

Retrograde Type A Aortic Dissection

A review of the available literature on this rare but potentially catastrophic complication.

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The definition of *acute thoracic aortic syndrome* has usually included ruptured thoracic aneurysm, acute type B dissection, traumatic blunt aortic injury, and symptomatic penetrating aortic ulcer. Retrograde type A aortic dissection (RTAD) is defined as a dissection that originates distally to the ascending aorta with retrograde flap progression into the ascending aorta.¹ It was a known complication during conventional cardiac surgery, but recently, it has been described as one of the most alarming complications after thoracic aortic endovascular repair (TEVAR).² It is potentially lethal and in all respects deserves to be included in acute thoracic aortic syndrome.

INCIDENCE

The true incidence of RTAD has not yet been established, but it is an increasingly observed phenomenon, with an estimated overall incidence of 1% to 4%.³⁻⁶ In their recent systematic meta-analysis, Luebke and Brunkwall⁷ calculated a weighted event rate of 7% for RTAD. This figure significantly exceeds the 0% to 7.5% incidence rate reported in even the most extensive series, but it should be pointed out that their review analyzed only acute RTAD after complicated type B aortic dissections, whereas the others presented mixed entities of RTADs. Therefore, the real incidence for RTAD reported in the literature could be underestimated.

Aortic specialists must be aware that RTAD may have an acute or delayed presentation, potentially occurring acutely during either the indexed TEVAR procedure or in the postoperative period. Dong et al⁴ detected an acute presentation in 27.3% of their first 11 cases, with a mean onset time of 11 ± 16 months in a recently updated analysis of 23 cases.⁸ An insight analysis from the European registry on endovascular aortic repair complications revealed that RTAD occurred dur-

ing the indexed procedure in 35.4% of the cases and in 64.6% within the first 30 days.⁵ Most importantly, even though it is rare, RTAD has been detected as an incidental finding during a follow-up control, although with an asymptomatic clinical profile.

PREDISPOSING FACTORS

The limited experience compiled on RTAD raises concerns regarding whether or not it is the consequence of a single independent event, because the available data seem to support the hypothesis of a combination of different causes. RTAD etiopathogenesis could be distinguished as:

- Disease related, due to the natural progression of the underlying disease
- Procedure related, when caused by ballooning or wire and sheath manipulation, typically in an acute aortic arch
- Endograft related, also defined as a stent graft–induced new entry site (SINE), which refers to wall injury that seems potentially related to the use of endografts. This type of aortic lesion has frequently been identified intraoperatively during open conversion.

Most cases have been detected after TEVAR for type B dissections or its variants. It is suggested to consider the fragility of the aortic wall (meaning the predominant presence of dissection, intramural hematoma, or connective tissue disorders like Marfan syndrome) as the pathological background and TEVAR maneuvers as the provoking trigger. Out of the 71 cases piled up from two of the most extensive series reported on RTAD, the presence of a connective tissue disorder accounted for 8.4% of the underlying disease.^{4,5} In particular, the proportion of new SINE among Marfan syndrome patients was 33.33% in the study by Dong et al,⁸ which was significantly higher than the 3.26% among non-Marfan patients.

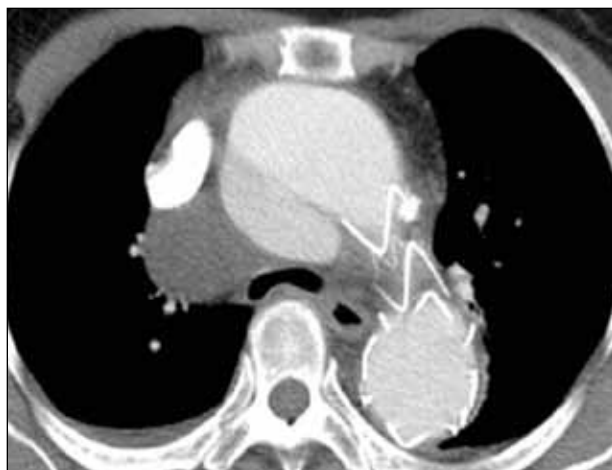


Figure 1. RTAD following TEVAR in an acute arch. The proximal SINE was located near the endograft (proximal bare spring).

