograft modular components or a tear or fracture of the endograft is uncommon and can be avoided by generous graft overlapping in the setting of modular stent grafts. When this is seen, a type 3 endoleak is significantly less common following TEVAR for AD compared with aortic aneurysms, as the intact dissection flap usually prevents an endoleak even if the endografts are separated (Fig. 4).

Persistent or Late-Onset False Lumen Perfusion

FLP is the main reason for increasing sac size in the long term. As in the early post-operative period, the main

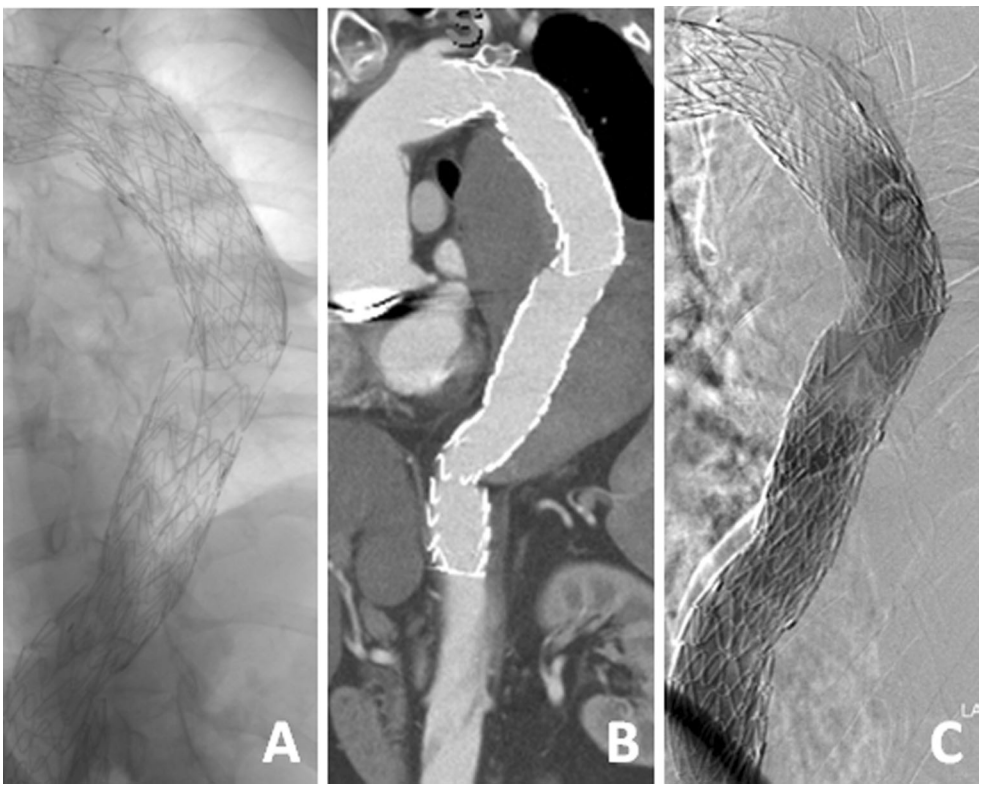


Fig. 4 Endograft separation. Lateral fluoroscopy acquisition (**A**) and sagittal CTA image (**B**) show separation of the thoracic endografts at the mid-descending aorta. Catheter angiogram (**C**) shows no filling of the false lumen because the dissection flap is intact preventing an endoleak

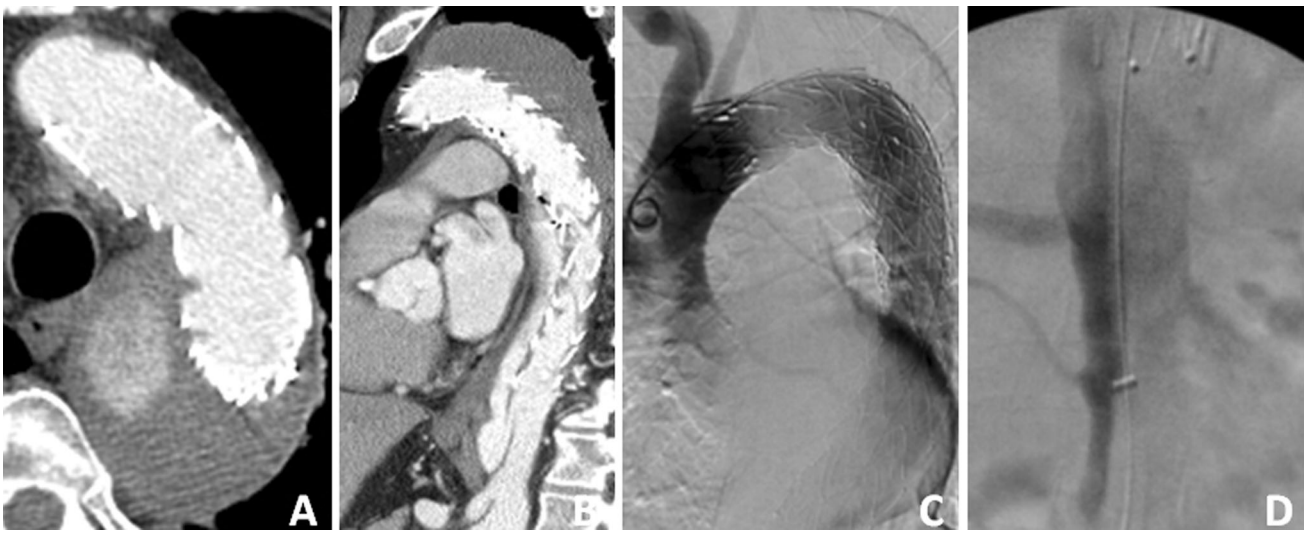


Fig. 5 Application of catheter angiography in identifying the source of false lumen perfusion. Axial (**A**) and Sagittal (**B**) CTA images show persistent false lumen perfusion following TEVAR but the source of this remained undetermined. Catheter angiography (**C**, **D**) performed to assess whether false lumen is filling proximally or distally. Angiogram with the tip of catheter beyond the proximal endograft (**C**) shows no perfusion of the false lumen. Angiogram with the catheter tip positioned in the abdominal aorta (**D**) shows filling of the false lumen, confirming presence of a significant open distal fenestrations

