Current Trends and Evidence on Post-Thoracic Endovascular Aortic Repair Aorto-Pulmonary and -Bronchial Fistula: A Narrative Review

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Background: Aorto-bronchial or aorto-pulmonary fistulas (ABPF) are a rare but life-threatening complication following thoracic endovascular aortic repair (TEVAR). This narrative review aims to provide an overview of the current trends and available evidence on ABPF following TEVAR, evaluating risk factors, diagnostic approaches, and possible preventive and therapeutic strategies.
Methods: Relevant publications on post-TEVAR ABPF were selected through a literature search on PubMed. Studies providing data on post-TEVAR ABPF concerning pathogenesis, clinical presentation, diagnostic tools, surgical approaches, and outcomes were included.
Results: The literature search obtained 169 articles. After selection, 37 studies, published between 2000 and 2024, remained for analysis. The primary symptom of ABPF is typically hemoptysis, which may be recurrent or persistent. The diagnostic pathway for ABPF encompasses medical history, clinical evaluation, blood tests, sputum and blood cultures. Computed tomography angiography is the first-line imaging modality in suspected ABPF, while bronchoscopy is reserved for hemodynamically stable patients. Moreover, 18-fluoro-deoxyglucose positron emission tomography can aid in the diagnosis of graft infection. Post-TEVAR ABPF are associated with multiple risk factors, mainly patient-specific clinical conditions (i.e. chronic inflammatory conditions, underlying mediastinal oncologic pathologies, and infectious diseases) and anatomical characteristics, mainly large aortic aneurysms, severe aortic tortuosity, and an extensive periaortic hematoma. Strategies to minimize the risk of ABPF formation include appropriate device oversizing, long sealing zones, limited extent of aortic coverage, selective hematoma evacuation, and strict adherence to sterility and antibiotic prophylaxis. Radical surgical interventions have shown superior outcomes compared to endovascular approaches, which may be adopted as initial step to stabilize the patient in a staged treatment.
Conclusion: Post-TEVAR ABPF is a rare and life-threatening condition. Literature is scarce and a deeper understanding of risk factors, diagnostic pathways, and therapeutic strategies is essential for the prevention and management of ABPF secondary to TEVAR.

Disclosures: The authors have nothing to disclose.

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Ann Vasc Surg 2025; 114: 398–404
https://doi.org/10.1016/j.avsg.2024.12.061

Manuscript received: December 13, 2024; manuscript accepted: December 29, 2024; published online: 22 January 2025

INTRODUCTION

Aorto-bronchial or aorto-pulmonary fistulas (ABPF) are abnormal connections between the thoracic aorta and the bronchial tree or the pulmonary parenchyma, respectively. Primary ABPF arise from pathological conditions, mainly oncological, infectious, or traumatic causes. Secondary or iatrogenic ABPF occurs as a consequence of surgical or endovascular procedures − often cardiothoracic surgery. ABPF present in most instances with severe or intermittent bleeding into the airways (hemoptysis), and are typically related to infectious complications and high mortality.

Thoracic endovascular aortic repair (TEVAR) is a well-established procedure to treat a wide range of pathological conditions affecting the thoracic aorta, for example, thoracic and thoraco-abdominal aortic aneurysms, blunt traumatic aortic injury, intramural hematoma, penetrating aortic ulcer and (un)complicated type B aortic dissections (TBAD).[1] Nowadays, TEVAR represents the first choice for the management of these pathologies in the descending aorta, both in an elective or in an urgent setting. In the European Registry for Endovascular Aortic Repair Complications (EuREC), the incidence of ABPF after TEVAR was 0.40/1000 TEVARS/year (range: 0.08−2.36).[2] Others have reported the incidence of post-TEVAR ABPF to range between 0.5 and 0.8%.[3,4] A recent retrospective study reported an incidence of 1.9% (1 patient) among a cohort of 52 patients previously treated with a TEVAR for a chronic TBAD.[5] On the other side, TEVAR has been identified as the underlying cause of an ABPF in 14% of the cases.[6] Despite the technical and design advancements of the contemporary available thoracic aortic endografts, this rare life-threatening complication of TEVAR shows indeed an increasing incidence.[6] ABPF usually arise late after TEVAR and are always fatal if left untreated.[4,7−10] Apart from ABPF, aortoesophageal fistulas represent another life-threatening complication after TEVAR. While ABPF and AOF share the same pathogenesis and poor prognosis, AOF present with an earlier onset and with hematemesis as the main symptom, rather than hemoptysis.[2,11,12] Both ABPF and AOF have been demonstrated to have comparable incidence following open aortic repair or TEVAR.[4]

As a result of the low incidence of ABPF after TEVAR high quality evidence on the true epidemiology, diagnosis and management of post-TEVAR ABPF is lacking. This narrative review aims to provide an overview of the current trends and available evidence on ABPF following TEVAR, evaluating risk factors, diagnostic approaches, and possible preventive and therapeutic strategies.

