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Hellenic Journal of Vascular and Endovascular Surgery

Contents

EDITORIAL

Chronic Limb-Threatening Ischaemia (CLTI) Revascularisation: Which Patients Benefit from Endovascular Therapy and Which from Open Bypass?

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Chronic limb-threatening ischaemia (CLTI) represents the most severe clinical manifestation of peripheral arterial disease (PAD), characterised by rest pain, tissue loss, or both, and associated with a markedly elevated risk of limb loss, cardiovascular events, and premature mortality. Patients presenting with CLTI are not defined solely by their arterial pathology; rather, they represent a complex, multimorbid population frequently burdened by diabetes mellitus, chronic kidney disease, coronary artery disease, frailty, and social deprivation. These co-existing conditions profoundly influence both procedural risk and long-term outcomes, and therefore must be central to any revascularisation strategy.[1,2]

Despite decades of innovation in both surgical and endovascular techniques, the fundamental question remains unresolved: which patients benefit most from endovascular therapy, and which from open surgical bypass? The answer is nuanced, highly individualised, and dependent on anatomical, physiological, and system-level considerations. Importantly, it is also shaped by the evolving evidence base derived from randomised controlled trials.

The original BASIL (Bypass versus Angioplasty in Severe Ischaemia of the Leg) trial was a landmark study that provided the first high-quality evidence comparing bypass surgery with balloon angioplasty in patients with severe limb ischaemia and femoro-popliteal disease.[3] Its principal finding - that patients with a life expectancy exceeding two years and suitable anatomy for bypass derive greater long-term benefit from surgical revascularisation - continues to influence contemporary decision-making. However, a critical limitation of BASIL was the absence of a defined or standardised assessment of patient fitness. Clinicians were left to interpret "expected survival" without clear criteria, a challenge that persists in modern practice.

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Equally striking was the poor overall survival observed in BASIL (Bypass versus Angioplasty in Severe Ischaemia of the Leg), a finding that has remained largely unchanged in subsequent trials conducted decades later. BASIL-2, BASIL-3, and BEST-CLI (Best Endovascular versus Best Surgical Therapy in Patients with Critical Limb Ischemia), (major studies published more recently) have all reported similarly sobering mortality rates, despite substantial advances in endovascular technologies, perioperative care, and imaging.[4-6] This stagnation in survival highlights a crucial point: revascularisation alone cannot address the systemic atherosclerotic burden that defines CLTI.

Indeed, there is compelling evidence that best medical therapy (BMT) - including antiplatelet agents, statins, blood pressure control, glycaemic optimisation, and smoking cessation - confers a substantial survival benefit in patients with symptomatic PAD.[7-9] Yet, real-world data consistently demonstrate suboptimal implementation of these therapies.[8,9] This therapeutic gap represents a missed opportunity to improve outcomes in a population at exceptionally high cardiovascular risk.

A study by our group CHABLIS (Community and Hospital cAre Bundle to improve the medical treatment of severe cLaudIcation and critical limb iSchaemia) further reinforces the importance of structured, multidisciplinary care pathways in CLTI.[9] By integrating vascular intervention with optimisation of medical therapy, wound care, and patient education, and other complex clinical interventions we can meaningfully improve adherence to BMT and overall patient outcomes; this should be the mainstay of CLTI treatment. This underscores that revascularisation should not be viewed in isolation, but as one component of a broader, holistic CLTI-management strategy.

Turning to contemporary trial evidence, the BEST-CLI trial provides critical insights into the comparative effectiveness of surgical and endovascular strategies.[6] In patients with suitable great saphenous vein conduit and infra-inguinal disease, a bypass-first approach was associated with superior limb-related outcomes compared with endovascular intervention. However, this benefit was contingent upon the availability of high-quality venous conduit. In the absence of such conduit, outcomes between strategies were more closely aligned, highlighting the importance of patient selection.

Similarly, BASIL-3 evaluated endovascular technologies in patients with infrapopliteal disease and CLTI, demonstrating that an endovascular-first strategy is currently the most prag-

matic approach in this cohort.[5] While no single device or technique emerged as unequivocally superior, the trial reflects the ongoing evolution of endovascular therapy and its central role in contemporary practice.

It is also important to consider the SWEDEPAD (Swedish Drug-Elution Trial in Peripheral Arterial Disease) trial, which examined drug-coated devices in PAD and CLTI populations.[10] Although not directly comparing surgical and endovascular strategies, its findings contribute to the broader understanding of device safety and efficacy in this space.

Emerging evidence suggests that adjunctive technologies such as vessel preparation - encompassing atherectomy, intravascular lithotripsy, and specialty balloons - may enhance the durability of endovascular interventions, particularly in heavily calcified or complex lesions.[11,12] While these approaches are promising, robust randomised data remain limited, and their role within treatment algorithms continues to evolve.

From a pragmatic standpoint, several principles can be distilled from the available evidence. First, patients with CLTI and femoropopliteal disease who are deemed fit for surgery - based on local or regional assessments, which remain poorly standardised - and who possess suitable autologous venous conduit, are likely to benefit from a bypass-first strategy, particularly in the context of complex or long-segment disease. This approach is supported by BEST-CLI and aligns with the long-term findings of BASIL.

Second, for patients with infrapopliteal disease, an endovascular-first strategy is generally appropriate. This is particularly relevant given the anatomical challenges and the morbidity associated with distal bypass procedures. However, the success of this approach is contingent upon meticulous optimisation of medical therapy and wound care, without which revascularisation alone is unlikely to achieve durable limb salvage.

Third, the concept of patient “fitness” must be refined. Current literature provides little guidance on how best to assess physiological reserve, frailty, or operative risk in this population. There is an urgent need for validated tools that can inform decision-making and ensure that patients are matched to the most appropriate intervention.

Beyond the choice of revascularisation strategy, it is essential to recognise that CLTI care extends far beyond the operating theatre or catheter laboratory. Holistic management is the cornerstone of effective treatment and must include aggressive risk factor modification, structured rehabilitation, and high-quality wound care. Early intervention is also critical; delays in revascularisation are associated with worse outcomes, and a target of intervention within three days of decision-making should be considered a benchmark for best practice.

The economic implications of CLTI management further emphasise the need for effective, durable interventions. Limb salvage strategies, while resource-intensive upfront, are associated with lower long-term costs compared with major amputation, which carries substantial healthcare and societal burdens.[13,14] Reinterventions, particularly target lesion revascularisations, contribute significantly to overall costs and highlight the importance of selecting the most durable initial strategy.[14]

Finally, the importance of ongoing research cannot be overstated. Despite recent advances, many questions remain unanswered, particularly regarding optimal patient selection, the role of emerging technologies, and the integration of medical and interventional therapies. Participation in randomised controlled trials should be considered a fundamental component of CLTI care where available. Moreover, there is a pressing need for continued investment in high-quality research at both national and institutional levels.

In conclusion, the management of CLTI requires a nuanced, patient-centred approach that integrates anatomical considerations with physiological assessment and holistic care. While surgical bypass offers clear benefits in selected patients with suitable conduit and life expectancy, endovascular therapy remains the cornerstone for many, particularly those with infrapopliteal disease or significant comorbidity. Ultimately, the goal is not merely limb salvage, but the preservation of life and quality of life in a population at extreme risk. Achieving this will require not only technical excellence, but also a commitment to comprehensive care and continued scientific enquiry.

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ORIGINAL ARTICLE

Early and Late Outcomes Among Patients Undergoing Carotid Endarterectomy with a Contralateral Internal Carotid Artery Occlusion or Severe Stenosis

George Galyfos, Konstantinos Saliaris, Maria Kapoutsi, Georgios Sigkounas, Iasonas Priftis, Marios Moula, Nikos Ntanos, Antonios Palaios, Frangiska Sigala, Konstantinos Filis

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Abstract:

Introduction: The role of prophylactic carotid endarterectomy (CEA) in asymptomatic octagenarians remains controversial. However, a contralateral internal carotid artery (ICA) occlusion or severe stenosis may be associated with a higher stroke risk among these patients. Aim of this study is to evaluate early and late outcomes among patients with severe contralateral ICA disease, and to further compare outcomes between asymptomatic patients older and younger than 80 years of age.

Methods: This was a retrospective study. All patients with a contralateral ICA occlusion or ≥80% stenosis undergoing CEA in our Vascular Unit within five years (01/2020-12/2024) were included. All basic characteristics were recorded. Early and late outcomes were evaluated. All outcomes were also compared between asymptomatic patients ≥ and < 80 years old.

Results: In total, 47 patients (18 with a contralateral ICA occlusion and 29 with a contralateral ≥80% ICA stenosis) were included. Mean stenosis of the treated carotid artery was 82±7%. Overall, 42% (n=20) of patients were symptomatic (11 transient ischemic attacks (TIAs) and 9 strokes). Regarding early outcomes, mortality was null, stroke rate was null, TIA rate was 4%, bleeding/major hematoma rate was 4%, cranial nerve injury was 4% and symptomatic myocardial infarction rate was 4%. Within a mean follow-up of 22 ± 4 months, no new stroke or TIA was reported. Eight late deaths of other causes were observed. When asymptomatic patients <80 years old (n=18) were compared with asymptomatic patients ≥80 years old (n=9), perioperative outcomes were not different.

Conclusion: Early and late outcomes after CEA are acceptable among patients with a severe contralateral ICA disease. Asymptomatic patients over 80 years old with a severe contralateral ICA disease seem to have similar outcomes compared to younger patients. Therefore, surgery should be considered for eligible older patients within this high-risk subgroup.

Keywords: contralateral occlusion; internal carotid artery stenosis; asymptomatic; octagenarians

INTRODUCTION

According to recent guidelines on proper management of atherosclerotic carotid disease, asymptomatic patients with >60% internal carotid artery (ICA) stenosis should preferably be treated conservatively.[1] Especially for octagenarians, the role of prophylactic carotid endarterectomy (CEA) remains controversial. There are, however, some specific factors that could increase the risk for stroke and suggest a benefit from carotid intervention. Such factors include contralateral ICA occlusion or severe contralateral stenosis.[2] Octagenarians have long been considered of high risk for any intervention and have been excluded from the NASCET (North American symptomatic carotid endarterectomy trial) and ACAS (asymptomatic carotid atherosclerosis study) trials.[3-5] Additionally, these trials have recruited a relatively small number of patients with contralateral occlusion or severe contralateral ICA stenosis.[4,5]

Therefore, aim of this study is to evaluate early and late outcomes in a series of patients with contralateral carotid occlusion or severe contralateral stenosis undergoing CEA. The effect of increased age over 80 years old among asymptomatic patients will be also evaluated among these patients.

METHODS

In this retrospective study, all patients undergoing a CEA with contralateral ICA occlusion or contralateral significant ICA ste-

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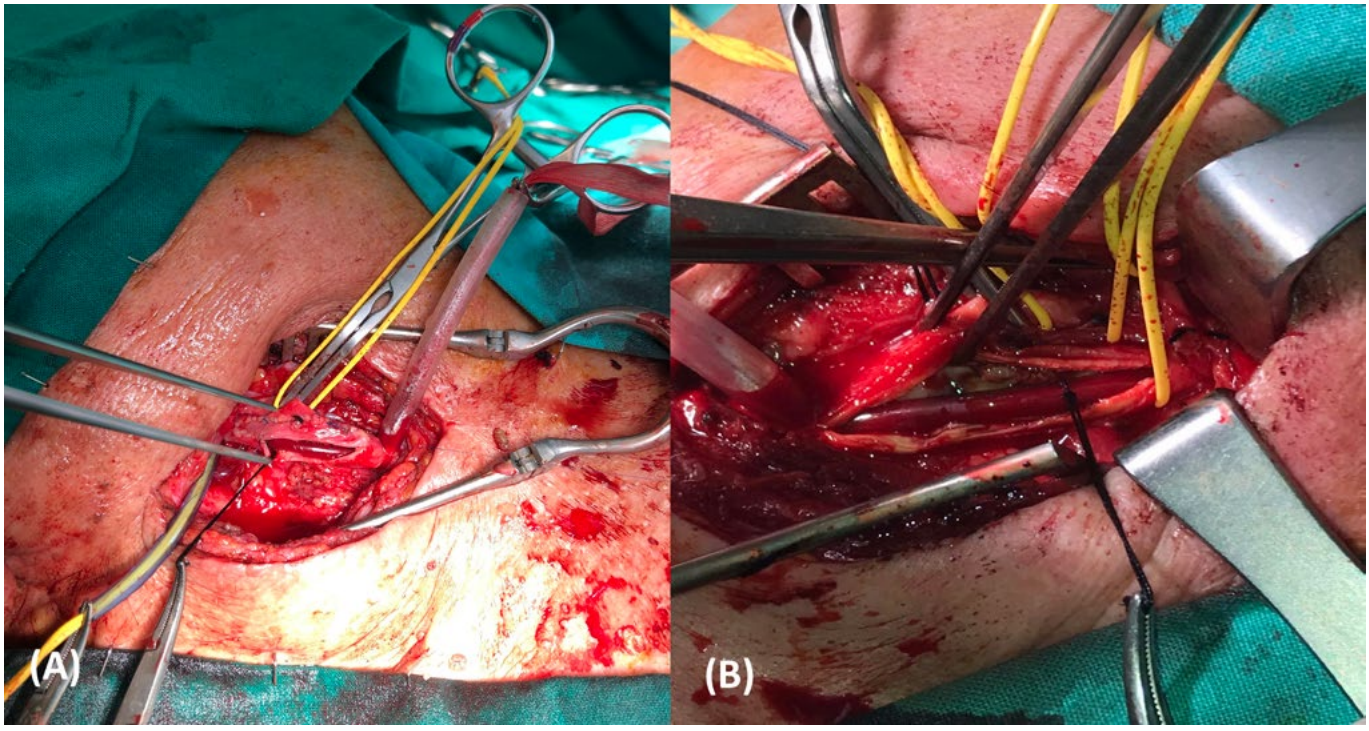

Fig 1. (A and B) Intraoperative images showing a shunt placement during a carotid endarterectomy procedure in patients with contralateral internal carotid artery occlusion.

nosis (≥80%) were included. All patients were treated in our Vascular Unit, within a 5-year period (01/2020 - 12/2024). Main indication for CEA in our institution includes a symptomatic ICA stenosis >50% or an asymptomatic stenosis >60% with high-risk characteristics (such as ultrasonographic characteristics of higher risk or contralateral occlusion). Ultrasonographic plaque characteristics suggestive of higher risk include intraplaque hemorrhage, ulcerative plaque or high echolucency, according to recent guidelines.[1]

All basic characteristics of patients were recorded including gender, mean age, main comorbidities and antithrombotic treatment. Technical characteristics, early and late outcomes were also evaluated. Technical characteristics included the type of patch, the type of CEA (traditional or eversion) and the use of shunt. Early outcomes included 30-day death, transient ischemic attack (TIA), stroke, bleeding/hematoma, cranial nerve injury and symptomatic myocardial infarction (MI) rates. Late outcomes included death and TIA/stroke rates within the follow-up period. Additionally, all outcomes were compared between asymptomatic patients <80 years old and ≥80 years old. Bleeding/hematoma was defined as any bleeding or hematoma that required blood transfusion or re-operation. Symptomatic MI was defined as any ischemic symptoms combined with alteration on electrocardiogram (ECG) or serum troponin elevation. Cranial nerve injury included any hoarseness, swallowing difficulty, tongue deviation or other symptom attributed to injury of major cranial nerves.