Recently, Williams et al⁹ identified an ascending aorta diameter of > 40 mm as another potential predictor of RTAD and reported a higher incidence of this complication in such patients (4.8% vs 0.9%; $P = .047$). The incidence of RTAD increased markedly with combinations of higher-risk scenarios, such as the association of dissection plus an ascending aortic diameter ≥ 40 mm, which further increased to 25% using the native “zone 0” as a proximal landing zone for the endograft. A dilated ascending aorta or similar condition—such as a bicuspid aortic valve—are likely markers of diffuse aortic disease and is an inherent weakness of the diseased aortic wall, thus predisposing patients to this complication. Other anatomic conditions at the proximal neck may play a primary role in determining RTAD after TEVAR, such as the presence of an angulated ($> 60^\circ$) “gothic” arch and the absence of a regular neck for safe deployment, which is frequently detected in acute B dissection and may be associated with retrograde dissection.¹⁰

Several authors have reported that reiterative balloon remodeling of the endograft may cause RTAD.² The ballooning was judged to be necessary because the endograft did not adapt perfectly to the inner curve of an angulated aortic arch. However, it should be observed that in most of these reported instances, a “fragile” aorta was the indication for TEVAR intervention.

Further procedure-related RTAD-provoking factors were recently described to be strictly linked to the debranching technique.^{11,12} The risk of wall injury during aortic side-clamping under pulsatile flow has been documented during off-pump coronary artery bypass surgery, especially in the presence of a dilated ascending

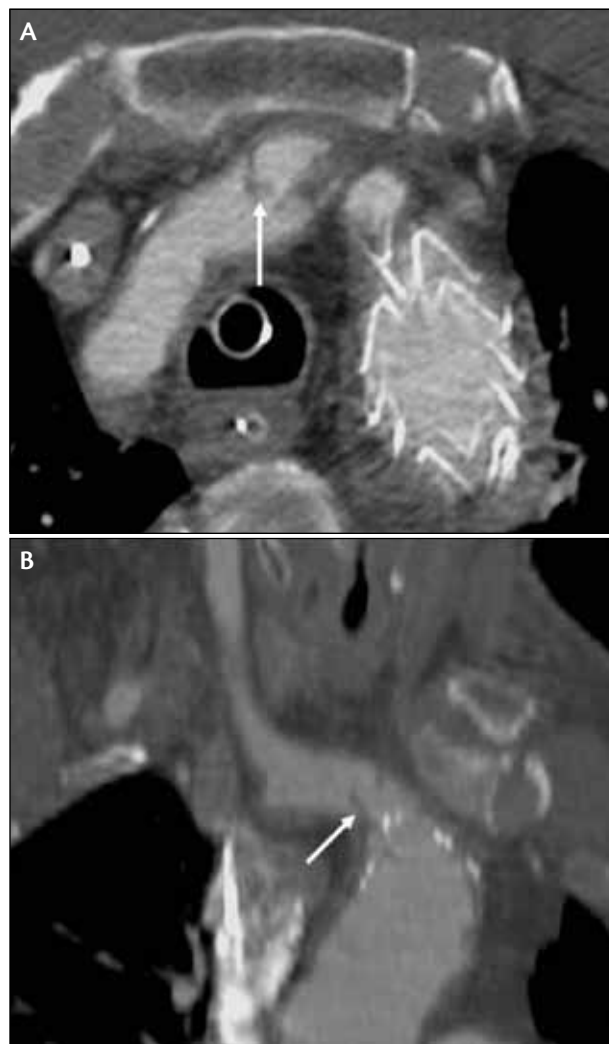


Figure 2. A case of RTAD following TEVAR for an aortic arch aneurysm. SINE involved the supra-aortic trunks (A, arrow) and was located at the proximal edge (B, arrow) of the endograft (no bare spring).

aorta. The risk of developing RTAD after total arch debranching could be amplified by the large anastomosis required to complete the rerouting of the supra-aortic vessels. In support of this hypothesis, an incremental risk of RTAD was observed for the “zone 0” proximal landing zone and a dilated (≥ 4 cm) ascending aorta.⁹ The combination of these scenarios associated with use of endografts with proximal barbs showed an overall 10.7% incidence of RTAD.^{6,9} This finding is of particular importance in light of data published by van Prehn et al,¹³ who illustrated the significant pulsatility of the ascending aorta. This might further be evaluated as a concurrent mechanism for the increased incidence of RTAD that is seen with the “zone 0” proximal landing zone.