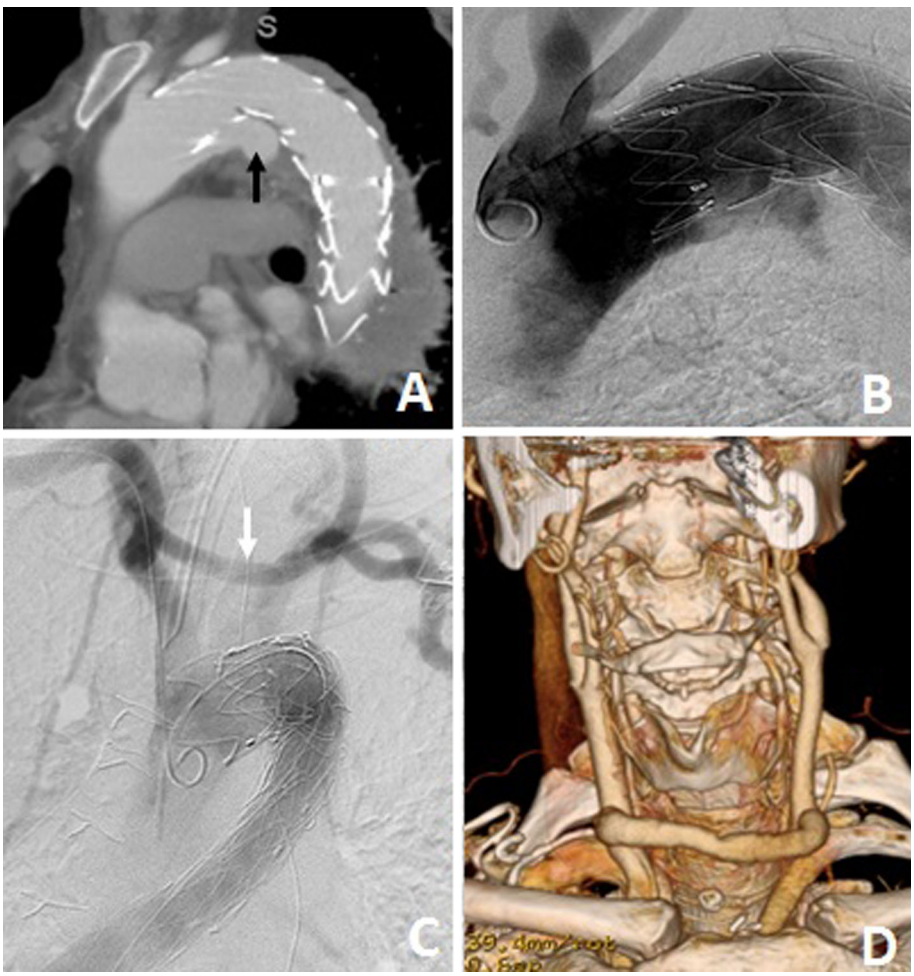


Fig. 6 Persistent proximal false lumen perfusion due to a proximal type 1 endoleak treated by endograft extension and bypass surgery. Coronal oblique CTA image (**A**) shows false lumen filling from the proximal attachment site (*black arrow*). This is confirmed on catheter angiography (**B**) angiogram (**C**) following proximal endograft extension and right to left carotid (*white arrow*) and subclavian bypass (*arrow head*) shows satisfactory exclusion of false lumen and perfusion of the arch vessels. 3D CTA reconstruction (**D**) illustrating right to left carotid bypass

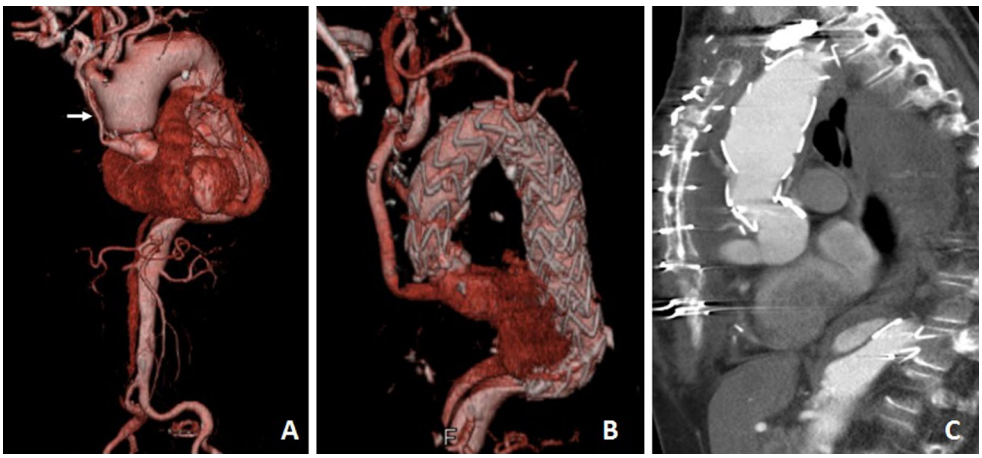


Fig. 7 Hybrid approach for type A aortic dissection. 3D CTA reconstruction (**A**) shows ascending aorta to innominate and left common carotid artery bypass (*arrow*) prior to TEVAR for a type A dissection. 3D CTA image following TEVAR repair with endograft extending from proximal ascending aorta to the abdominal aorta (**B**) shows exclusion of the false lumen. Sagittal CTA image (**C**) showing the corresponding 2D appearance at the level of ascending aorta post-TEVAR