All patients were treated according to the Declaration of Helsinki. No special approval was required by the Institutional Review Board of our institution as this was a retrospective study.

Table 1. Basic characteristics of all patients

Male gender	35 (74%)
Mean age	75±10
Symptomatic patients	20 (42%)
Arterial Hypertension	35 (74%)
Diabetes Mellitus	10 (21%)
Coronary artery disease	16 (33%)
Dyslipidemia	39 (83%)
Chronic obstructive pulmonary disease	7 (15%)
Smoking history	35 (75%)
Antithrombotic medication	
ASA	23 (49%)
Clopidogrel	18 (38%)
Dual antiplatelet	6 (13%)
Technical characteristics	
Synthetic patch	10 (21%)
Bovine patch	30 (64%)
Venous patch	6 (13%)
Eversion endarterectomy	1 (2%)
Shunt placement	32 (68%)

Statistical analysis was conducted using the StatsDirect Statistical software (Version 2.8.0, StatsDirect Ltd, Cambridge, UK). Quantitative variables are presented with mean values and qualitative variables with absolute numbers and rates. Values were compared between groups using the exact Fisher test. P values < 0.05 were considered of statistical significance.

RESULTS

In this study, a total of 47 patients (18 patients with contralateral ICA occlusion and 29 patients with contralateral ≥80%

ICA stenosis) were included. Overall, 74% of patients were of male gender and 34% of patients were ≥80 years old. Overall, 42% (n=20) of patients were symptomatic (n=9 with stroke and n=11 with TIA/amaurosis fugax).

The following comorbidities were recorded: Smoking history (75%), Arterial hypertension (74%), Dyslipidemia (83%), Diabetes mellitus (21%), Coronary artery disease (33%), Chronic obstructive pulmonary disease (15%). The following antithrombotic treatment was recorded: ASA (n=23, 49%), Clopidogrel (n=18, 38%), dual antiplatelet (n=6, 13%). Regarding the technical characteristics, a venous patch was used in 6 cases (13%), a bovine patch in 30 cases (64%), a polytetrafluoroethylene (PTFE) patch in 10 cases (21%), an eversion technique was performed in 1 case (2%) and a shunt was placed in 68% of cases **(Table 1, Fig. 1)**.

Thirty-day outcomes included 0% death rate, 0% stroke rate, 4% (n=2) TIA rate, 4% (n=2) bleeding/hematoma rate, 4% (n=2) cranial nerve injury rate and 4% (n=2) symptomatic MI rate. Regarding bleeding, one case was re-operated within the first postoperative day due to bleeding through a suture-line defect and one case was re-operated after 2 weeks due to a persistent neck hematoma under dual antiplatelets. All cranial nerve injuries were referring to hoarseness that subsided within the following 6 months **(Table 2)**.

Within a mean follow-up of 22 ± 4 months, no new ipsilateral cerebrovascular event occurred. Eight late deaths occurred during the follow-up that were not related to the carotid disease.

Out of the 27 asymptomatic patients, 18 patients were <80 years old and 9 patients were ≥80 years old. Early and late outcomes were not different between the two groups **(Table 3)**.

Table 2. Early and late outcomes among all patients.

30-day outcomes	
Death	0 (0%)
Transient Ischemic Attack	2 (4%)
Stroke	0 (0%)
Bleeding/major hematoma	2 (4%)
Cranial nerve injury	2 (4%)
Symptomatic Myocardial Infarction	2 (4%)
Late outcomes (mean follow-up 22±4 months)	
Death	8 (17%)
Ipsilateral cerebrovascular event	0 (0%)

DISCUSSION

In this study, we found that both early and late outcomes are satisfying among patients with contralateral ICA occlusion or contralateral severe ICA stenosis undergoing CEA. Furthermore, age over 80 years old did not affect outcomes among asymptomatic patients with severe contralateral ICA disease.

To date, many studies have underlined the higher stroke risk associated with a contralateral ICA occlusion among patients with an ICA stenosis. AbuRhama et al. have found that patients with 60% to 70% ICA stenosis and contralateral ICA occlusion have a higher incidence of ipsilateral stroke as well as any stroke.[6] Therefore, performing a prophylactic CEA in those patients may be justified. The recent European guidelines on carotid artery disease management have also included the contralateral ICA occlusion among the risk factors to recommend an intervention in an asymptomatic patient with carotid artery stenosis.[1]

The status of contralateral ICA and its effect on outcomes after CEA has also been studied in literature. Data from the ACAS (Asymptomatic Carotid Atherosclerosis Study) and the NASCET (North American Symptomatic Carotid Endarterectomy Trial) trials suggest that contralateral ICA occlusion is a predictor of poor outcomes after CEA.[4,5] However, there has been a controversy lately about whether this factor is associated with adverse outcomes post CEA. A recent meta-analysis has showed that contralateral ICA occlusion is associated with a higher risk for perioperative neurological complications although there is no significant effect on perioperative mortality and 5-year stroke-free survival.[7] This concurs with our findings as the 4% stroke/TIA rate in the present cohort is higher than the overall rate among cases with or without contralateral disease treated in our unit as published before.[8] Additionally, the present cohort showed very low early mortality and long-term ipsilateral stroke/TIA rates concurring with previous published data.[8] Finally, outcomes seem to be similar between CEA and CAS among patients with contralateral occlusion, even among asymptomatic patients.[9] However, another meta-analysis has shown that CEA in patients with contralateral carotid occlusion is associated with an increased risk of perioperative stroke,

Table 3. Comparison of characteristics between asymptomatic patients <80 years old and ≥80 years old.

Outcomes	< 80 years old (n=18)	≥80 years old (n=9)	P value
30-day outcomes			
Death	0	0	NS
Transient Ischemic Attack	0	0	NS
Stroke	0	0	NS
Bleeding/major hematoma	0	1	NS
Cranial nerve injury	1	1	NS
Symptomatic Myocardial Infarction	1	0	NS
Late outcomes (mean follow-up 22±4 months)			
Death	1	3	NS
Ipsilateral cerebrovascular event	0	0	NS

NS, not significant.

death or TIA, while CAS in the presence of a contralateral occlusion is associated with an increased risk of periprocedural death but not stroke or TIA.[10]

Regarding the procedure, it has been found that patients with significant contralateral disease, namely occlusion or >70% stenosis, are correlated with a poor collateral perfusion pressure and require shunt placement during CEA.[11] The presence of cross-filling also plays an important role for the requirement of shunt in such patients. In this cohort, almost 70% of patients underwent a shunt placement concurring with previous series where over half of the cases with severe contralateral ICA disease needed a shunt.[7,12] Other factors associated with adverse neurological events among such patients with contralateral disease include female gender, preoperative symptoms, advanced age and severe renal insufficiency.[13-15]

In this study, preoperative symptoms did not affect outcomes after CEA. Furthermore, age >80 years old did not increase the risk for perioperative complications among asymptomatic patients with severe contralateral disease. This concurs with larger studies where symptomatic and asymptomatic patients with contralateral carotid occlusion had similar perioperative outcomes after CEA.[16] Additionally, Qumsiyeh et al. have compared outcomes of CEA between patients below and over 80 years of age.[17] Both age groups showed similar stroke rates after surgery and age >80 years old did not affect outcomes as well. Similarly, Ballotta et al. evaluated outcomes of octagenarians with contralateral disease undergoing CEA and found a very low neurological complications rate concurring with our findings.[18] Another large study showed that perioperative outcomes in asymptomatic patients aged ≥80 years with a contralateral ICA occlusion were similar to those in patients aged <80 years, validating the rationale to consider surgery in eligible older asymptomatic patients at elevated stroke risk.[19]

There are certain limitations in this study. First, this is a retrospective study, and there may be certain risks of bias considering patients' selection, shunt selection as well as the medical treatment. Second, the number of patients compared in each group was small and this could affect the level of significance for the analysis. Third, the small number of patients precluded the conduct of any multivariate analysis in order to evaluate potential predictors.

In conclusion, early and late outcomes after CEA are acceptable among patients with a severe contralateral ICA disease. Asymptomatic patients over 80 years old with a severe contralateral ICA disease seem to have similar outcomes compared to younger patients. Therefore, surgery should be considered for eligible older patients within this high-risk subgroup.

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DEBATE: PROS VS CONS

What Is the Best Option for Non-Infected Proximal Failure After EVAR? Endovascular Repair

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Abstract:

Proximal failure after endovascular aneurysm repair (EVAR) remains a major challenge in contemporary aortic surgery. Management requires a meticulous assessment of multiple factors, including patient anatomy, disease progression, aneurysm sac behavior, and endograft characteristics. Many patients facing failed EVAR tend to be older, frail, and often have serious cardiovascular issues, which complicates the decision-making process for clinicians. Open surgical conversion (OSC) is technically challenging due to the need for extensive surgical preparation and the frequent necessity for suprarenal or supraceliac aortic clamping. Consequently, perioperative morbidity and mortality remain considerable, particularly in urgent and emergent settings. Conversely, advanced endovascular techniques like fenestrated, branched, chimney, and physician-modified endografts have shown promising results, offering a lower physiological burden compared to OSC. Although there are ongoing discussions about long-term durability, current literature and our own institutional experience indicate that mid-term outcomes are encouraging. At our institution, we treated 38 patients with advanced endovascular methods for proximal failed EVAR. We achieved a 30-day survival rate of 94.7%, with target-vessel patency at 100% and an 88.1% rate of freedom from reintervention during a median follow-up of 20 months. Importantly, we did not observe any aortic-related deaths or recurrent type Ia endoleaks. These results advocate for an endovascular-first approach for most patients dealing with proximal failed EVAR, particularly through the use of custom-made fenestrated and branched devices. High-volume centers should be able to provide patients with the choice between the two strategies, after a thorough optimization process of patient physiological status, aortic anatomical criteria, surgical risk, patient disposition for long-term postoperative follow-up, and life prognosis.

Endovascular abdominal aortic aneurysm repair (EVAR) is the recommended treatment for infrarenal abdominal aortic aneurysms (AAA) in patients with adequate anatomy, according to the recent European Society of Vascular Surgery (ESVS) Guidelines, due to the lower early morbidity and mortality compared to open surgical repair (OSR).[1,2] However, reinterventions still question the durability of the repair and signify future proximal sealing loss.[3,4] Proximal failed EVAR encompasses a wide range of mechanisms accounting from sealing failure and leading to aneurysm sac pressurization and expansion, with proximal seal loss being the most common and most challenging to treat.[3-5] As EVAR rates continue to rise compared to OSR, so does the incidence of failed EVAR due to endoleak type Ia.[5] Proximal disease progression, EVAR outside instruction for use (IFU), inadequate individualized long-term postoperative surveillance, persistent type II endoleaks with sac expansion and the use of older generation endografts with device fatigue are some of the main reasons behind the increasing rates of proximal failed EVAR.[5-7]

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Treatment of proximal failure after EVAR requires meticulous assessment of the underlying etiology, as it rarely represents a single technical event, rather complex dynamic interactions incorporating patient anatomy, disease progression, sac behaviour and endograft performance.[5,7,8]

Patients presenting with failed EVAR frequently represent a highly comorbid vascular population characterized by advanced age, extensive atherosclerotic burden, and substantial cardiopulmonary risk, even more compared to their initial status of primary repair.[9] Male sex, history of smoking, coronary artery disease, chronic kidney disease, chronic obstructive pulmonary disorder and peripheral arterial disease all represent comorbidities consistently represented among patients with proximal failed EVAR.[4] Frailty and diminished physiological reserve, which often guide these patients towards primary AAA repair via endovascular means, is exacerbated during failed EVAR, ultimately affecting treatment selection and postoperative outcomes.[5] The often diminished physiological status of these patients is aggravated during urgent and emergent repairs.