One of the most debated predisposing factors of RTAD has been the configuration of the endograft. Currently, we do not have a definitive indication that a specific configuration may significantly increase the risk of developing RTAD. Injury from endografts featuring a proximal bare spring (Figure 1) was considered first because this configuration was designed to strengthen proximal fixation. However, proximal SINE has also been observed in patients treated with endografts without the bare spring. In the experience of Kpodonu et al,³ the RTAD incidence among endografts without bare springs (Figure 2) was 2.4%, which was fairly similar to the 2.5% incidence reported in a Chinese study using endografts with bare springs.^{4,8} In a European registry, the evidence that RTAD tended to occur using many different endografts suggested that the semirigid design of the endograft might be responsible for the aortic tear rather than the proximal bare springs.⁵ Therefore, we can speculate that the majority of endograft-related RTADs could be ascribed to the lack of pathology-specific devices, especially for those cases involving a “fragile” aorta.⁷

It has been suggested that the radial force of the endograft, which also depends on its oversizing, in combination with the inherent tendency of the endograft to spring back to its initial straight status, generates stress on the aortic curvature and may provoke the intimal injury. If oversizing is part of the dynamic effect that contributed to the development of RTAD, it could be speculated that excessive endograft oversizing (> 20%) should be frequently detected in RTAD cases, regardless of the primary aortic disease. Kpodonu et al³ reported a mean 21.4% oversizing as the causative factor in 28% of their RTADs, but a mean of only 9.6% was observed in the acute cases. However, RTADs have been reported with increased frequency, even with low oversizing rates, so oversizing as a determining factor for RTAD does not seem to be considered as playing a primary role.

OUTCOMES AND RESULTS OF SURGICAL REPAIR

Mortality rates after RTAD have been reported up to 42%, including sudden deaths, and have shown to be higher than the rate for spontaneously occurring acute type A aortic dissection.^{2,5,7} Analysis of subgroups showed that patients in whom RTAD occurred during the TEVAR procedure, especially those with a proximal SINE, had the worst outcomes compared with patients in whom RTAD occurred during follow-up.⁸ Hence, subsequent open conversion should be the treatment of choice in an effort to avert these life-threatening com-

plications. In the report from the Duke group, 66% of RTADs occurred intraoperatively, 75% of which were initially detected by transesophageal echocardiography or intravascular ultrasound.^{6,9} These data suggest that rapid diagnosis is of paramount importance and that intraoperative diagnostic tools may play a key role in facilitating early detection of RTAD.

RTADs ascribed to wire/sheath manipulation seem to be limited to the distal ascending aortic arch or proximal aortic arch in half of patients.⁵ In addition, proximal SINE was more frequently located at the greater curve and involved the entire ascending aorta in the majority (83%) of cases.⁸ Therefore, the presumed cause and location of RTAD may have a significant influence on treatment strategy. The substitution of the entire aortic arch with suturing of the vascular graft directly to the endograft was the preferred type of surgical repair.^{2,14} The complete removal of the endograft and Dacron graft replacement using the modified “elephant trunk” technique was rarely performed as an alternative strategy.^{1,2} Conservative treatment was used in selected cases, especially for those with wire-induced wall injury or in the presence of focal and asymptomatic lesions.²

RECOMMENDATIONS

RTAD after TEVAR is a serious potential complication that may occur either intraoperatively or during follow-up. Due to the substantial mortality of RTAD, larger registries are needed to provide the necessary information to better define a treatment strategy. Similarly, further studies are needed to clarify the role and incidence of spontaneous retrograde extension of type B dissection in patients both with and without previous TEVAR. Nevertheless, RTAD during or after TEVAR may be considered a new acute aortic syndrome based on its recent description, anatomical involvement, and clinical characteristics.

Definitive risk factors have not yet been identified, although it seems reasonable to note the potential of some conditions in predisposing RTAD, such as the presence of connective tissue disorders in patients affected by type B dissection. A predictive score might help physicians lessen the risk of RTAD, especially during TEVAR.