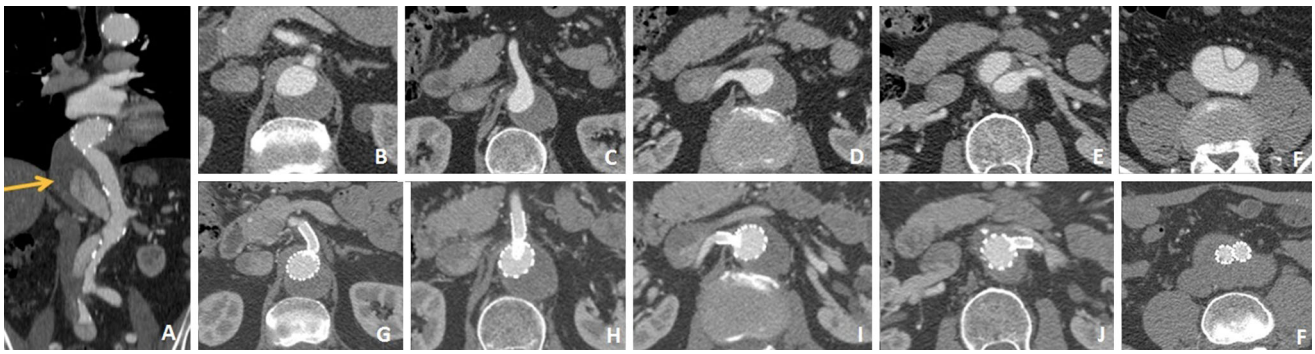


Fig. 8 4 Vessel Fenestration repair for thoracoabdominal aneurysmal dissections. Coronal CTA image (**A**) demonstrating a type B AD extending to the iliac acteries (*arrow*). Axial CTA images before (**A**–**D**) and after (**E**–**H**) placement of a 4-vessel fenestrated aortic endograft in order to achieve total exclusion of the false. Images obtained at the level of coeliac axis (**B**, **G**), superior mesenteric artery (SMA) (**C**, **H**), right (**D**, **I**) and left renal artery (**E**, **J**) and aortic bifurcation (**F**, **K**)

question is whether the FL is filling proximally or distally. If the CTA is equivocal, catheter angiography can be helpful in assessing the site of communication (Fig. 5). Simultaneous flat-panel rotational CT during catheter-directed contrast injection can be further helpful in difficult cases. In the absence of a proximal cause for persistent FLP and good coverage of the proximal fenestration, it can be assumed that the FLP is due to open distal fenestrations in the thoracic or abdominal aorta. Although these are not usually initially treated, if there is an increase in aortic size, treatment by extension of endograft coverage distally, ideally to celiac trunk is indicated.

If a proximal type 1 endoleak FLP is found, this may be new (i.e. late-onset) or long-standing. The latter is typically due to poor coverage of the proximal fenestration at the initial TEVAR procedure and present early. Late-onset causes of proximal FLP include a new proximal tear, expansion of the aortic arch or migration of the stent graft.

Management of FLP

Proximal FLP (i.e. type 1 endoleaks) should be treated by the extension of endograft coverage proximally.

However, the insertion of an endograft more proximally alone may not be possible as there may be insufficient space between the previous endograft and the supra-aortic arteries. Therefore, extending proximal endograft coverage often requires extension to the origin of the left common carotid artery (CCA), and consequent occlusion of the LSA.

Without pre-emptive revascularisation, potential complications of occluding the LSA include left upper extremity ischaemia, posterior circulation stroke (more common in patients with a dominant left vertebral artery), spinal cord ischaemia (due to LSA perfusion of the anterior spinal artery) and reperfusion of the FL via the LSA [42, 43]. However, these complications are not common and despite