On a technical note, OSR of failed EVAR remains one of the most challenging and technically demanding procedures in contemporary vascular surgery.[3] Suprarenal fixation struts, prior endograft incorporation as well as the need for extensive aortic and visceral dissection often required for proximal aortic clamping, substantially increase procedural complexity compared to primary OSR.[10] The effect of suprarenal or even supraceliac aortic cross clamping is detrimental for patient physiology and is frequently necessitated, while being asso-

ciated with significant risk for mesenteric and renal ischemia, cardiac complications and major blood loss.[9] The mismatch between procedural complexity and patients' physiological reserve is obvious in the high morbidity and mortality rates depicted in open surgical conversion of failed EVAR.[5,9] These outcomes are even more aggravated in urgent/emergent cases with aortic ruptures, presence of symptoms.[5] Even with the implementation of less aggressive strategies, including "semi-conversion" techniques with partial graft explantation, aiming towards the reduction of ischemia duration and operative trauma, as well as the advances in perioperative care and aortic reconstruction techniques, OSR of failed EVAR carries significant risk of perioperative morbidity and mortality for this quite frail patient cohort.[5] In any case an OSR approach requires the admission of the patient to Intensive Care Unit (CU) for at least the day of surgery, which carries a significant risk of infection if his/her stay needs to be prolonged. Admittingly, the perioperative management, from an anaesthesiologist's and intensivist's standpoint, and the postoperative care of patients treated via open surgical repair, even for standard infrarenal AAA, is significantly more meticulous and tiresome compared to patients undergoing endovascular repair. The extensive implementation of endovascular strategies, both for standard and complex endovascular repair, has reduced the experience of all associated healthcare personnel with patients treated via open surgery ('de-skilling' phenomenon), inadvertently affecting outcomes.[11]

The treatment of proximal failed EVAR has shifted towards the implementation of complex endovascular reconstruction, focusing on, but not contained to, more proximal sealing zones alongside the incorporation of target vessels via fenestrated (FEVAR) or branched (BEVAR) endografts.[7,12] Elective repair of failed EVAR is mainly managed through patient-specific custom-made devices (CMD), offering a wide range of solutions via fenestrations, branches, either in an inner or outer configuration, while more recently the semi-branch and bidirectional branch technologies have been successfully applied in anatomically challenging cases.[13,14] Urgent and emergent repair is rendered feasible through "off-the-shelf" multibranched endografts, physician-modified endografts (PMEG) or the use of parallel endografts (Chimneys). The immediate availability of "off-the-shelf" endografts is counterbalanced via their extensive proximal coverage of thoracic aorta, increasing the risk of spinal cord ischemia.

F/BEVAR offers less invasive repair, avoiding laparotomy and aortic cross clamping, while preserving end-organ perfusion. Several technical challenges arise, including access vessels, aortic angulation, presence of suprarenal struts in front of target vessel orifices, endograft alignment through the previous endograft, but also the usually short existing distance between lowest renal artery and previous endograft bifurcation.[5,7] F/BEVAR following proximal failed EVAR requires standardized preoperative imaging protocols in dedicated software, expertise in complex aortic endovascular repair and optimized patient management for ideal outcomes. Perioperative outcomes are most consistent in high-volume, specialized centres, including technical success, survival and reintervention rates.[15] Esposito et al. provide data on lower perioperative complications in endovascular repair of failed EVAR when compared to OSR, especially in elderly or frail patients, deemed unfit or high-risk for OSR.[5] Contemporary series have demonstrated excellent perioperative results, with very high technical success and comparable morbidity and mortality to primary elective F/BEVAR while when compared to OSR, the major systemic complications remained relatively low (15.7% vs. 21.3%).[5] Contemporary data on urgent and emergent cases treated via PMEGs demonstrated technical success rates consistently exceeding 90%, with acceptable target-vessel durability and sac stability during mid-term surveillance, supporting their role when CMDs are either unavailable or time-prohibitive.[12] Similarly, "off-the-shelf" branched endografts have shown excellent perioperative outcomes, with high-technical success rates, albeit being associated with higher mortality rates, especially in symptomatic patients.[13] The use of parallel/Chimney (ChEVAR) endovascular repair in urgent and emergent failed EVAR cases is also feasible.[16] In the Parallel Endografting And Chimney Endovascular (PEACE) registry, 18% of patients were treated with ChEVAR for either emergent or ruptured failed EVAR. Perioperative technical success (90.6%) and mortality (17.7%) appear to be acceptable for emergent and urgent cases, while low mid-term survival rates (45 ± 6%) are expected given the profile of treated patients.[16] The increased risk for perioperative complications related to OSR of failed EVAR commonly guides treatment towards endovascular strategies, rather than long-term durability alone.[5,7] Nevertheless, sac dynamics and regression is crucial for long-term durable outcomes. Sac stabilization or even regression should be the aim of complex endovascular salvage of failed EVAR, as it has been associated with lower future reintervention rates and adverse aortic events.[11] Sac dynamics are of particular importance, as data suggests that sac stabilization or regression is associated with better overall long-term outcomes.[12] Sac expansion is indicative of accumulating reintervention burden, associated with poor outcomes.[15]

While long-term data is missing, mid-term outcomes of F/BEVAR for failed EVAR could be considered acceptable, taking into account patients' frailty, physiological reserve and ultimately, prognosis. Mid-term survival following endovascular salvage remains satisfactory but inferior to open conversion in several comparative analyses.[5,17] Esposito et al. demonstrated an overall survival of 81.6% at approximately 18 months after F/BEVAR for failed EVAR.[5] Importantly, this reduced survival likely reflects baseline patient frailty and comorbidity burden rather than procedural inadequacy alone.[5,19] However, reinterventions still are the Achille's heel for durable, long-term outcomes. Reintervention remains the principal limitation of secondary endovascular repair. The same meta-analysis demonstrated a pooled mid-term reintervention rate of 26% after F/BEVAR, significantly higher than after elective open conversion (4.5%).[5] Reinterventions following F/BEVAR for failed EVAR are most commonly related to target vessel instability.[17,18] The European Multicentric Experience with Fenestrated-Branched ENDOvascular Stent-grafting after Previous FAILed Infrarenal Aortic Repair (EU-FBENDO-FAIL) registry,

depicts that F/BEVAR is feasible in both failed EVAR and failed OSR, with excellent primary patency rates even in this complex patient cohort.[20] Hostalrich et al. in their recent propensity score matching analysis showed comparable midterm survival between patients treated with OSR (88 ± 4%) and FEVAR (94 ± 3%) for failed EVAR.[21] However, comparison between the OSR and F/BEVAR for failed EVAR should be done carefully, as patients often selected for OSR are more fit, with less physiological burden.[2]

In our department, during an 8-year time period (2018-2026), a total of 38 patients (all male, median age 78 years old) have been treated for failed EVAR, applying a combination of F/BEVAR, as well as the parallel graft technique. Twenty-five (65.8%) of patients were treated electively, while 13 patients (34.2%) were treated urgently, with four ruptures. A total of 24 branched (63.1%), five fenestrated (13.1%) and nine chimney graft (23.6%) repairs were executed, based on urgency of repair and aortic anatomy. Eight patients (21%) were treated with an "off-the-shelf" branched endograft, requiring extensive proximal aortic coverage. One case of spinal cord ischemia was observed, in a patient treated with an "off-the-shelf" branched endograft for a rupture. The 30-day survival and major adverse events rates were 94.7% and 18.4%, respectively. The target-vessel patency was 100%. Only one reintervention related to vascular access was required. During a median follow-up of 20 months (Q1: 1, Q3: 80), the estimated survival was 72.5% (SE: 9%), without aortic-related deaths and the estimated freedom from reintervention was 88.1% (SE: 8%). No type Ia endoleak was recorded while two type IIIc endoleaks were managed with relining. Our accumulative experience and patient outcomes have led us towards an endovascular first approach for failed EVAR cases, implementing the use of CMDs, while confining the use of "off-the-shelf" branched endografts and the chimney graft technique only in urgent and emergent cases.

Endovascular and open surgical repair of failed EVAR should not be considered as counterparts but rather as both equally important weapons in modern vascular surgery. High-volume centres should be able to provide patients with the choice between the two strategies, after a thorough optimization process of patient physiological status, aortic anatomical criteria, surgical risk, patient disposition for long-term postoperative follow-up, and life prognosis.

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DEBATE: PROS VS CONS

What Is the Best Option for Non-Infected Proximal Failure After EVAR? Open Conversion

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Abstract:

Endovascular aneurysm repair (EVAR) has changed the treatment of abdominal aortic aneurysms by reducing early perioperative morbidity and mortality compared with open repair. Its long-term durability, however, depends on a stable proximal seal, secure fixation, and careful follow-up. Non-infected proximal failure after EVAR - type Ia endoleak, migration, neck dilatation, graft malapposition, or sac expansion - is not a minor imaging finding. It indicates failure of the central purpose of EVAR: exclusion of the aneurysm sac from systemic pressure. Secondary endovascular treatment with cuffs, endoanchors, parallel grafts, or fenestrated/branched repair is valuable in selected patients, especially those at prohibitive operative risk. However, in physiologically fit patients with complex, recurrent, or anatomically unsuitable proximal failure, open conversion remains the most definitive option. The essential point is timing. Elective open conversion has acceptable results in experienced centers, whereas delayed conversion after rupture or repeated failed endovascular attempts carries substantially higher risk. Open conversion should therefore not be reserved only as a final bailout procedure but considered early when it offers the best chance of durable repair.

EVAR was introduced as a less invasive alternative to open abdominal aortic aneurysm repair. Its early advantages are well known: lower perioperative mortality and faster recovery in appropriately selected patients.[1,2] However, EVAR does not remove the aneurysm. It excludes it. The success of the operation, therefore, depends on something that may change with time: the relationship between the endograft and the native aortic neck.

Non-infected proximal failure is one of the most important late problems after EVAR. It may present as type Ia endoleak, proximal migration, neck enlargement, graft malapposition, or sac expansion. Forbes et al. showed early on that most late open conversions were performed for persistent aneurysm perfusion, highlighting that the real problem is failure of sac exclusion.[3] A type Ia endoleak is very different from a low-flow type II endoleak. It reflects direct systemic pressurization of the aneurysm sac and is associated with continued expansion and rupture risk.[4,5] Hostile neck anatomy and progressive neck dilatation are recognized mechanisms that lead to loss of seal, migration, and late failure.[6,7] The 2024 European Society for Vascular Surgery guidelines recognize a compromised proximal seal after EVAR as a clinically important problem and state that elective open conversion may be considered in selected patients.[8]

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In a debate on failed EVAR, technical feasibility is not enough. A further endovascular maneuver may look attractive, particularly because it avoids laparotomy and aortic clamping. The more important question is whether it offers a durable solution for the individual patient. For a fit patient with non-infected proximal failure, open conversion corrects the failed repair directly and restores aneurysm exclusion through surgical reconstruction.

Endovascular options for proximal failure include balloon molding, proximal cuffs, endoanchors, chimney or parallel graft techniques, and fenestrated or branched repair. These methods are important and, in many patients, entirely appropriate. Frail patients, those with hostile abdomens, or patients with prohibitive cardiopulmonary risk may benefit most from an endovascular strategy. Fenestrated and branched repair has also expanded the possibilities for treating failed infrarenal EVAR by moving the seal zone into the pararenal or visceral aorta.[9]

Nevertheless, the ability to perform another endovascular procedure should not be confused with the ability to provide a durable repair. Secondary endovascular repair may convert an infrarenal problem into a paravisceral reconstruction requiring fenestrations, branches, bridging stents, and continued surveillance. Endoanchors may improve fixation and sealing in selected cases, but they cannot reliably compensate for severe neck degeneration, major migration, or absence of an adequate landing zone.[10] Parallel graft strategies may be useful in urgent or constrained anatomy, but the risk of gutters, recurrent endoleak, and reintervention remains a concern. Contemporary meta-analytic data suggest that fenestrated and branched endovascular repair can provide an effective rescue option for proximal endograft failure after EVAR, with high

technical success and acceptable early mortality; however, midterm reintervention rates remain substantial, reinforcing the need to judge endovascular feasibility against expected durability in each patient.[11]

There is also a practical surgical issue. Repeated endovascular attempts may make later open conversion more difficult. They may extend the metal framework proximally, involve the renal or visceral arteries, increase the complexity of explantation, and remove favorable clamp sites. For this reason, an "endovascular until impossible" strategy may turn a controlled elective conversion into a more complex and hazardous operation.

The appeal of open conversion is simple: it addresses the failed seal directly. Instead of searching for another compromised landing zone, the surgeon re-establishes control of the aorta and reconstructs the aneurysmal segment with a sutured graft. The increasing use of open conversion in contemporary practice should not be seen as a failure of surgery, but as a consequence of the cumulative burden of late EVAR complications. Mohapatra et al. reported an increasing use of open conversion for late EVAR complications and concluded that elective conversion for endoleak is reasonably safe and durable, including when graft-preserving strategies are used.[12]

The usual objection is operative risk. This concern is real, but timing changes the entire discussion. In the systematic review by Kouvelos et al., which included 641 patients undergoing late open conversion after EVAR, the overall 30-day mortality was 9.1%. However, mortality was 3.2% after elective conversion and 29.2% after nonelective conversion.[13] A later systematic review and meta-analysis showed a similar message, with approximately tenfold higher 30-day mortality for urgent compared with elective late open conversion, while elective conversion approached the mortality of primary elective open abdominal aortic aneurysm repair.[14]

Other data support the same principle. Scali et al. showed that elective open conversion for type Ia endoleak was not associated with increased morbidity or mortality compared with primary open juxtarenal aneurysm repair in selected patients.[15] More recently, Kahlberg et al. compared elective late open conversion after EVAR with primary open repair in a high-volume center. After propensity score matching, in-hospital mortality and 30-day reintervention rates were not significantly different between the two groups.[16] These studies matter because they challenge the reflex to reserve open conversion until all endovascular options have been exhausted.

The conclusion is not that every proximal failure after EVAR requires open surgery. Rather, the threshold for elective conversion should be lower when the patient is fit, the sac is expanding, the endograft has migrated, the neck has degenerated, or previous endovascular correction has failed. In such cases, another endovascular procedure may delay the definitive solution rather than provide it.

Open conversion is most compelling in patients with acceptable physiological reserve, reasonable life expectancy, and anatomy that makes durable endovascular salvage uncertain. This includes significant sac expansion with type Ia endoleak, major migration, progressive neck degeneration, recurrent proximal failure after prior reintervention, lack of an adequate landing zone, or anatomy unsuitable for a reliable fenestrated or branched repair.

Endovascular salvage remains preferable in many patients: the very elderly, the frail, those with severe cardiopulmonary disease, hostile abdomen, or limited life expectancy. The argument is not open conversion for all. The argument is an open conversion for the patient in whom a definitive surgical repair is safer in the long term than repeated attempts to extend a failing endovascular solution. A practical decision-making algorithm for managing non-infected proximal EVAR failure is shown in **Figure 1**.