METHODS

Given the expected low quality of evidence and low incidence of post-TEVAR ABPF, we did not aim to provide a systematic review and/or meta-analysis on any of the aspects of post-TEVAR ABPF. We performed the search below in order to cover the most relevant literature on the topic and did not apply any in- or exclusion criteria in order to provide a comprehensive overview on all aspects of post-TEVAR ABPF.

A literature search was performed using PubMed (US National Library of Medicine, Bethesda, MD, USA). No temporal restrictions on the publication dates were applied to the included studies. The following keywords were used: "Aorto-pulmonary fistula", "Aorto-bronchial fistula", "Aortopulmonary fistula", "Aortobronchial fistula", "Aorto-pulmonary shunt", "Aorto-bronchial shunt", "TEVAR", "Thoracic Endovascular Aortic Repair", "Endovascular Repair of Thoracic Aorta", "postoperative complications", "post-surgical complications", "post-procedural complications", "outcomes", "management", "treatment", "current trends", "evidence". The publications providing data about ABPF secondary to TEVAR procedures or analyzing their pathogenesis, clinical presentation, diagnostic tools, surgical approaches, and outcomes were included.

RESULTS

The literature search retrieved 169 articles. After the removal of 49 duplicates, 120 articles were screened by the title and abstract. Cross-referencing yielded 1 additional study. Eighty-four records were excluded, including those focusing solely on AOF, those addressing ABPF without TEVAR, and those discussing both ABPF and AOF without specific data on ABPF patients. Thirty-seven studies, published between 2000 and 2024, were included and further examined (Fig. 1). Among these studies, 26 were case reports, 7 were monocentric retrospective studies, 1 cross-sectional observational study from a national survey, 1 retrospective observational study with data from an international multicenter registry, and 2 narrative reviews (published in 2003

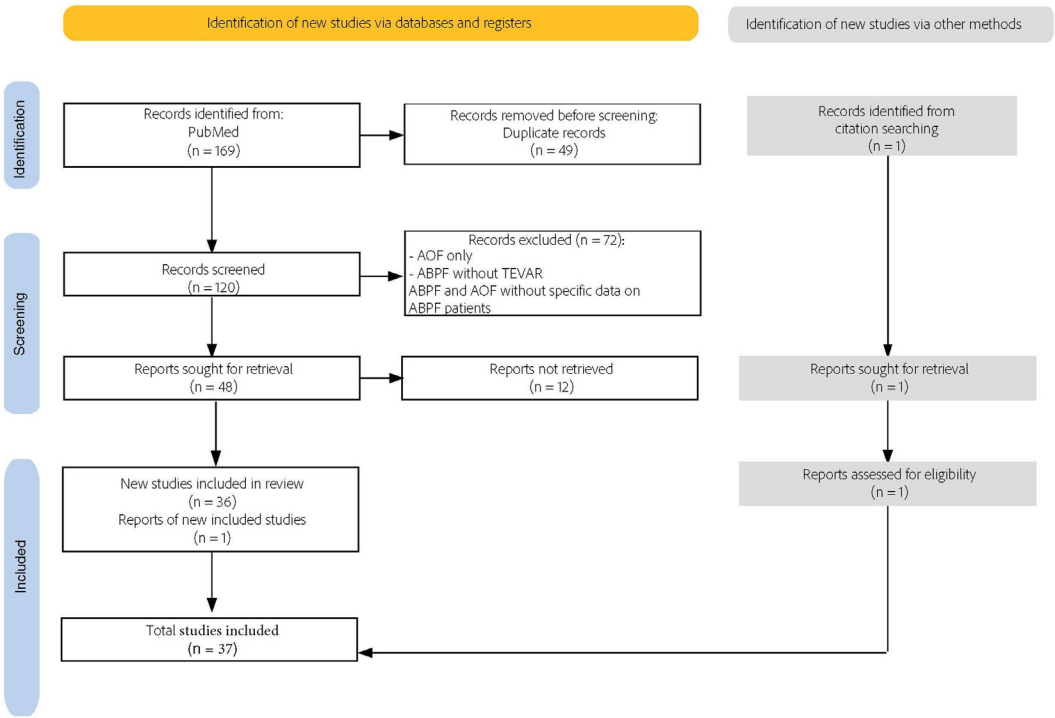

Fig. 1. Literature search.

and 2015). These studies were not all separately covered in this narrative review, but references provided in Supplementary Table 1.

Clinical Presentation

Patients with ABPF may present with a range of symptoms, depending on the severity and evolution of the condition. Common clinical manifestations include hemoptysis, recurrent or persistent chest pain, dyspnoea, recurrent fever, and signs of systemic inflammation.[4,13] Hemoptysis is often the cardinal presenting symptom.[2,4,12] It can also be the only symptom and is often intermittent, especially if the size of the ABPF is small and is temporarily occluded by a clot.[14] Usually even small fistulas tend to enlarge over time, with more frequent and profuse bleeding while the fistulous communication enlarges.[12,14,15] Most of the time, hemoptysis brings the patient to seek medical attention.[6] However, sometimes the first clear manifestation is a massive bleeding, possibly leading to respiratory distress and hemodynamic instability. Hypovolemic shock has been reported to be the primary clinical presentation of ABPF in 6.46% up to 23% of the cases.[6,12] The first medical doctor encountered by a patient with an ABPF typically is not a vascular surgeon. This underlines the importance of the awareness of this complication of TEVAR among other specialties experts.