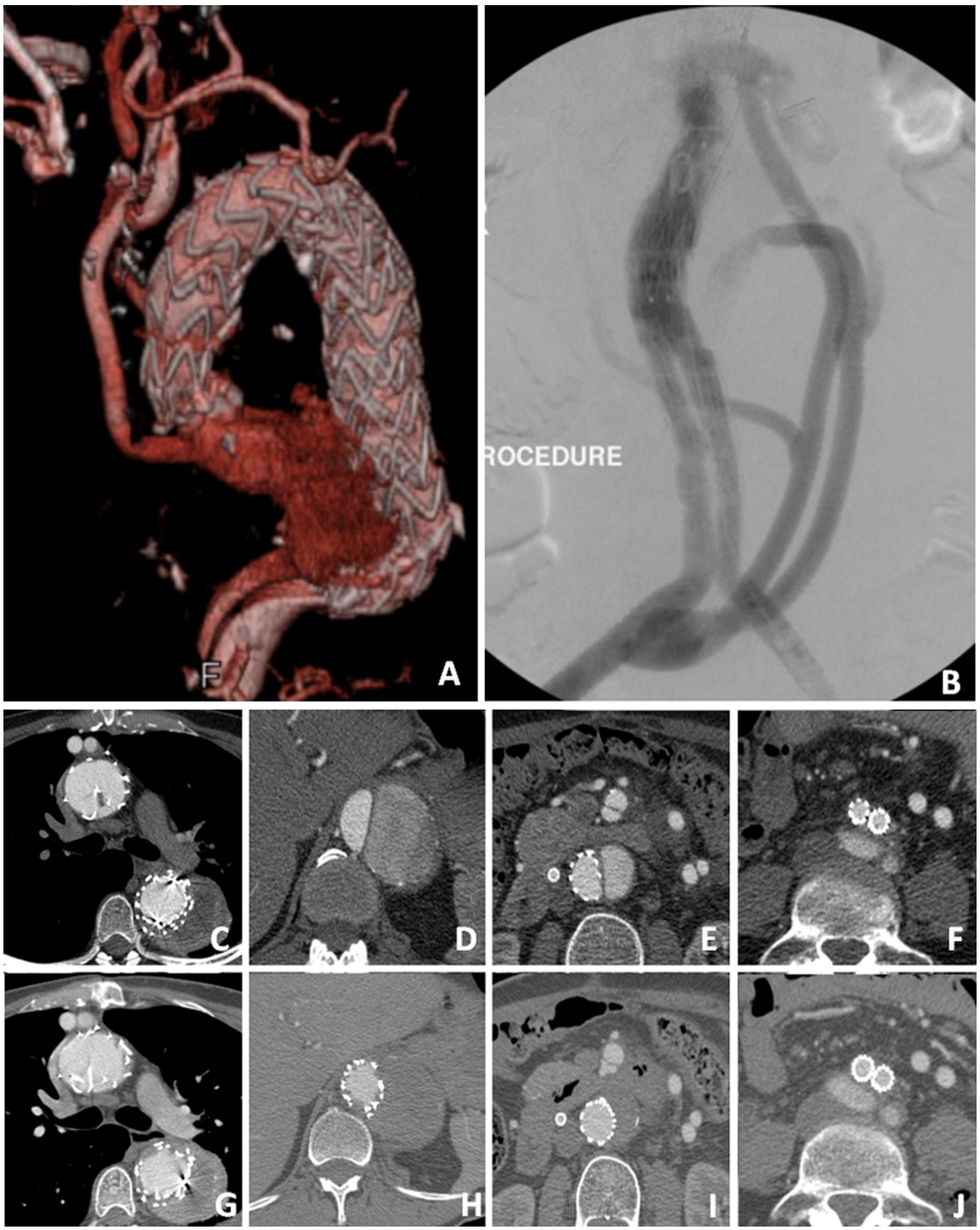


Fig. 9 Hybrid solution for enlarging thoracoabdominal aneurysmal dissections. 3D CTA reconstruction of a patient with previous innominate artery bypass, TEVAR to diaphragm and previous EVAR to Aortobifemoral graft; developing enlarging FL between TEVAR and EVAR endografts (**A**). **B** Angiogram following 4-vessel visceral bypass from right common iliac artery (SMA, Celiac trunk, both renal arteries) and additional aortic endograft stenting to cover the area between the TEVAR and EVAR endografts to achieve total endograft coverage of true lumen. **C**–**F** show axial CTA images before hybrid procedure and **G**–**J** after total exclusion of the false lumen by aortic endograft extension and 4-vessel visceral bypass

the Society for Vascular Surgery guidelines suggesting routine preoperative revascularisation [44], management of the LSA during TEVAR remains controversial. In practice, revascularisation surgery is often only performed expectantly for symptomatic individuals with pre-emptive revascularisation reserved for selected cases such as those with an

incomplete circle of Willis, hypoplastic right vertebral artery, patent left internal mammary artery graft or functional left arm dialysis fistula [42, 45]. Where performed, revascularization procedures generally consist of a transposition of the subclavian artery to the carotid artery or carotid to subclavian artery bypass using a prosthetic polytetrafluoroethylene (PTFE) graft in cases with a very proximal vertebral artery origin [46].

When coverage of the left CCA origin is required, revascularisation is required and is usually achieved by a right to left carotid bypass using a prosthetic graft (Fig. 6). In some cases, extension of endograft coverage to the ascending aorta is needed; necessitating a hybrid (surgical and endovascular) approach involving complete aortic arch debranching. This usually involves surgical bypass to the innominate and left common carotid arteries from the

Fig. 11 **A** Patient with Marfan's syndrome, chronic thoracoabdominal aortic dissection and abdominal aortic aneurysm requiring multiple procedures over many years. Axial CTA images (**A**–**D**) showing chronic type 2 AD extending to the iliac arteries with an aneurysmal abdominal aorta and iliac arteries. Endovascular endograft repair (**E**–**H**). Closure of the main thoracic fenestration with a Talent aortic endograft (**E**, **F**) and a bifurcated aortic endograft placed from the aortic true lumen into the iliac arteries (**G**, **H**). CTA images obtained 2 days post-surgery (**I**–**L**) show persistent false lumen perfusion. **B** Hybrid procedure in the same patient to treat persistent FLP. 18 month follow-up axial CTA images (**A**–**C**) show an increase the size of the abdominal aorta. A hybrid surgical bypass and fenestrated endovascular procedure was performed to close all of the communications to the false lumen by bridging the gap between the thoracic and abdominal endografts (**D**). Visceral bypass grafts from right CIA to SMA, Celiac trunk and RRA prior to endograft placement. It was not technically possible to bypass to the left renal artery and a tube graft with a single fenestration for the left renal artery was inserted (**E**). 3D CTA image **F** following hybrid surgery showing exclusion of the false lumen ▶