Open conversion after EVAR should be planned as a complex aortic reconstruction. Preoperative computed tomography angiography must define the proximal extent of failure, renal and visceral anatomy, endograft configuration, fixation system, iliac anatomy, and absence of infection. The surgeon should be prepared for suprarenal or supraceliac control, renal protection, difficult iliac reconstruction, and partial or complete endograft explantation.

Importantly, open conversion does not always mean aggressive total explantation. In non-infected cases, partial preservation of incorporated components may reduce operative trauma, particularly when suprarenal fixation is firmly embedded. Recent multicentre data comparing partial and total stent-graft removal during late open conversion for non-infectious EVAR failure showed numerically lower 30-day mortality with partial conversion, similar long-term survival, and similar freedom from late complications.[17] No graft infection or thrombosis was reported in the partial-conversion group during follow-up. This supports a pragmatic approach: the objective is durable correction of failed aneurysm exclusion, not removal of every endograft component at all costs. Techniques such as neo-neck or graft-preserving reconstruction have been described to achieve durable repair while avoiding unnecessary injury during explantation.[18]

Non-infected proximal failure after EVAR is serious because it represents a loss of the central principle of aneurysm exclusion. Endovascular salvage remains valuable, particularly in frail or high-risk patients. However, in a physiologically fit patient with complex, recurrent, or anatomically unsuitable proximal failure, open conversion offers the most definitive and durable repair.

The decisive issue is timing. Elective open conversion in experienced centers can achieve acceptable outcomes and may approach those of primary complex open repair. By contrast, delayed conversion after rupture, instability, or repeated failed endovascular attempts carries substantially higher risk. Open conversion should therefore not be kept only as a last-resort bailout. In selected patients, early planned conversion may be the safest way to restore durable aneurysm exclusion before the situation becomes an emergency. For suitable patients with non-infected proximal EVAR failure, open conversion remains the benchmark against which secondary endovascular solutions should be judged.

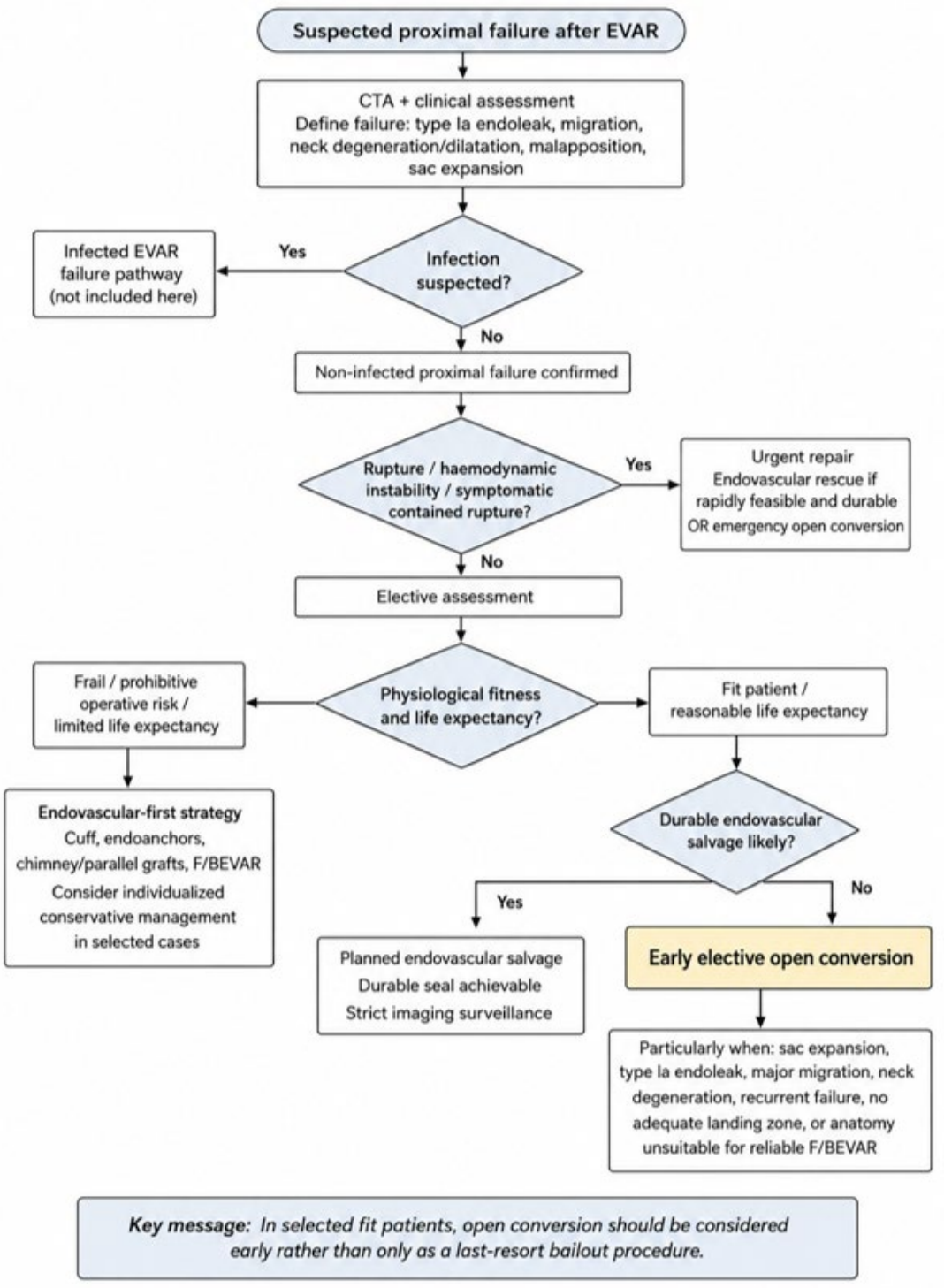

Figure 1. Treatment algorithm for non-infected proximal failure after EVAR.

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REVIEW

Current Management of Blunt Thoracic Aortic Injury: A Comparative Review of the ESVS and SVS guidelines

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Abstract:

Blunt thoracic aortic injury (BTAI) is a life-threatening condition, arising from high-energy non-penetrating trauma and it is associated with significant fatality rates. This clinical entity demands prompt evaluation and rapid and carefully-selected therapeutic approach. Thoracic endovascular aortic repair (TEVAR) represents a significant milestone in the management of this condition and has largely replaced open surgery, offering various benefits to the patients. This review aims to compare the most recent guidelines from the Society of Vascular Surgery (SVS) and the European Society of Vascular Surgery (ESVS) regarding the management of BTAI. It presents the similarities and differences, while special attention is given to areas of controversy and particularly Grade II injuries treatment.

Keywords: TEVAR, BTAI, aortic injury, expert opinion

INTRODUCTION

Blunt thoracic aortic injury (BTAI) is a rare condition associated with high mortality rates. Although its incidence is less than 1%, it is the second most common cause of trauma related mortality following head injury.[1] It has been estimated that 80% of BTAI patients die before they arrive at the hospital and of those who arrive, the majority have multiple injuries leading to an in-hospital mortality rate around 46%.[2] In the last two decades, with the advent of endografts, management of high-grade BTAI has changed significantly as open surgical repair has largely been replaced by thoracic endovascular aortic repair (TEVAR).[3] In addition, amassing evidence support expectant management for low grade aneurysms with blood pressure and heart rate control.[1] Scientific societies including the Society of Vascular Surgery (SVS) and the European Society of Vascular Surgery (ESVS) have published guidelines for diagnosis and management of BTAI. While these guidelines share core principles, there are some areas that differ including the timing of intervention and the preferable therapeutic approach. The aim of this review is to evaluate and compare these guidelines, emphasize the similarities, the differences and the corresponding outcomes.[4,5]

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Natural history of BAI

Blunt aortic trauma is defined as a tear in the aorta which results come from a rapid deceleration injury that generates shear and stretch forces.[2] Clinical outcomes of blunt aortic trauma are largely determined by the anatomical location and the extent of injury. Trauma can affect any part of the aorta, but the majority of them affect the descending thoracic segment and specifically the isthmus. The abdominal or the ascending aorta are rarely involved although injuries at these segments may be devastating. In patients who survive and arrive at the hospital the progression of injury depends on the severity of aortic wall disruption and may vary significantly.[1,6] Injury grading is based on CT imaging and is a key determinant of suggested management. Interestingly, SVS and ESVS have issued management guidelines using slightly different classification systems. While SVS provides a four-grade injury classification guideline, ESVS classification lumps the lower severity (Grade 1 and 2) SVS classification into one grade (Grade 1) thus suggesting a corresponding three grade staging **(Table 1)**. While the major image characteristics are shared by both SVS and EVS classification systems, there are differences in the evaluation of imaging findings and the assigned injury grade creating some controversy with regards to the most appropriate management.

Management

ESVS and SVS guidelines have proposed evidence-based recommendations for imaging, grading and therapeutic approaches for BTAI, aiming to optimize outcomes in these patients. According to ESVS guidelines a CTA of the aorta is the most essential diagnostic procedure, while chest X-ray is considered unreliable. In patients with no rupture at the time of admission, the risk is increased in the first few hours after the

Table 1 Classification system of aortic injuries based on the ESVS and SVS guidelines

ESVS	SVS	Description
Grade 1	Grade I	Intimal tear
	Grade II	Intramural hematoma
Grade 2	Grade III	Pseudoaneurysm
Grade 3	Grade IV	Rapture

ESVS: European Society of Vascular surgery, SVS: Society of Vascular Surgery

incidence. As reported by studies, strict blood pressure control significantly reduces that risk. Accordingly, systolic blood pressure is indicated to be maintained 90-110 mmHg and the pulses <100/minute. Injuries involving intimal tears or intramural hematoma (ESVS Grade 1) are best managed with blood pressure control and repeated CTA, if there is no other injury. Occasionally, operative management (TEVAR) should be considered in patients with a concomitant brain injury requiring raising the mean arterial prerssure in order to improve cerebral perfusion, or when blood pressure control is difficult. For patients presenting with disruption of the external wall of the aorta or pseudoaneurysm (ESVS Grade 2) the indicated management is TEVAR. In cases where complete wall laceration and rupture occur (ESVS Grade 3) immediate intervention is required. Open surgical repair is currently considered only in limited cases and only when the anatomical configuration is suboptimal for stent graft placement. In these cases, active distal aortic perfusion is advised in order to reduce the risk of paraplegia.

As outlined in SVS guidelines, CTA is the preferred modality for the prompt evaluation of BTAI. In patients with intimal tears (Grade I) injuries, expectant management with serial imaging and blood pressure control is recommended. In Grade II lesions, TEVAR is recommended regardless of the patient condition and co-existing injuries. For patients with Grade III and Grade IV lesions TEVAR is the procedure of choice. Open surgical repair is applicable only in cases with specific anatomic indications. Left subclavian artery revascularization is suggested only in specific cases, considering the experience of the surgeon and the patient's condition.

In the past two decades, TEVAR has become the preferred repair solution (if available) compared to open surgical repair. The procedure can be performed expeditiously with minimal blood loss and anesthesia requirements. There is ongoing debate regarding the optimal timing of TEVAR in stable patients, particularly those with multiple injuries. Indications for urgent repair (<24h) include imaging findings that reflect more significant injury such as bleeding in mediastinum, left hemothorax or compression effects resulting in narrowing of aorta. On the contrary, a number of studies have suggested that delayed intervention (>24h) is associated with a survival benefit in patients who don't have the previously mentioned high-risk features. However, the ideal time of intervention still remains uncertain. If urgent TEVAR is required, graft oversizing should take into consideration the degree of intravascular volume depletion and the potential underestimation of the aortic diameter above and below the injury. Oversizing in well resuscitated patients should not exceed 5-15% but in volume depleted patients graft oversizing by 20-30% is not unreasonable and should be considered. Key factors that should be considered are the anatomical features and the technical specifications of the selected endograft.

The use of systemic heparinization during TEVAR in patients with BTAI and concomitant injuries is also debated. It is suggested but at a lower dose than in elective TEVAR procedures. The decision to use heparin during TEVAR should be tailored on a case-by-case basis weighing the risk of bleeding, thromboembolism and the type/severity of any associated traumatic injury particularly those involving solid organs and the brain.

Every effort to preserve flow to the left subclavian artery should be made but if coverage of its origin during TEVAR is deemed necessary for appropriate seal, delayed revascularization may be appropriate for those with ischemic symptoms.

Follow-up requires imaging with Computed Tomographic Angiography (CTA) or Magnetic Resonance Angiography (MRA) at one month and one year after TEVAR for BTAI, with continued surveillance for at least five years. The optimal follow-up protocol after TEVAR for BTAI remains undefined and experts of the field express concerns due to cumulative radiation, contrast exposure and late endograft complications. Opinions vary significantly, with some suggesting imaging every 2-5 years after an initial complication-free period of 12-36 months. Others argue that follow-up should be the same as for those who underwent elective TEVAR. There is partial agreement, that combination of chest X-ray and MRA may be preferable to CTA, considering the metallic artifacts introduced by the endograft.[4] CTA or MRA is advised at one year and at five years following open surgical repair. Patients who did not receive any intervention CTA or MRA at one month and then yearly until remodeling is acceptable.[5] Treatment recommendations for each aortic injury grade based on the ESVS and SVS guidelines are summarized in **Table 2**.