Efforts to minimize the occurrence of this complication may focus on several strategic elements:

Careful patient selection. Close attention to specific etiologies of the underlying aortic disease is extremely important. A “fragile aorta” looks more susceptible to developing RTAD, especially in patients with aortic dissection or those who are affected by connective tissue disorders.

CASE REPORT

As reported in the *Journal of Vascular Surgery* in April 2010,¹ a 76-year-old man was referred to our department and presented with an asymptomatic penetrating thoracic aortic ulcer (PAU) that had developed from a previous acute aortic syndrome caused by a type B intramural hematoma that was managed medically. The preliminary thoracoabdominal computed tomographic (CT) scan showed an acute (60°) aortic arch with an enlarged (maximum diameter, 47 mm) ascending aorta and the presence of a PAU (Figure 1A). Under general anesthesia, the right common femoral artery was exposed in a standard fashion, and a single endograft (36-mm X 15-cm TAG, Gore & Associates, Flagstaff, AZ) was deployed during controlled hypotension (< 90 mm Hg), with planned partial overstenting of the origin of the left subclavian artery. To better adapt the endograft to the inner curve of the aortic arch, gentle ballooning was performed once inside the proximal extremity of the endograft. Final control imaging confirmed complete exclusion of the ulcer and adequate endograft adherence to the aortic curvature.

Four hours later, while the patient was being extubated, profound and persistent hypotension and a disparity of the pupils were noted. Immediately, CT angiography was performed, showing exclusion of the PAU but the development of an RTAD due to a proximal SINE with a retrograde extension to the ascending aorta and causing hemopericardium. Both the origin of the brachiocephalic trunk and the left common carotid artery were dissected, and the true lumen was compressed by the false lumen (Figure 1B).

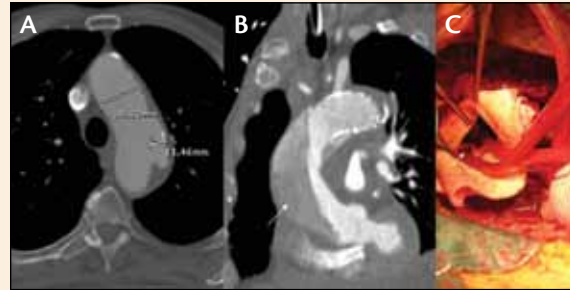


Figure 1. A case of RTAD. Preoperative CT angiography scan before TEVAR for a penetrating aortic ulcer of the distal arch (A). Postoperative maximum intensity projection CT angiography (B) showing the RTAD involving the ascending aorta (arrow). Intraoperative view (C) showing the proximal SINE at the proximal edge of the endograft on the outer curve of the distal arch (arrow).

Immediate surgical repair was performed of the total ascending/arch using hypothermic circulatory arrest. The ascending aorta and the proximal ventral part of the aortic arch were replaced with a 30-mm woven graft (Uni-Graft, B. Braun Interventional Systems, Inc., Bethlehem, PA) with epiaortic vessel reimplantation. The distal part of the graft was sutured to the proximal end of the endograft with a polypropylene suture. Intraoperatively, we confirmed the proximal SINE in correspondence to the proximal end of the endograft (Figure 1C).

1. Piffaretti G, Mariscalco G, Tozzi M, et al. Acute iatrogenic type A aortic dissection following thoracic aortic endografting. *J Vasc Surg.* 2010;51:993-999.

Technical aspects. Avoid excessive catheter and guide-wire manipulations, landing zones in a severe angulation (“gothic arch”) or in a dilated ascending aorta, and balloon dilatation when not absolutely required; oversizing of the endograft should be restricted to 10% in acute cases or “fragile aortas.”

Device options. RTAD has been reported in all device configurations, either with or without bare springs. Many authors have urged manufacturers to provide an endograft designed specifically for use in difficult anatomies or delicate etiologies. This has led multiple manufacturers to address device implications and improve their platforms in this regard, particularly in terms of conformability to the aortic anatomy. Technology continues to develop, and additional endograft refinements may further decrease RTAD-related morbidity and mortality. ■

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