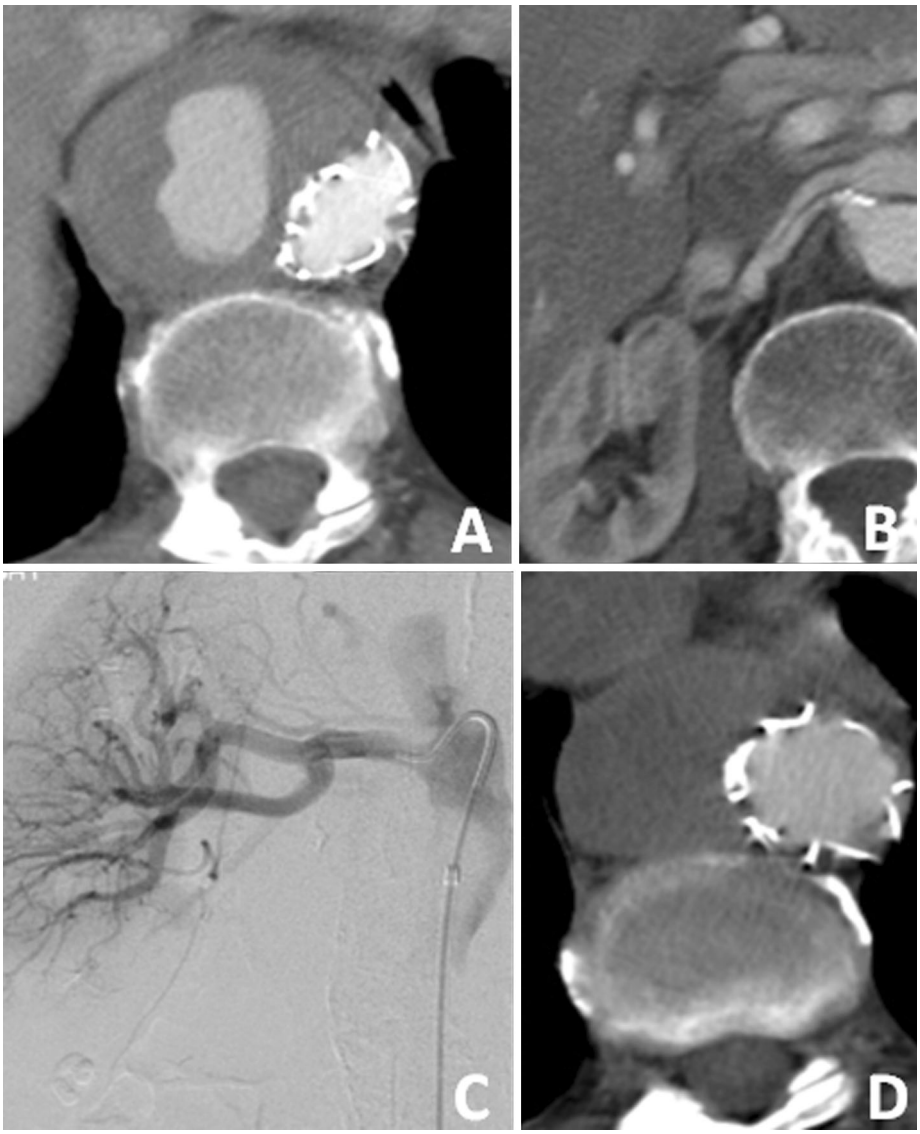
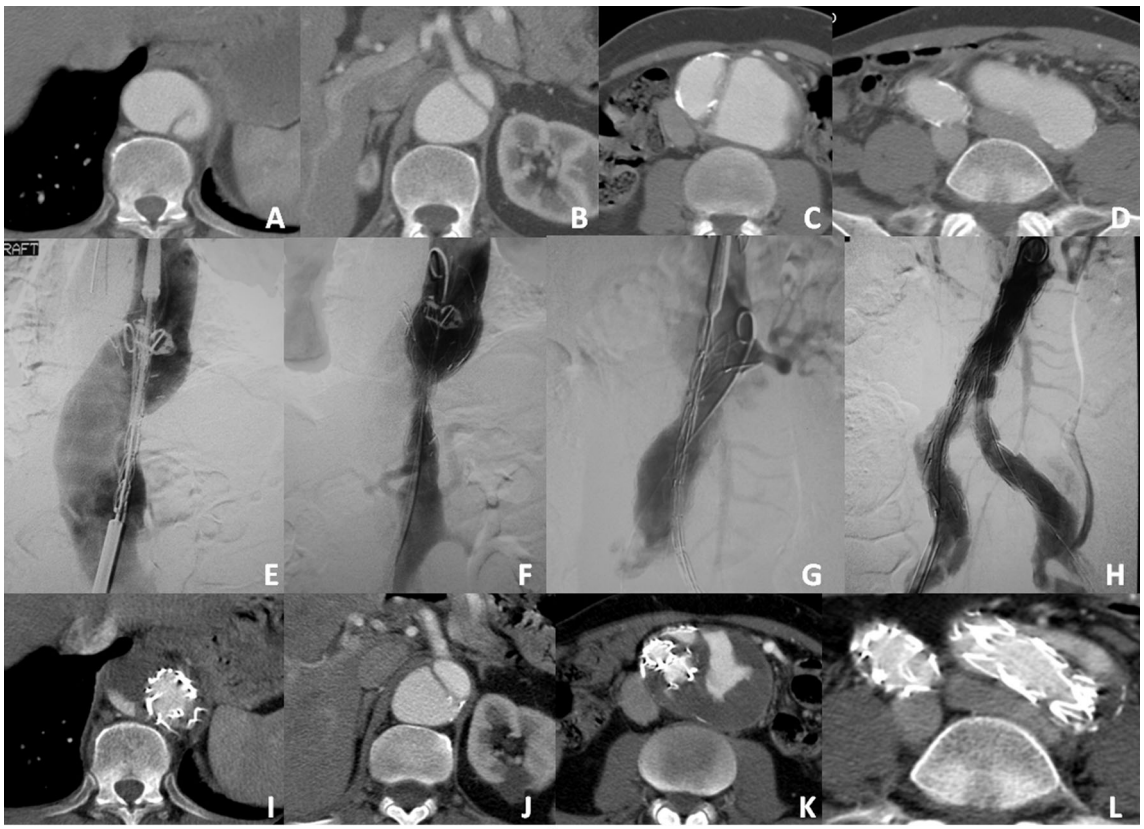
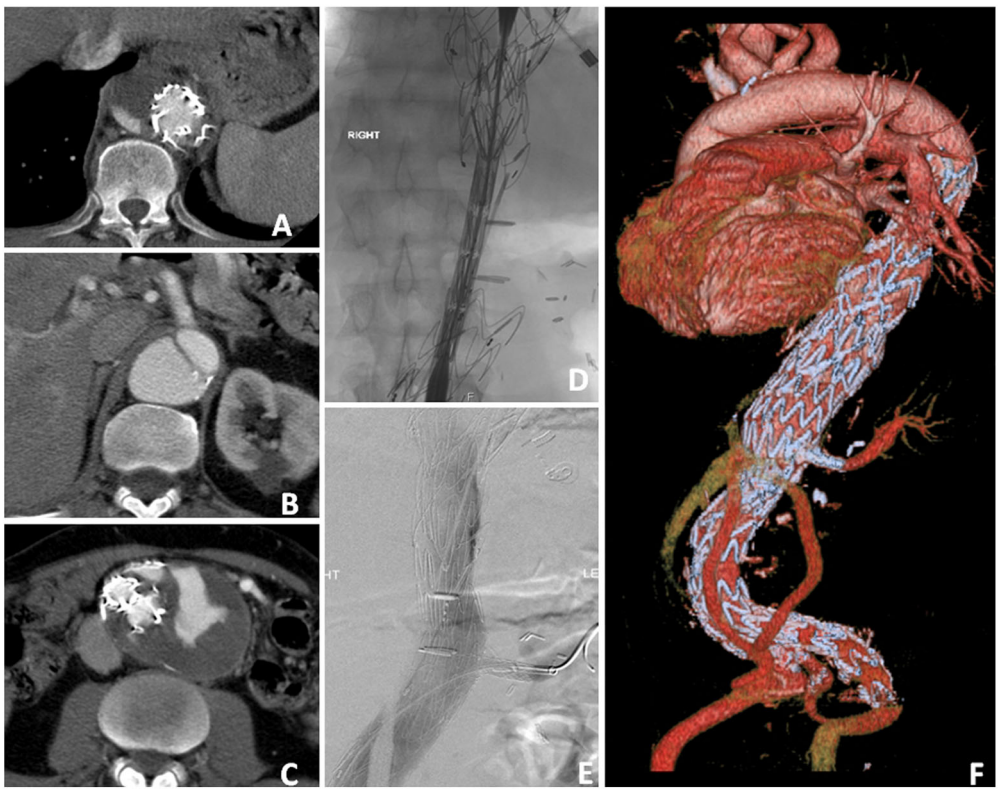