EXPERTS' OPINION

Traumatic blunt thoracic aortic injury (BTAI) is a complex clinical entity that presents therapeutic challenges and requires careful management. Both SVS and ESVS guidelines offer a comprehensive approach to the diagnostic work-up, treatment pathways and follow-up. While there is concurrency that Grade I lesions should be managed expectantly and Grade III and Grade IV operatively, discrepancies especially in Grade II management underline the need for further research to clarify the optimal approach.[4,5] Grade II injuries are defined

Table 2 Treatment recommendations for each aortic injury grade

Topic	ESVS	SVS
Grading System	Grade I-IV	Grade I-IV
Grade I management	Non-operative	Non-operative
Grade II management	Conservative (TEVAR only in selected cases	TEVAR
Grade III management	TEVAR	TEVAR
Grade IV management	TEVAR	TEVAR
Timing of intervention	Delayed (if patient is stable)	Urgent or delayed

ESVS: European Society of Vascular Surgery, SVS: Society of Vascular Surgery, TEVAR: Thoracic Endovascular Aortic Repair

as intramural hematoma without disruption of external wall. As various studies reveal, this type of injury may remain stable or even regress with non-operative management. In some cases, they may progress to Grade III (pseudoaneurysm).[7] Several studies illustrate similar mortality rates between operative and non-operative management, especially in patients who are hemodynamically stable. TEVAR is indicated when the following high risk imaging features are present: posterior mediastinal hematoma >10 mm, mediastinal hematoma causing mass effect, pseudo-coarctation of the aorta, large left hemothorax, involvement ascending aorta, aortic arch, or great vessel and aortic arch hematoma.[5]

Although TEVAR is a minimally invasive procedure it still carries immediate, perioperative and long-term risks. The short-term risks of this procedure include endoleaks, misplacement of the graft, access site complications (e.g. hematoma, pseudoaneurysm), occlusion of the left subclavian or left common carotid arteries, stroke, spinal cord ischemia, acute kidney injury due to the use of contrast and even thrombosis. Complications can also be seen during the follow-up several years after the procedure including migration of the graft, incomplete aortic remodeling and endoleak.[5,7]

The process of aortic remodeling after TEVAR is a significant factor of long-term durability of the results. The remodeling is defined as enlargement of the aorta especially the ascending and the mid-aortic arch. Furthermore, the diameter of the proximal and distal landing zone is usually extended remarkably.[8] The remodeling is a dynamic process influenced by anatomical and physiological factors. The major risk factor for remodeling is elevated systemic blood pressure and especially in patients with pre-existing chronic hypertension, which increases sear stress across the stent segment, enhances continued aortic enlargement.[9] The initial period after TEVAR, especially the first 4-6 weeks aggressive blood pressure control is not indicated, and moderate hypertension is well tolerated. After that, tight control of blood pressure (<120 mmHg) and heart rate (<80 bpm) are strongly recommended and associated with more favorable outcomes.[10] Additional parameters that are associated with remodeling are a larger initial aortic diameter, the excessive oversizing of the graft and patient youth.[9]

CONCLUSION

Blunt thoracic aortic injury represents a challenging and potentially fatal condition that demands prompt management strategies. TEVAR undeniably represents a major evolution in the therapeutic approach offering several advantages and superior outcomes. Despite the progress in imaging and operative methods, there is uncertainty and controversy particularly in the management of Grade II injuries between SVS and ESVS guidelines.[4,5] An increasing number of studies depict that conservative management of these cases with close follow-up imaging is a favorable approach. However, further studies and more robust prospective data are essential in order to better define the indications for non-operative management and TEVAR in patients with lower grade BTAI. Future guidelines should incorporate this data and provide a more structured framework for the approach to these injuries. Until then and considering the complexity of BTAI treatment, decisions should be made case-by-case and taking into account patients' anatomical and physiological factors. A multidisciplinary approach and close cooperation between trauma centers and vascular societies is crucial for optimizing the treatment strategies and improving the outcomes.[11]

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REVIEW

Combined Transcatheter Aortic Valve Implantation and Endovascular Aortic Repair: A Review

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Abstract:

Background: The coexistence of severe aortic valve stenosis and aortic aneurysmal disease presents a complex therapeutic challenge, particularly in elderly patients at high surgical risk. Advances in minimally invasive techniques have enabled combined transcatheter aortic valve implantation (TAVI) and endovascular aortic repair (EVAR / TEVAR) as an alternative to open surgery.

Methods: A comprehensive review of the literature was performed to identify published studies reporting outcomes of combined TAVI and endovascular aortic repair, either as a simultaneous or staged strategy. Study characteristics, procedural details, and reported outcomes were analyzed descriptively.

Results: The available evidence consists predominantly of case reports and small case series involving high-risk patients. Both simultaneous and staged approaches were reported, with TAVI most commonly performed prior to aortic repair. Procedural success rates were high, and short- to mid-term outcomes were generally favorable. Complications were infrequently reported in small studies, although larger series of isolated procedures indicate that relevant adverse events may occur.

Conclusion: Combined TAVI and endovascular aortic repair appear to be a feasible and minimally invasive treatment option for selected high-risk patients with concomitant aortic valve and aortic pathology. However, evidence remains limited, and optimal patient selection and procedural sequencing are yet to be defined. Larger studies and prospective registries are required to establish standardized treatment strategies.

Keywords: transcatheter aortic valve implantation, endovascular aortic repair, review

INTRODUCTION

The prevalence of concomitant aortic aneurysms involving the thoracic and abdominal aorta in patients with severe aortic valve stenosis has increased in the elderly population, and is reported in approximately 6% of patients undergoing transcatheter aortic valve implantation (TAVI).[1-3] Currently, there are no established recommendations regarding the optimal management of these patients, rendering their treatment particularly challenging.

Historically, management of patients requiring aortic valve replacement (AVR) in the presence of an aortic aneurysm has favored a staged approach, with simultaneous repair reserved for cases involving large or symptomatic aneurysms, primarily due to concerns regarding the increased risk of aneurysm rupture following open AVR.[4] In recent years, in an effort to mitigate perioperative mortality and the invasiveness associated with combined open procedures, a single-stage approach consisting of TAVI followed by thoracic or endovascular aortic aneurysm repair (TEVAR) has emerged as an attractive and less invasive alternative.[5-7] The aim of the present review is to summarize the available published cases of patients with concomitant aortic aneurysms undergoing TAVI, treated either in simultaneous or staged manner.

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METHODS

Design and registration

The present systematic review was designed and reported in accordance with the Preferred Reporting Items for Systematic reviews (PRISMA) statement **(Figure 1)**.

Eligibility criteria

Observational cohort studies (prospective or retrospective), and case series/report studies, published in English, and reporting data on TAVI and TEVAR or EVAR, were considered eligible. Studies which provided data on hybrid procedures (open aortic valve replacement +TEVAR or EVAR), or those with TAVI and open repair of thoracic and abdominal aortic aneurysm were excluded.

Search strategy

A thorough literature search was conducted in MEDLINE (via PubMed; 1966 to November 2025), EMBASE (via Ovid; 1980

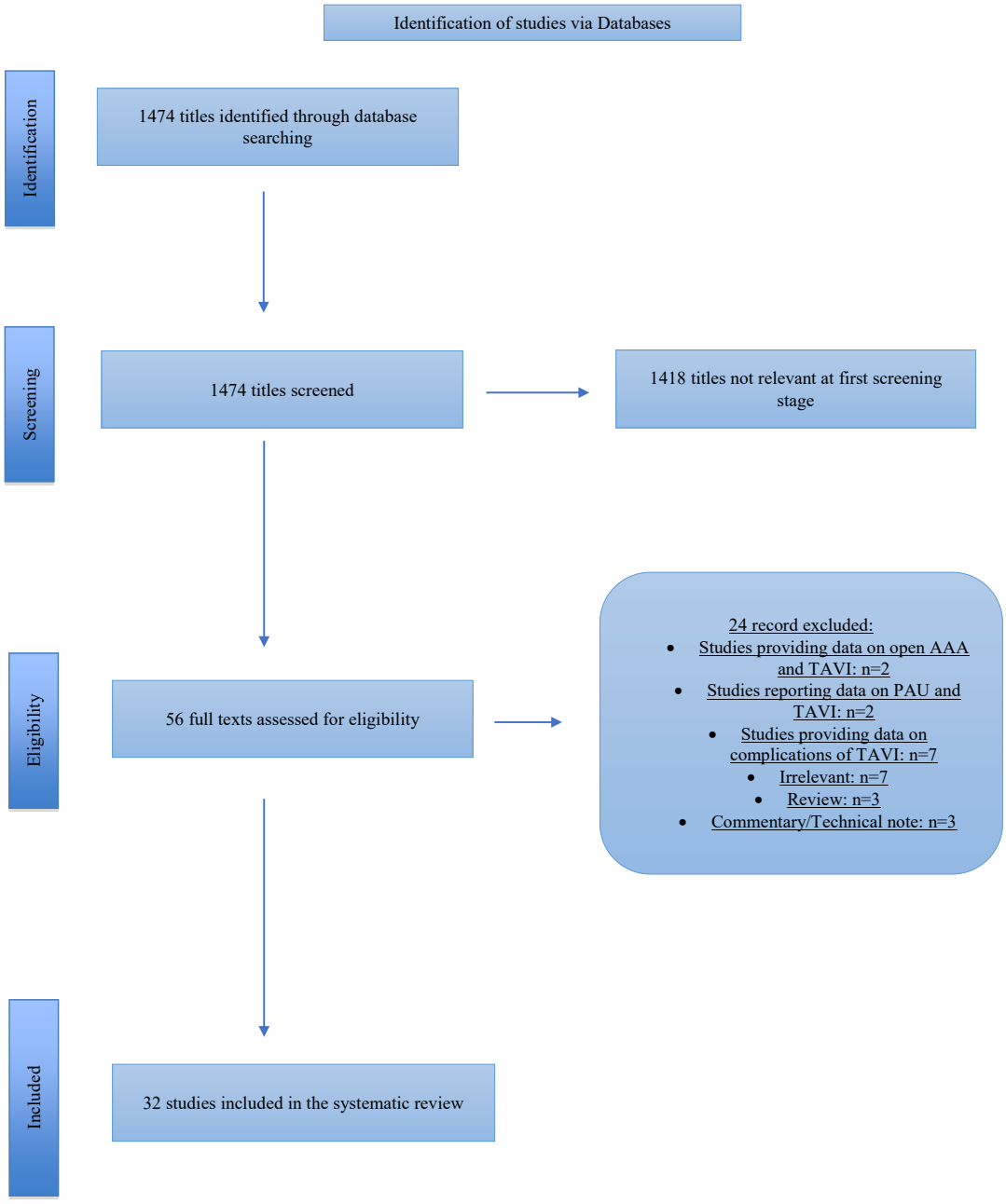

Figure 1: Study flow chart ("Preferred Reporting Items for Systematic reviews and Meta-Analysis" diagram)

to November 2025), the Cochrane Central Register of Controlled Trials (CENTRAL) (through November 2025), and Google Scholar (through November 2025). A snowball process of the reference lists from the eligible studies was performed after the retrieval of relevant reports from the databases searches. The following search items, including expanded Medical Subject Headings (MeSH) terms were used in various combination: ((transcatheter aortic valve implantation) OR (replacement)) AND (aneurysm) AND (endovascular aortic repair). **Figure 1** depicts the eligible studies available in the literature, including 22 studies[2, 5, 7-26] with TAVI and EVAR and 10 studies[5, 6, 27-34] with TAVI and TEVAR. Unfortunately, most of the included studies were case reports (n = 24),[3, 6, 8, 10-17, 22-24, 27, 28, 30-35] while only three studies[2, 5, 7] included more than 10 patients and six studies[9, 20, 21, 25, 26, 29] were small case series comprising six or fewer patients. One study reported outcomes on TAVI and TEVAR or EVAR.[5] Owing to the limited sample sizes and heterogeneity of the available data, no formal statistical analysis was performed in the present review.

Definitions

Major adverse events were defined as all-cause mortality, myocardial infarction, respiratory failure requiring prolonged (>24 hours from anticipated) mechanical ventilation or reintubation, renal function decline resulting in >50% reduction in baseline eGFR or new-onset dialysis, bowel ischemia requiring surgical resection or not resolving with medical therapy, major stroke, and paraplegia.[36]

All events that occurred intraoperatively or within 30 days

postoperatively were defined as early outcomes, whereas RTAD events occurring more than 30 days after surgery were defined as late outcomes.

Pathophysiological Considerations

Severe aortic valve stenosis and aortic aneurysmal disease frequently coexist in elderly patients, sharing common risk factors such as advanced age, hypertension, atherosclerosis, and degenerative changes of the aortic wall. The presence of severe aortic stenosis leads to chronic pressure overload of the left ventricle, resulting in reduced cardiac output and altered aortic hemodynamics. These changes may partially mask the true hemodynamic stress exerted on the aneurysmal aortic wall prior to valve intervention.[37, 38] Relief of aortic stenosis following aortic valve replacement, particularly after TAVI, results in an abrupt increase in cardiac output and systolic blood pressure. This sudden hemodynamic shift may increase wall shear stress and transmural pressure within the aneurysm, potentially predisposing to aneurysm expansion or rupture, especially in large or unstable aneurysms.[39, 40] This phenomenon has historically raised concerns regarding the safety of isolated valve replacement in patients with concomitant aortic aneurysms. Furthermore, manipulation of the aorta during valve procedures, including catheter and device passage through aneurysmal segments, may increase the risk of embolization, dissection, or rupture, particularly in extensively diseased or thrombus aorta.[41] These risks underscore the importance of meticulous preprocedural imaging and planning.

Conversely, endovascular aortic repair (EVAR or TEVAR) stabilizes the aneurysmal segment by excluding it from systemic arterial pressure, potentially reducing the risk associated with subsequent increases in cardiac output following valve intervention. For this reason, combined or staged endovascular and transcatheter strategies have been increasingly considered to mitigate these opposing pathophysiological forces.[42]

Understanding the complex hemodynamic interplay between aortic valve stenosis and aneurysmal disease is crucial for optimizing procedural sequencing and minimizing perioperative risk. This interplay forms the physiological basis for contemporary combined treatment strategies using TAVI and endovascular aortic repair.

RESULTS

A. Thoracic endovascular aneurysm repair and Transcatheter aortic valve implantation

In the present review, ten studies,[5, 6, 27-34] comprising eight case reports[6, 27, 28, 30-34] and two case series[5, 29] with a total of 18 patients reported outcomes following combined thoracic endovascular aortic repair (TEVAR) and transcatheter aortic valve implantation (TAVI) **(Table 1)**. The mean patient age was 80 years (range, 60-88 years). The mean maximum aortic diameter was 66 mm (range, 20-90 mm). Twelve patients underwent a single-stage procedure, whereas five patients were treated using a staged approach.