Fig. 10 Treating aneurysmal dissection of thoracoabdominal aorta with true and false lumen communication in a renal artery. Axial CTA image (**A**) shows false FLP following TEVAR for aneurysmal dissection of thoracoabdominal aorta. The only communication between FL and TL was within the right renal artery (**B**). Covered stent placed in the right renal artery to close fenestration and aortic endograft extended (**C**). Subsequent CTA (**D**) shows good result with expansion of the TL, thrombosis of the FL and reduction in aortic diameter



A



B

lower ascending aorta (Fig. 7). Another approach in these cases is the placement of an elephant trunk graft for treatment of ascending/aortic arch lesions and subsequent stent grafting of the descending aorta, with a good overlap between the two prostheses [8]. A "chimney graft" technique has also been described in TEVAR [47, 48]. This is well established for abdominal aortic aneurysms and involves placement of stents in side branches of the aorta alongside the main endovascular stent graft. Custom-made single-branched or multi-branched aortic stent grafts are still under evaluation and are not commercially available for TEVAR.

Late Increase in Thoracoabdominal Aortic Diameter

An inherent disadvantage of TEVAR for AD is that although perfusion of the FL in the endografted thoracic aorta is usually reduced or completely abolished, FL perfusion persists below the level of the endografts due to retrograde flow through distal natural fenestrations or reentry tears. This infers an increased risk of late aneurysmal degeneration and consequent aortic rupture [49]. A review of aortic morphology following TEVAR for AD showed that up to 20 % of patients exhibited distal thoracic aortic dilatation and a significant number of patients exhibited dilatation of the abdominal aorta, with 6 % of cases requiring subsequent repair of an abdominal aortic aneurysm [50]. Proximal stent grafting with distal bare stenting has been proposed to improve TL perfusion and diameter. However, a recent systematic review showed that this fails to completely suppress FL patency and is associated with significant morbidity [49].

Treatment options for isolated infra-renal aortic aneurysms with a dissected but non-aneurysmal thoracic aorta are surgery or EVAR as determined by the aneurysm and iliac anatomy. However, patients with thoracoabdominal aneurysmal dissections require complex treatments including fenestrated or branched endografts (Fig. 8), hybrid procedures that involve surgical bypass grafts to the visceral arteries and endograft coverage of the entire thoracoabdominal aorta (Fig. 9), complete surgical repair or a combination of these options [51, 52]. The aim of treatment in chronic thoracoabdominal aneurysmal dissection is to exclude FLP, which requires closure of all the main communications between the true and FL (Fig. 10). Accurate imaging is essential to establish all communications. Due to the complexity of these cases, multiple subsequent interventions and re-interventions may be required to ensure maintenance of FL exclusion (Fig. 11).

Conclusion

Follow-up CT imaging is mandatory after TEVAR for AD, as there is a lifelong potential for the development of complications of both TEVAR and AD. The main aim of follow-up imaging soon after TEVAR is to detect acute type A dissection and to confirm successful closure of the proximal fenestration by endograft coverage has been achieved. The aims of late follow-up imaging are mainly to detect expansion of the FL above and/or below the diaphragm and to enable selection of the appropriate treatment options for these patients. The aims of treatment for late FL expansion are the closure of important (or all) remaining communications between the TL and the FL. In a large proportion of patients, this may involve combinations of bypass surgery and insertion of additional endografts.

Late rupture due to FL expansion is invariably fatal. Therefore, it is essential that all radiologists who are involved in the assessment of follow-up imaging after TEVAR for AD are able to recognise complications that mandate further therapy.

Conflict of interest Seyed Ameli-Renani, Raj Das, and Robert Morgan have no conflicts of interest.

References

1. Grabenwöger M, Alfonso F, Bachet J et al (2012) Thoracic endovascular aortic repair (TEVAR) for the treatment of aortic diseases: a position statement from the European Association for Cardio-Thoracic Surgery (EACTS) and the European Society of Cardiology (ESC), in collaboration with the European Association. Eur J Cardiothorac Surg 42(1):17–24
2. Criado F (2011) Aortic dissection: a 250-year perspective. Tex Heart Inst J 38(6):694–700
3. Fattori R, Cao P, De Rango P et al (2013) Interdisciplinary expert consensus document on management of type B aortic dissection. J Am Coll Cardiol 61(16):1661–1678
4. Dake MD, Kato N, Mitchell RS et al (1999) Endovascular stent-graft placement for the treatment of acute aortic dissection. N Engl J Med 340(20):1546–1552
5. Grabenwöger M, Alfonso F, Bachet J et al (2012) Thoracic endovascular aortic repair (TEVAR) for the treatment of aortic diseases: a position statement from the European Association for Cardio-Thoracic Surgery (EACTS) and the European Society of Cardiology (ESC), in collaboration with the European Association. Eur Heart J 33(13):1558–1563
6. Fontes-Carvalho R, Braga P, Rodrigues A et al (2012) Treatment of thoracic aortic disease using endovascular stent-grafts: from therapeutic indications to possible complications. Rev Port Cardiol 31:207–214
7. Parsa CJ, Schroder JN, Daneshmand MA, McCann RL, Hughes GC (2010) Midterm results for endovascular repair of complicated acute and chronic type B aortic dissection. Ann Thorac Surg 89(1):97–102 discussion 102–4