The Edwards Sapien transcatheter heart valve (Edwards Lifesciences, Irvine, CA, USA) was used in six studies[29-34] while in two studies[6, 27] was selected the Evolut Pro (Medtronic, Minneapolis, MN, USA). Regarding the type of thoracic aortic endografts used, the c-TAG device (W.L. Gore & Associates, Flagstaff, AZ, USA) was used in four patients,[6, 29, 30, 33] the Valiant Captivia device (Medtronic, Minneapolis, MN, USA) in two patients,[27, 31] and the Najuta thoracic stent graft (Kawasumi Laboratories, Inc., Tokyo, Japan) in two patients.[28, 32] In the remaining cases, the type of thoracic endograft was not specified.

Early outcomes

One patient died during the 30-day period who underwent a staged procedure with TEVAR and TAVI. An 84-year-old male who underwent EVAR first and TAVI after 86 days; the 2nd postoperative course was complicated by urinary sepsis causing final exitus. Major adverse events were reported in five patients including cardiac events (n=1), respiratory adverse events (n=3) and cerebrovascular adverse events (n=1).[5] Transient spinal cord injury appeared in one patient 48 hours postoperatively who underwent a simultaneous treatment. Magnetic resonance imaging of the thoracic spine revealed no evidence of spinal cord infarction or epidural haematoma. The patient fully recovered with vasopressor therapy.[6] The mean length of stay was 12 days (range, 4-21 days).

Late outcomes

The mean follow-up duration was 10 months (range, 1-25

Table 1: Baseline characteristics of the eligible studies (TAVI+TEVAR)

Author	Year of publication	Study Design	Number of Participants	Sex	Age	TAA size (mm)
Liu Y et al[27]	2025	CR	1	m	82	TBAD
Gallitto et al[5]	2024	CS	8	m/f	82	58
Ikeda et al[28]	2023	CR	1	m	79	60
Painchaud-Bouchard AS et al[34]	2022	CR	1	f	78	78
Hayakawa et al[32]	2021	CR	1	f	83	55
Kadoya et al[6]	2020	CR	1	m	87	58
De Backer et al[31]	2015	CR	1	f	78	70
Allen et al[29]	2015	CS	2	f	60	90
Komlo et al[33]	2014	CR	1	f	88	60
Ayhan et al[30]	2014	CR	1	m	83	20

CR: case report; **CS:** case series; **TBAD: type B aortic Dissection**

(Table 1 continue)

Table 1: Baseline characteristics of the eligible studies (TAVI+TEVAR)

Author	Type & size of cardiac valve (mm)	Type & size of Aortic endograft (mm)	Type of repair and number of patients (n)		Complications		LOS (days)	FU (month)
			Simultanous	2nd stage	Early	Late		
Liu Y et al[27]	29-mm EvolutPRO	Valiant Captivia 36x36x150	1		none	none	nr	3
Gallitto et al[5]	nr	nr	5	3	Death: n=1 MAE N=5	none	13	25
Ikeda et al[28]	nr	Najuta		1	nr	nr	nr	nr
Painchaud-Bouchard AS et al[34]	26-mm Sapien	Zenith	1		nr	nr	19	12
Hayakawa et al[32]	26-mm Sapien	Najuta	1		nr	nr	nr	nr
Kadoya et al[6]	23-mmEvolut PRO	c-TAG	1		paraplegia	nr	21	12
De Backer et al[31]	Sapien	Valiant		1	none	none	nr	6
Allen et al[29]	26-mm Sapien	c-TAG	1		none	none	4	12
Komlo et al[33]	26-mm Sapien	Gore c-TAG	1		none	none	8	14
Ayhan et al[30]	26 mm Sapien	Gore c-TAG	1		none	none	7	1

MAE: major adverse events; **nr:** no reported; **LOS:** length of stay; **FU:** follow-up

months). During this period, no complications were reported in any of the included studies.

B. Endovascular aortic aneurysm repair and Transcatheter aortic valve implantation

Twenty-two studies[2, 5, 7-26, 30] with an overall of 226 patients reported data based on TAVI and EVAR **(Table 2)**. The mean patient age was 79 years (range, 67-91 years). The mean maximum aortic diameter was 55 mm (range, 30-70 mm). Twelve patients underwent a single-stage procedure, whereas five patients were treated using a staged approach.

The Evolut Pro (Medtronic, Minneapolis, MN, USA) was selected in five studies,[8, 9, 15, 16, 18, 21, 26], the Edwards Sapien transcatheter heart valve (Edwards Lifesciences, Irvine, CA, USA)

Table 2: Baseline characteristics of the eligible studies (TAVI+EVAR)

Author	Year of publication	Study Design	Number of Participants	Sex	Age	AAA size (mm)
PU et al[2]	2025	RS	145	m/f	83	nr
Lu J et al[7]	2025	RS	11	m/f	84	70
Boljevic et al[8]	2025	CR	1	m	75	46
Sanoussi et al[10]	2025	CR	1	m	77	nr
Gallitto et al[5]	2024	RS	36	m/f	82	58
Naoum et al[9]	2023	RS	6	m	81	54
Medda et al[21]	2023	RS	5	m	77	56
Bramucci et al[13]	2023	CR	1	m	80	60
Yammine et al[25]	2021	CS	5	m	80	nr
Schizas et al[24]	2021	CR	1	f	78	47
Koutsias et al[26]	2020	CR	2	m	77/88	60/66
Mauri et al[20]	2019	CR	2	f/m	83/72	62/60
Sato et al[23]	2018	CR	1	m	83	57
Rashid et al[22]	2017	CR	1	m	79	60
Kawashima et al[17]	2017	CR	1	f	91	45
Marchi et al[19]	2015	CR	1	m	78	59
Koudoumas et al[18]	2015	CR	1	m	74	50
Binder et al[12]	2015	CR	1	m	67	60
Aluko et al[11]	2015	CR	1	m	75	55
Chakraborty et al[14]	2013	CR	1	m	81	30
Smith MD et al[15]	2012	CR	1	m	85	70
Smith MD et al[16]	2012	CR	1	m	80	68

RS: Retrospective study; **CR:** case report; **CS:** case series; **AAA:** Abdominal aortic aneurysm

Table 2 (continue)

Table 2: Baseline characteristics of the eligible studies (TAVI+EVAR)

Author	Type & size of cardiac valve (mm)	Type & size of Aortic endograft (mm)	Type of repair and number of patients (n)		Complications		LOS (days)	FU (month)
			Simultanous	2nd stage	Early	Late		
PU et al[2]	nr	nr	98	47	nr	nr	nr	nr
Lu J et al[7]	nr	nr	11		ALI/femoral end-arterectomy and patch angioplasty	nr	3	nr
Boljevic et al[8]	34-mm EvolutPRO	nr	1		nr	type II endoleak	nr	6
Sanoussi et al[10]	26mm Sapien	Custom-made COOK Zenith	1		type II endoleak	nr	6	12
Gallitto et al[5]	nr	nr	25	19	TYPE II endoleak (N=2), MAE N=11	PTA recoil and pseu-doaneurysm	13	25
Naoum et al[9]	Sapien S3/ Evolut R	Zenith Alpha/ Endurant IIs/ Excluder	6		type II endoleak	nr	8	1
Medda et al[21]	Portico; Evolut Pro; Myval; Sapien;	Excluder		5	nr	nr	7	nr
Bramucci et al[13]	34-Evolut PRO	Zenith Alpha	1		nr	nr	5	2
Yammine et al[25]	nr	nr	5		type II endoleak	nr	5	12
Schizas et al[24]	25mm Portico	InCraft	1		nr	nr	13	1
Koutsias et al[26]	29 mm Evolut R PRO	Endurant (Medtronic)	2		nr	nr	10/8	24/12
Mauri et al[20]	26mm Sapien	Endurant / Excluder	2		nr	nr	8/12	12/6
Sato et al[23]	26mm CoreValve	Excluder	1		none	nr	8	nr
Rashid et al[22]	27mm Lotus	Cook Zenith Flex	1		none	none	nr	6
Kawashima et al[17]	23-mm Sapien XT	Cook Zenith Flex		1	none	none	22	nr
Marchi et al[19]	nr	nr	1					
Koudoumas et al[18]	31 mm Evolut R PRO	Ovation	1		Type III endoleak	nr	7	3
Binder et al[12]	27 mm LotusTM valve	nr	1		none	nr	nr	3
Aluko et al[11]	26 mm Edwards Sapien	Endurant	1		none	none	3	12
Chakraborty et al[14]	26-mm Sapien XT	Excluder	1		none	none	nr	nr
Smith MD et al[15]	29 mm Evolut R PRO	Cook Zenith Flex		1	none	none	14	6
Smith MD et al[16]	29 mm Evolut R PRO	Cook Zenith Flex	1		none	none	5	nr

nr: no-reported; **LOS:** length of stay; **FU:** follow-up

was used in five studies,[9, 10, 14, 17, 20] the Portico (Abbott Laboratories, Abbott Park, IL, USA) in two studies[21, 24] and the Lotus (Boston Scientific Corporation, Marlborough, MA, USA) in two studies.[12, 22] Main aortic endografts were the Zenith platform (Cook Medical, Bloomington, IN, USA) which was used in seven studies[9, 10, 13, 15-17, 22] the Excluder (W. L. Gore & Associates, Flagstaff, AZ, USA) was selected in five studies[9, 14, 20, 21, 23] the Endurant aortic device (Medtronic, Minneapolis, MN, USA) in four studies[9, 11, 20, 26] the InCraft (Cordis, Miami Lakes, FL, USA) and the Ovation (Endologix, Irvine, CA, USA) devices were selected in two studies.[18, 24] In the remaining cases, the type of thoracic endograft was not specified.

Early outcomes

During the early period, no deaths were noted. Type II endoleak was the most common complication after EVAR and was reported in four studies.[5, 9, 10, 25] One type III endoleak was reported in one study after use of the Ovation platform originating in the overlap of the limbs and the main body.[18] Serial balloon dilatations was performed and the final angiography revealed absence of any type endoleak and exlusion of the aneurysm sac.[18] Other additional complications were access related (n=2; pseudoaneurysm and dissection in the puncture site) with endarterectomy of common femoral artery and patchoplasty. Major adverse events were reported in six cases including cardiac events (n=1), respiratory adverse events (n=2) and cerebrovascular adverse events (n=1).[5] The mean length of stay was 8 days (range, 5-22 days).

Late outcomes

The mean follow-up duration was 7 months (range, 1-25 months). During this period, a recoil of iliac artery has been detected and was treated successfully putting a new stent graft.[5] Additionally one new pseudoaneurysm was detected and was treated in the same study.[5] Type II endoleak was detected after six months and was managed conservatively.

DISCUSSION

The coexistence of severe aortic valve stenosis and aortic aneurysmal disease represents a complex clinical scenario that is increasingly encountered in the aging population.[43] Advances in minimally invasive therapies have expanded treatment options for patients who are considered high or prohibitive risk for open surgery. In this context, the combination of transcatheter aortic valve implantation (TAVI) and endovascular aortic repair (EVAR or TEVAR) has emerged as a feasible alternative to conventional open approaches, aiming to reduce perioperative morbidity and mortality.[43,44]

The present review demonstrates that combined transcatheter and endovascular treatment has been applied predominantly in elderly, high-risk patients, with encouraging procedural success and acceptable short- to mid-term outcomes. The reported experience, although limited, suggests that both simultaneous and staged strategies are technically feasible.[2,5] However, the staged approach most frequently with TAVI performed prior to aortic repair appears to be favored in the majority of published cases.[26] This preference likely reflects concerns regarding the hemodynamic consequences of relieving aortic stenosis, which may increase aortic wall stress and theoretically predispose to aneurysm expansion or rupture if the aneurysm is left untreated.[45]

From a pathophysiological perspective, the sequences of procedures remains a matter of debate. Performing TAVI first may improve cardiac output and hemodynamic stability, thereby reducing the risk associated with subsequent endovascular aortic repair.[26] Conversely, aneurysm exclusion prior to valve intervention may theoretically protect against post-TAVI increases in systolic pressure and wall stress. [26] The available literature does not provide sufficient comparative data to support one strategy over the other, underscoring the importance of individualized decision-making based on aneurysm size, morphology, symptomatology, and overall patient risk profile.[41,42]

Current clinical guidelines from the American Heart Association (AHA) and American College of Cardiology (ACC) do not provide specific recommendations on the management of patients with concomitant aortic valve stenosis and aortic aneurysmal disease requiring combined transcatheter and endovascular repair.[44,45] However, the 2022 ACC/AHA Guidelines for the Diagnosis and Management of Aortic Disease, which addresses thoracic aortic pathology more broadly, underscores the importance of multidisciplinary care and procedural expertise when managing complex aortic conditions, including the use of endovascular techniques such as TEVAR in anatomically suitable patients.[44] In addition, established ACC/AHA valvular heart disease guidelines emphasize that TAVI should be performed by experienced multidisciplinary Heart Team programs with comprehensive preprocedural planning to optimize outcomes.[46] These guideline principles implicitly support cautious application of combined strategies, but specific evidence-based recommendations for sequencing or integration of TAVI and TEVAR / EVAR in the same patient remain unavailable, reflecting the need for further data.

In contrast to the small case reports and case series included in the present review where no procedural complications were documented, data from larger observational cohorts studies of TEVAR / EVAR and TAVI in similar high-risk populations have reported notable adverse event rates.[2,5] Specifically, late complications following thoracic endovascular aortic repair (TEVAR), including endoleaks, and need for secondary interventions, have been observed in up to approximately 38% of patients in broad clinical series, with reinterventions reported in nearly one-quarter of cases.[2,5,43] This highlights that while individual case reports may not capture the full spectrum of complications, aggregate experience from larger populations demonstrates important procedural risks that must be considered when planning combined interventions. Likewise, large registries of TAVI have identified vascular access complications, stroke, and conduction disturbances as recognized adverse outcomes, underscoring that procedural safety in real-world cohorts may differ from isolated case descriptions.[43]

Future research should focus on multicenter registries and collaborative studies to better define patient selection criteria, procedural sequencing, and long-term outcomes. The establishment of standardized reporting frameworks may further enhance comparability across studies. Until higher-level evidence becomes available, the management of patients with concomitant severe aortic stenosis and aortic aneurysmal disease should rely on a multidisciplinary Heart-Vascular Team approach, integrating cardiology, cardiac surgery, and vascular surgery expertise.[47]

CONCLUSION

In conclusion, combined TAVI and endovascular aortic repair represents a contemporary, minimally invasive treatment option for selected high-risk patients with concomitant aortic valve and aortic pathology. While early outcomes are encouraging, further evidence is required to establish standardized treatment algorithms and long-term safety.

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CASE REPORT

Open conversion for atypical endoleak after EVAR without endograft explantation: a case report

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Introduction: Endoleaks are significant complications following Endovascular Aortic Aneurysm Repair (EVAR). However, persistent aneurysm sac enlargement without an identifiable endoleak remains challenging.

Case Report: We report a 78-year-old man who developed progressive aneurysm sac expansion to 64 mm five years after EVAR despite prior inferior mesenteric artery embolization. Computed Tomography Angiography (CTA) demonstrated focal delayed enhancement within the aneurysm sac without evidence of type I, II, or III endoleak. Open surgical exploration revealed diffuse oozing from the intimal surface without identifiable branch inflow or endograft defect. Sacotomy with endograft preservation was performed, including oversewing of a small bleeding vessel, adjunctive argon plasma coagulation (APC), and fibrin sealant application. The postoperative course was uneventful, and follow-up imaging demonstrated sac shrinkage without recurrent endoleak.

Conclusion: This case highlights open surgical repair with endograft preservation as a valuable option for persistent sac enlargement after EVAR when endovascular treatment fails or no discrete bleeding source is identified in selected patients.

Keywords: EVAR, atypical type II endoleak, aneurysm sac expansion, open conversion, endograft preservation

INTRODUCTION

With the development of endovascular aortic techniques, endovascular aortic aneurysm repair (EVAR) has become a primary approach for the treatment of Abdominal Aortic Aneurysms (AAA).[1] However, aneurysm sac enlargement not responsive to endovascular treatment represents a leading cause of reintervention after EVAR.[2] Persistent sac expansion can lead to potential rupture, which compromises not only the endograft but also the patient's life.[3,4]

Current clinical guidance emphasizes imaging-based characterization of post-EVAR sac enlargement, prioritizing the exclusion of type I and III ELs before attributing sac growth to type II EL, endotension, or hidden sources.[5] The European guidelines define atypical type II EL (aT2EL) as sac expansion in the absence of a clearly defined endoleak cavity or identifiable arterial inflow, often associated with delayed-phase mural enhancement and hypertrophied adventitial vasa vasorum on imaging; this entity has also been described as "Moyamoya endoleak" in selected reports.[6,7]

This report describes a case of persistent aneurysm sac enlargement after EVAR despite prior embolization of the Inferior Mesenteric Artery (IMA), ultimately managed with open sacotomy and endograft preservation. Written informed consent for publication was obtained from the patient.

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CASE REPORT

A 78-year-old man was referred to our center because of progressive aneurysm sac enlargement reaching 64 mm, five years after EVAR performed with a Medtronic Endurant stent graft (Medtronic, Minneapolis, MN, USA). The aneurysm sac had progressively enlarged despite previous embolization of the inferior mesenteric artery performed six months before presentation.

Computed tomography angiography (CTA) performed at admission demonstrated the absence of patent lumbar arteries or inferior mesenteric artery inflow, with focal delayed enhancement within the aneurysm sac thrombus. No evidence of type I, II, or III endoleak was identified. The endograft remained patent, with preserved perfusion of the iliac vessels **(Figure 1A, 1B)**.

On presentation, the patient was asymptomatic and hemodynamically stable. His medical history was significant for end-stage renal disease requiring chronic hemodialysis. Open surgical repair was performed through a xipho-umbilical midline laparotomy using an Alexis® retractor (Applied Medical Resources Corp., Rancho Santa Margarita, CA, USA) **(Figure 2).** The aneurysm was exposed from the level of the renal arteries to the iliac bifurcation. The aneurysm sac appeared tense but non-pulsatile, and intra-sac pressure measurements confirmed the absence of pulsatility. The proximal aortic neck and iliac arteries were encircled with Teflon felts and secured using transfixing 2-0 polypropylene sutures incorporating both

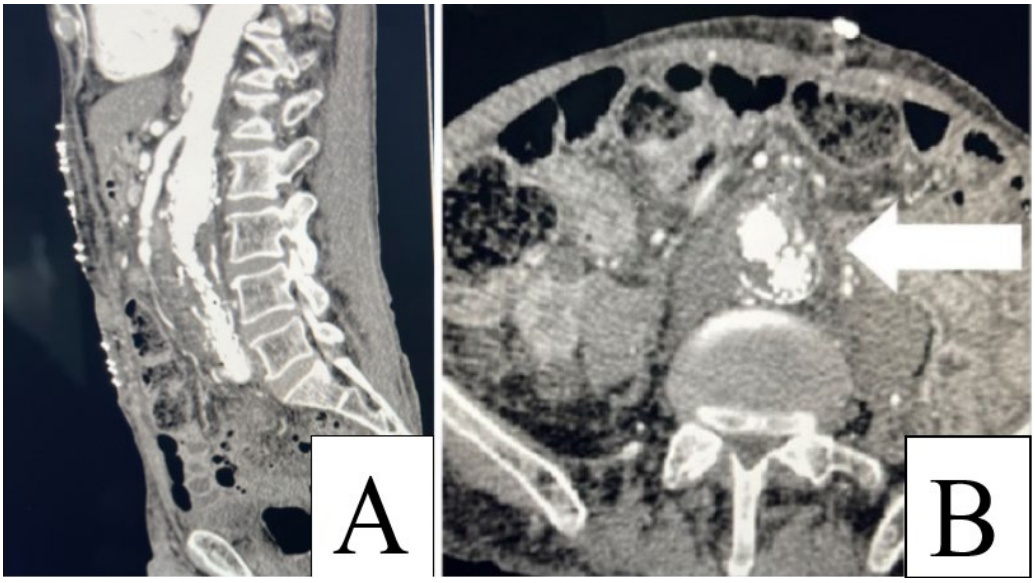

Figure 1. Contrast-enhanced computed tomography images five years after EVAR. **(A)** Sagittal reconstruction and **(B)** axial images of the aneurysm sac 64 mm. Hyperattenuating foci seen within the aneurysm sac indicate the presence of endoleak (arrow).

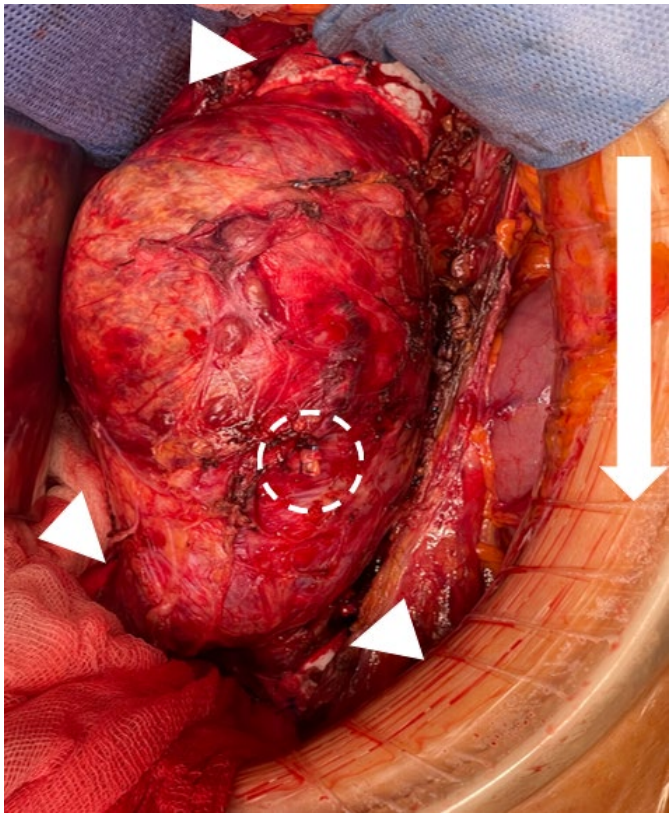

Figure 2. Xipho-to-umbilical exposure of the abdominal aorta using an Alexis® retractor (arrow), with no evisceration. The infra-renal aorta and proximal common iliac arteries were encircled with Teflon felts for proximal and distal support (arrowhead). The previously embolized inferior mesenteric artery was ligated (dashed circle).

the endograft and the native aortic wall. The previously embolized IMA was identified, dissected, transected, and ligated using 4-0 polypropylene sutures **(Figure 2)**.

The aneurysm sac was opened without aortic cross-clamping. Following decompression, the cavity contained chronically organized thrombus mixed with fresh blood. After thrombus evacuation, diffuse oozing from the aneurysm sac intimal surface was observed without identification of a discrete bleeding source. The proximal and distal endograft attachment sites appeared intact. Additional transgraft fixation sutures were placed at the overlap of the iliac limb to improve mechanical stability and prevent device displacement during surgical manipulation **(Figure 3)**.

A small posterior wall bleeder was oversewn to achieve focal hemostasis. Because diffuse oozing persisted, coagulation diathermy and Argon plasma coagulation (APC; Erbe Elektromedizin GmbH, Tübingen, Germany) were applied to the aneurysm sac surface to achieve complete hemostasis **(Figure 3A, 3B)**.

After sac reduction, adjunctive fibrin sealant was applied to reinforce hemostasis **(Figure 4)**. The residual aneurysm sac was then closed over the preserved endograft, and the retroperitoneum was drained and closed **(Figure 4A, 4B)**.

The postoperative course was uneventful, and the patient was discharged on postoperative day 4. The CTA performed at three months demonstrated sustained reduction in sac size, with no evidence of EL.

DISCUSSION

Endoleaks remain one of the principal determinants of long-term EVAR durability. Persistent aneurysm sac enlargement in the absence of a clearly identifiable EL represents a particular-

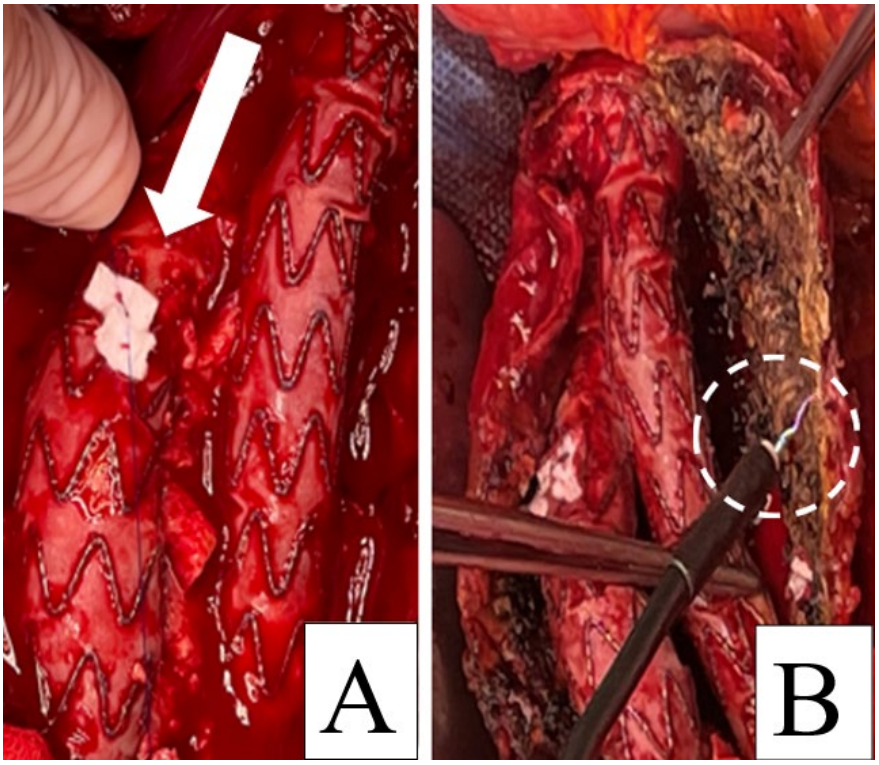

Figure 3. (A) Transgraft suture (arrow) is passed through the aortic endograft main body, and the overlap of the iliac limb to provide mechanical fixation and prevent graft displacement during manipulation. Multiple pledgets are secured to reinforce fixation. **(B)** Argon plasma coagulator applied to the aneurysm sac (dashed circle).