8. Iezzi R, Cotroneo AR, Marano R, Filippone A, Storto ML (2008) Endovascular treatment of thoracic aortic diseases: follow-up and complications with multi-detector computed tomography angiography. Eur J Radiol 65(3):365–376
9. Steuer J, Eriksson M-O, Nyman R, Björck M, Wanhainen A (2011) Early and long-term outcome after thoracic endovascular aortic repair (TEVAR) for acute complicated type B aortic dissection. Eur J Vasc Endovasc Surg 41(3):318–323
10. Thrumurthy SG, Karthikesalingam A, Patterson BO et al (2011) A systematic review of mid-term outcomes of thoracic endovascular repair (TEVAR) of chronic type B aortic dissection. Eur J Vasc Endovasc Surg 42(5):632–647
11. Canaud L, Faure EM, Ozdemir BA, Alric P, Thompson M (2014) Systematic review of outcomes of combined proximal stent-grafting with distal bare stenting for management of aortic dissection. Ann Cardiothorac Surg 3(3):223–233
12. Patel AY, Eagle KA, Vaishnava P (2014) Acute type B aortic dissection: insights from the International Registry of Acute Aortic Dissection. Ann Cardiothorac Surg 3(4):368–374
13. Cheng D, Martin J, Shennib H et al (2010) Endovascular aortic repair versus open surgical repair for descending thoracic aortic disease a systematic review and meta-analysis of comparative studies. J Am Coll Cardiol 55(10):986–1001
14. Cao CQ, Bannon PG, Shee R, Yan TD (2011) Thoracic endovascular aortic repair–indications and evidence. Ann Thorac Cardiovasc Surg 17(1):1–6
15. Ueda T, Fleischmann D, Rubin GD, Dake MD, Sze DY (2008) Imaging of the thoracic aorta before and after stent-graft repair of aneurysms and dissections. Semin Thorac Cardiovasc Surg 20(4):348–357
16. Hayter R, Rhea J, Small A (2006) Suspected aortic dissection and other aortic disorders: multi-detector row CT in 373 cases in the emergency setting 1. Radiology 238(3):841–852
17. Rousseau H, Chabbert V, Maracher MA et al (2009) The importance of imaging assessment before endovascular repair of thoracic aorta. Eur J Vasc Endovasc Surg 38(4):408–421
18. Hellinger JC (2005) Endovascular repair of thoracic and abdominal aortic aneurysms: pre- and postprocedural imaging. Tech Vasc Interv Radiol 8(1):2–15
19. Chin AS, Fleischmann D (2012) State-of-the-art computed tomography angiography of acute aortic syndrome. Semin Ultrasound CT MR 33:222–234
20. Fleischmann D, Mitchell RS, Miller DC (2008) Acute aortic syndromes: new insights from electrocardiographically gated computed tomography. Semin Thorac Cardiovasc Surg 20:340–347
21. Chin AS, Fleischmann D (2012) State-of-the-art computed tomography angiography of acute aortic syndrome. Semin Ultrasound CT MR 33(3):222–234
22. Clough RE, Hussain T, Uribe S et al (2011) A new method for quantification of false lumen thrombosis in aortic dissection using magnetic resonance imaging and a blood pool contrast agent. J Vasc Surg 54(5):1251–1258
23. Wicky S, Fan CM, Geller SC, Greenfield A, Santilli J, Waltman AC (2003) MR angiography of endoleak with inconclusive concomitant CT angiography. AJR Am J Roentgenol Am Roentgen Ray Soc 181(3):736–738
24. Hellinger JC (2005) Endovascular repair of thoracic and abdominal aortic aneurysms: pre- and postprocedural imaging. Tech Vasc Interv Radiol 8:2–15
25. Weigel S, Tombach B, Maintz D et al (2003) Thoracic aortic stent graft: comparison of contrast-enhanced MR angiography and CT angiography in the follow-up: initial results. Eur Radiol 13(7):1628–1634
26. Zoli S, Trabattoni P, Dainese L et al (2012) Cumulative radiation exposure during thoracic endovascular aneurysm repair and subsequent follow-up. Eur J Cardiothorac Surg 42(2):254–259 discussion 259–60
27. Stacul F, van der Molen AJ, Reimer P et al (2011) Contrast induced nephropathy: updated ESUR Contrast Media Safety Committee guidelines. Eur Radiol 21(12):2527–2541
28. Thomsen HS (2011) Contrast media safety-an update. Eur J Radiol 80(1):77–82
29. Johnson PT, Black JH, Zimmerman SL, Fishman EK (2012) Thoracic endovascular aortic repair: literature review with emphasis on the role of multidetector computed tomography. Semin Ultrasound CT MR 33:247–264
30. Stanley GA, Murphy EH, Knowles M et al (2011) Volumetric analysis of type B aortic dissections treated with thoracic endovascular aortic repair. J Vasc Surg 54(4):985–992 discussion 992
31. Kim KM, Donayre CE, Reynolds TS et al (2011) Aortic remodeling, volumetric analysis, and clinical outcomes of endoluminal exclusion of acute complicated type B thoracic aortic dissections. J Vasc Surg 54(2):316–324
32. Dake MD (2001) Endovascular stent-graft management of thoracic aortic diseases. Eur J Radiol 39(1):42–49
33. Eggebrecht H, Nienaber CA, Neuhäuser M et al (2006) Endovascular stent-graft placement in aortic dissection: a meta-analysis. Eur Heart J 27(4):489–498
34. Canaud L, Ozdemir BA, Patterson BO, Holt PJE, Loftus IM, Thompson MM (2014) Retrograde aortic dissection after thoracic endovascular aortic repair. Ann Surg 260(2):389–395
35. Eggebrecht H, Thompson M, Rousseau H et al (2009) Retrograde ascending aortic dissection during or after thoracic aortic stent graft placement: insight from the European registry on endovascular aortic repair complications. Circulation 120(11 Suppl):S276–S281
36. Gleason TG (2009) Endoleaks after endovascular aortic stent-grafting: impact, diagnosis, and management. Semin Thorac Cardiovasc Surg 21(4):363–372
37. Desai ND, Bavaria JE (2009) Endovascular treatment of acute descending thoracic aortic dissections. YOTCT 14(2):150–157
38. Pamler RS, Kotsis T, Go J (2002) Complications after endovascular repair of type B aortic dissection. J Endovasc Ther 9:822–828
39. Nienaber CA, Kische S, Ince H (2007) Thoracic aortic stent-graft devices: problems, failure modes, and applicability. Semin Vasc Surg 20:81–89
40. Patel PJ, Grande W, Hieb RA (2011) Endovascular management of acute aortic syndromes. Semin Intervent Radiol 28(1):10–23
41. Steinbauer MGM, Stehr A, Pfister K et al (2006) Endovascular repair of proximal endograft collapse after treatment for thoracic aortic disease. J Vasc Surg 43(3):609–612
42. Feezor RJ, Lee WA (2009) Management of the left subclavian artery during TEVAR. Semin Vasc Surg 22(3):159–164
43. Klocker J, Koell A, Erlmeier M, Goebel G, Jaschke W, Fraedrich G (2014) Ischemia and functional status of the left arm and quality of life after left subclavian artery coverage during stent grafting of thoracic aortic diseases. J Vasc Surg 60(1):64–69
44. Matsumura JS, Lee WA, Mitchell RS et al (2009) The Society for Vascular Surgery Practice Guidelines: management of the left subclavian artery with thoracic endovascular aortic repair. J Vasc Surg 50(5):1155–1158
45. Kotelis D, Geisbüsch P, Hinz U et al (2009) Short and midterm results after left subclavian artery coverage during endovascular repair of the thoracic aorta. J Vasc Surg 50(6):1285–1292
46. Vallabhaneni R, Sanchez LA (2010) Open techniques for arch vessel reconstruction during thoracic endovascular aneurysm repair (TEVAR). J Vasc Surg 52(4 Suppl):71S–76S
47. Hogendoorn W, Schlösser FJ, Moll FL, Sumpio BE, Muhs BE (2013) Thoracic endovascular aortic repair with the chimney graft technique. J Vasc Surg Soc Vasc Surg 58(2):502–511

48. Shahverdyan R, Gawenda M, Brunkwall J (2013) Triple-barrel graft as a novel strategy to preserve supra-aortic branches in arch-TEVAR procedures: clinical study and systematic review. Eur J Vasc Endovasc Surg 45(1):28–35
49. Canaud L, Faure EM, Ozdemir BA, Alric P, Thompson M (2014) Systematic review of outcomes of combined proximal stent-grafting with distal bare stenting for management of aortic dissection. Ann Cardiothorac Surg 3:223–233
50. Sayer D, Bratby M, Brooks M, Loftus I, Morgan R, Thompson M (2008) Aortic morphology following endovascular repair of acute and chronic type B aortic dissection: implications for management. Eur J Vasc Endovasc Surg 36(5):522–529
51. Kitagawa A, Greenberg RK, Eagleton MJ, Mastracci TM, Roselli EE (2013) Fenestrated and branched endovascular aortic repair for chronic type B aortic dissection with thoracoabdominal aneurysms. J Vasc Surg Soc Vasc Surg 58(3):625–634
52. Oikonomou K, Kopp R, Katsargyris A, Pfister K, Verhoeven EL, Kasprzak P (2014) Outcomes of fenestrated/branched endografting in post-dissection thoracoabdominal aortic aneurysms. Eur J Vasc Endovasc Surg 48:1–8