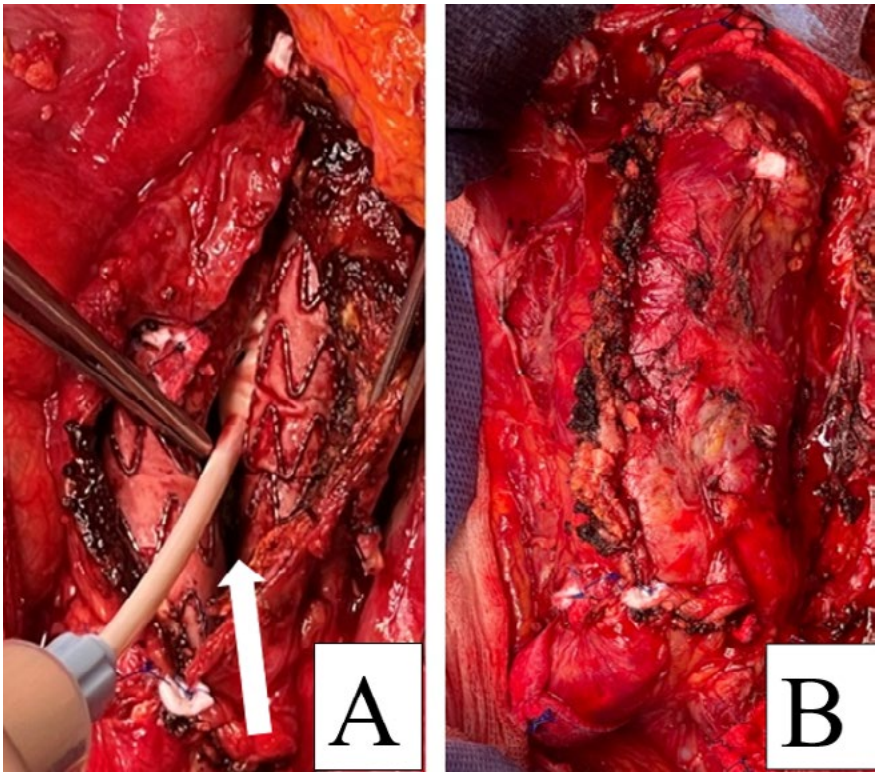

Figure 4. (A) Fibrin glue (arrow) is applied after sac reduction, with iliac branches carefully repositioned to allow complete access to the sac**.** Glue is placed posteriorly along the aneurysm sac to ensure thorough hemostasis and avoid oozing. (**B)** Sac reduction and closure over the endograft.

ly challenging clinical scenario because occult sac pressurization may ultimately result in aneurysm rupture. Although the exact criteria requiring intervention have not been definitively established, continued sac growth despite repeated imaging surveillance generally warrants active treatment.[8]

Several methods for managing type II ELs have been reported. To prevent aneurysm sac enlargement after EVAR, perioperative coil embolization via trans-arterial or trans-lumbar approaches is the most common method. Despite the success of this technique in treating type II ELs, repeated interventions are frequently required. Notably, 51% of patients treated with coil embolization alone undergo additional procedures. High rates of subsequent embolizations are reported in those undergoing these procedures. These reports suggest that patients require regular surveillance[10] after coil embolization and may require multiple interventions. Repeated interventions carry additional risks, including infection, nephrotoxicity from contrast agents - particularly relevant in patients with chronic renal failure - and increased radiation exposure during CT-guided trans-lumbar procedures.[3,4]

Current European Society for Vascular Surgery (ESVS) guidelines recommend consideration of elective open conversion, with or without endograft preservation, in patients with persistent aneurysm sac enlargement despite prior endovascular treatment attempts.[5] These recommendations underscore the role of open surgical repair as a valuable therapeutic option after failure of less invasive strategies.

Nevertheless, open surgical repair remains technically demanding and more invasive than embolization, with potentially increased morbidity and mortality related to aortic cross-clamping, endograft-associated inflammatory changes, and anastomotic bleeding.[7] The prevalence of advanced age, cardiovascular disease, pulmonary comorbidities, and impaired physiological reserve among EVAR patients further contributes to perioperative risk.

Sacotomy with preservation of the endograft offers several potential advantages because it avoids complete graft explantation and may eliminate the need for aortic cross-clamping. This approach may therefore reduce operative stress while allowing direct treatment of sac pressurization, and being feasible also in high - risk patients. However, sacotomy also has limitations, including the possibility of missed occult ELs, incomplete treatment, and future aneurysm sac re-expansion. Consequently, long-term postoperative imaging surveillance remains mandatory.

An interesting case report published by Kinugasa et al.[8] showed that one of the potential causes of EL is a microvascular conduit from the adventitia to the lumen across an injured intima-media interface. They reached this conclusion after treating a patient with persistent EL 10 years after EVAR, despite successful embolization of the IMA. Histopathological assessment of the sac showed a distinctive triad of: extensive intimal denudation, media exposure with luminally oriented microvessels, and adventitial vasa vasorum proliferation.

Onitsuka et al.[9] demonstrated that endoaneurysmorrhaphy that combines sacotomy, ligation of the back-bleeding vessels, and endograft preservation for type II EL can reduce operation time and result in a less invasive OSR than with graft removal. Short-term outcomes included shrinkage of the aneurysm sac with a stable diameter, no missed type II EL, no type II EL recurrence, and no evidence of endograft migration or disjunction. In addition, in cases of endograft preservation, the proximal neck banding method may further prevent aneurysm re-enlargement and type Ia EL secondary to neck-dilatation.

Ultimately, with the development of endovascular techniques, there is a gradual trend toward replacing open surgery. However, in complex cases, such as those with complicated anatomy or EVAR repair failure, traditional embolization materials or catheter techniques may not fully exclude ELs. In these cases, operative time may be prolonged, radiation exposure may increase, and the risk of surgical failure is higher. Additionally, repeated endovascular repairs may lead to long-term complications such as aneurysm sac enlargement, graft wear, infections, and other issues, which may sometimes require open surgery for definitive resolution.[1]

CONCLUSION

Management of atypical type II endoleaks (aT2ELs) associated with persistent aneurysm sac enlargement after EVAR remains challenging, particularly after failed embolization attempts. In selected patients, open surgical repair with endograft preservation represents a valuable treatment option when further endovascular or minimally invasive approaches are not feasible or have proven ineffective. This strategy allows direct control of sac pressurization, prevents further aneurysm sac enlargement, and may contribute to the long-term durability of the previous EVAR repair while avoiding the morbidity associated with complete graft explantation.

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CASE REPORT

A Huge Ruptured Pseudoaneurysm of the Radial Artery Presented One Year After the Coronary Intervention - A Case Report

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Introduction: Radial artery pseudoaneurysms (PSAs) are rare, especially when they are of huge size and present with rupture. There is no consensus regarding optimal management, and treatment varies according to size and presentation.

Case Report: We aim to present a case with a late rupture of a huge radial artery PSA, one year after coronary intervention.

Conclusion: Early detection and treatment is always recommended when the PSA increases in size or shows signs of imminent rupture.

Keywords: radial artery, pseudoaneurysm, rupture, skin necrosis, large size, treatment

INTRODUCTION

Radial artery pseudoaneurysms (PSAs) presenting after coronary interventions are extremely rare, with an estimated incidence of <0.1% according to literature.[1] They are usually associated with pain and discomfort if they keep increasing in size. If they are left untreated, they could lead to expansion and rupture.[2] Ruptured radial artery PSAs are even rarer, and they could threaten the patient's life if not treated emergently.[1]

We aim to report on the case of a huge radial artery PSA presented with a late rupture and treated in our department.

CASE REPORT

A 74-year-old patient with a history of coronary intervention and stent placement one year ago, atrial fibrillation (AF), and depression was transferred to our emergency department (ER) with active bleeding due to a huge radial artery PSA on his right forearm. The patient was under per os antiplatelet and anticoagulant treatment for both his coronary disease and AF. Although his personal cardiologist had diagnosed the PSA many months ago and suggested that the patient should visit a vascular surgeon, the patient did not follow his advice which led to the expansion and rupture of the PSA.

The PSA and the point of bleeding were securely pressed with bandages in the ER, and the patient was transferred immediately to the operating room (OR). The patient was still hemodynamically stable despite the blood loss although serum hemoglobin levels were lower than normal. The PSA was 10 x 7 cm in size with central skin necrosis due to expansion **(Fig. 1A)**. Under general anesthesia and proximal arterial occlusion with an external tourniquet device, a longitudinal incision was made and all the necrotic skin was removed. A large amount of thrombus and necrotic pseudo-membranes were removed

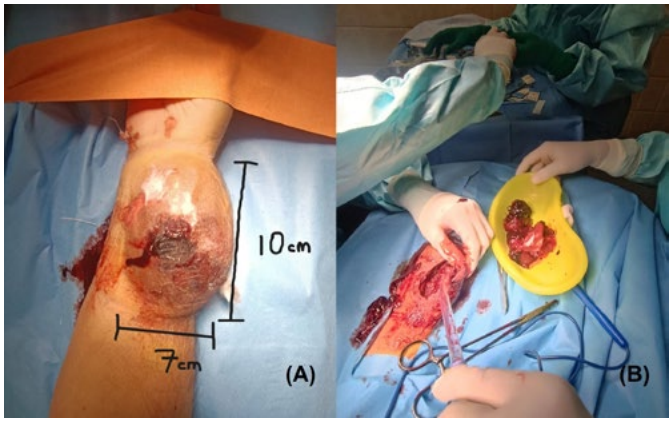

Fig 1. (A) Figure showing the size of the huge radial artery pseudoaneurysm. In the center, skin necrosis and bleeding through the skin are visible. **(B)** Large amount of thrombus and pseudo-membranes that were removed from the pseudoaneurysm.

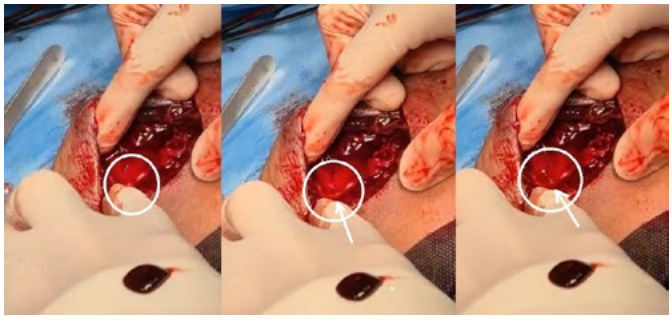

Fig 2. Figures showing the rupture site (circle) with the active bleeding when the tourniquet was depressurized.

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(Fig. 1B) and the point of rupture was detected after depressurizing the tourniquet **(Fig. 2)**. The artery was sutured with non-continuous Prolene 6-0 sutures. Additionally, hemostatic dressings were placed within the wound cavity due to diffuse bleeding (possibly due to the combined antiplatelet and anticoagulant treatment).

The radial artery was palpable distally after the closure of the artery. A vacuum drainage was placed and the skin was closed with Nylon 3-0 sutures. The postoperative course of the patient was uneventful.

DISCUSSION

Both a radial artery PSA delayed presentation and rupture are extremely rare in literature. Additionally, there is no consensus regarding proper management. In a large retrospective study by Harvey et al. including over 10,000 radial catheterizations, the incidence of a radial artery PSA was 0.06%.[3] Radial artery PSAs present after different types of interventions such as arterial line placement or coronary artery catheterization. They present usually after removal of the catheter or the sheath and inadequate pressure appliance over the puncture site.[4] The artery is mostly affected at the distal part where its position is more superficial. A significant predisposing factor is anticoagulant or dual antiplatelet treatment.[4] Also, local infection can be a predisposing factor.[5] According to literature, most of the cases progress slowly and only one third of the cases will require an urgent operation.[1]

The patient may present early after the intervention with local swelling or a pulsatile mass, pain, or discomfort.[6] Local signs of inflammation or infection may be present. Although the PSA could be asymptomatic when it is small, it may be painful when it increases in size. The ultrasound remains the first-choice method to diagnose the PSA and to determine its morphology as well. If it increases in size or when a local infection is present, the risk of rupture is higher. However, in some cases, a computed tomographic angiography (CTA) may be necessary.[1,4]

When an intervention is required, several treatments have been suggested. A percutaneous thrombin injection under ultrasound guidance remains one option.[7] However, one should keep in mind that a wide PSA neck will increase the risk for failure of PSA occlusion and for distal thrombin embolism. Additionally, the PSA could be occluded right after the thrombin injection and a recurrence could be detected during follow-up, especially if the patient is under full anticoagulant treatment.[7] Another option is plain ultrasound guided compression (UGC).[8] However, thrombin injection has been found to be superior to UGC as it achieves thrombosis within 6 seconds compared to UGC that may require up to 45 minutes.[8] UGC can also cause rupture to larger PSAs as well. According to some authors, UGC may be selected for pediatric cases.[8] When the PSA is under 2 cm in diameter, there is a 70% possibility that it will spontaneously get thrombosed. Spontaneous thrombosis may occur within 4 weeks. UGC may have an effectiveness between 60% and 90%.[8]

However, infection, skin ischemia and frail appearance of the PSA are contraindications for any pressure appliance.[9] In these cases, an open repair is recommended. Our patient presented late after the intervention (almost a year) with a huge, ruptured PSA combined with skin necrosis, that needed emergency surgical treatment. Usually, the point of arterial rupture is found and a simple suturing of the artery is adequate such as in our case. However, ligation of the artery remains an alternative when anastomosis or suturing the artery is not possible.[9] Ligation should be performed when it is tested that the ulnar artery is patent, when there are good collaterals in the angiography or excellent back bleeding from the distal stump.[6] Finally, there are some case reports referring to endovascular treatment of radial artery PSAs either with covered stent placement or embolization. However, these cases are extremely rare.[10]

CONCLUSION

In conclusion, ruptured artery PSAs of large size are rare but life-threatening. Therefore, early detection and treatment is always recommended when the PSA increases in size or shows signs of imminent rupture. When ruptured, open repair is the most frequent strategy with the aim to preserve the distal perfusion.

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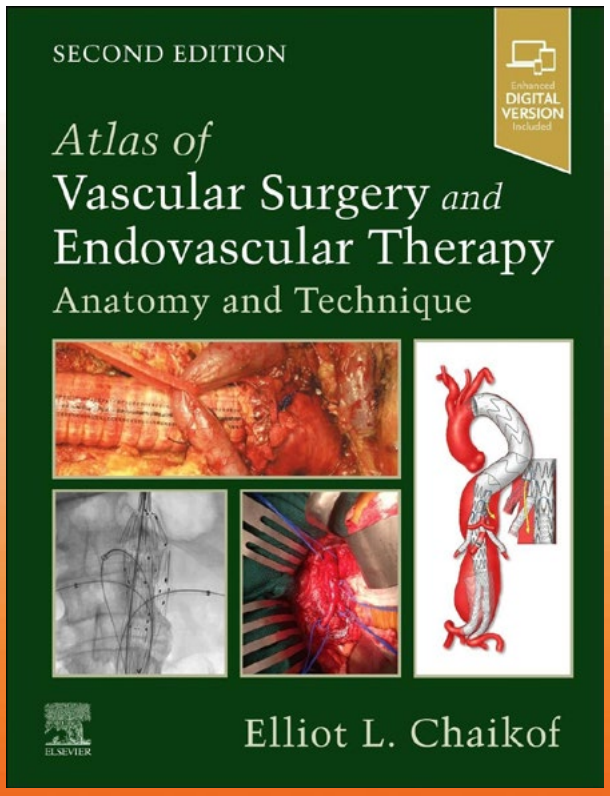

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