Diagnosis

The diagnostic path of an ABPF encompasses medical history, clinical evaluation, blood tests (mainly C-reactive protein, procalcitonin, white blood cell count), and sputum and blood cultures.[6,15,16]

Computed tomography angiography (CTA) is the first-line imaging modality in case of suspected ABPF,[15,17,18] thanks to high-resolution images that can identify and determine their presence, location and extent. Signs suggesting ABPF are periaortic hematoma with or without contrast extravasation and periaortic air, and lung parenchymal changes.[3] Periaortic hematoma has been reported to be present in up to 65% of the cases of ABPF, being the most common radiologic sign.[2]

Especially for patients with impaired kidney function, magnetic resonance imaging may represent an alternative that may also complement CTA information by providing details about the soft tissue involvement.[18] Bronchoscopy represents a further investigation, but might not be diagnostic when the ABPF is located in the peripheral lung parenchyma.[15,19] Bronchoscopy is indicated when a patient is hemodynamically stable. However, it

carries the risk of hemorrhagic complications during the procedure itself.[19,20] Bronchoscopy can help identifying the site the ABPF and collecting samples for microbiological tests.[6]

Furthermore, 18-fluoro-deoxyglucose positron emission tomography can help in differentiating between infectious processes and sterile inflammation.[15,17,18]

Pathophysiology and Risk Factors

ABPF following TEVAR typically involve the left lung due to the anatomical relationship with the descending thoracic aorta. However, right-sided ABPF are still possible,[21] as well as tracheal fistulation,[2] considering additional predisposing factors, such as large aneurysms, presence of bacterial infections, endograft migration or displacement, and severe tortuosity and angulation of the aorta.

Three main factors may potentially contribute to the formation of an ABPF after TEVAR: 1. The presence of a local infection, 2. An inflammatory component due to the foreign body response to the endograft, and 3. The reciprocal compression of the structures in the thorax. The stent graft itself can serve as the primary or secondary site for the onset and development of an infection. The infection may originate from several sources, including intraprocedural contamination, hematogenous seeding from a distant infectious source, or bacterial translocation from an adjacent focus of infection. The commercially available endografts use different materials. Available studies did not find a different susceptibility to infection among the different materials and endografts.[15,22] Indeed, multiple commercially available endografts have been reported with ABPF as late complication.[4] A specific analyses focused on the correlations between ABPF and type of endograft is hampered by the low sample size and hence type II statistical error. In some patients the endograft materials may induce an excessive local inflammatory response.

Chronic inflammation or the presence of an infection can both compromise the aortic vessel integrity and lead to consequent tissue weakening. Extrinsic compression may result from the implanted stent graft, the aneurysm sac or a periaortic hematoma derived from aortic rupture. Moreover, these factors are superseded by the continuous pulsatile arterial pressure waves. All these mechanisms together induce tissue necrosis and erosion, until final rupture of the arterial compartment into the pulmonary one leads to fistulation.[14] This pathological sequence of events explains the late occurrence of ABPF. The time interval between the initial TEVAR procedure and the development of ABPF is variable, mostly reported to be of several months.[2,4,7,13] It was 10.9 months in the study by Chiesa et al. and 10.2 months in the cohort of the European Registry for EuREC.[2] To prevent ABPF formation, as well as fistulation in other mediastinal organs, after emergent TEVAR, staged evacuation of the hematoma resulting from aortic rupture has been suggested.[11]

Endoleaks, leading to persistent pressurization of the aneurysmal sac and potential enlargement of its diameter, have been identified as a possible contributing factor in the formation of ABPF fistulas.[2,9,11,21] In the series reported by Czerny, 10 out of 26 (38%) patients with an ABPF were associated with a persistent endoleak after TEVAR (the majority being type I or III endoleaks), highlighting a potential link between the two.[2] In the case series described by Chiesa, which included only 6 ABPF, no significant correlation with endoleaks was identified.[4] Overall, the consensus among authors[6,11,12,21,23] emphasizes the importance of preventing type I and III endoleaks through adequate overlapping in secure landing zones. When necessary, this may require extension of the sealing zone proximally to the arch branch vessels, typically the left subclavian artery (LSA), to ensure proper sealing. This may include covering the LSA, in these cases a subclavian artery bypass or transposition or endovascular options to preserve the LSA can be considered.

In the EuREC cohort, the median aneurysm sac diameter among patients with ABPF was remarkably large, 9.2 cm.[2] This finding can be explained by the fact that a large aneurysm significantly alters the geometry of the mediastinal structures, potentially leading to mechanical compression of the bronchi or lung tissue.[21] The use of TEVAR in these cases, while less invasive than open surgery, may further distort the anatomy and exacerbate the compression, potentially increasing the risk of ABPF formation. For these reasons, the authors suggest considering an open surgical approach in these anatomical scenarios, or alternatively, a TEVAR combined with a saccotomy for decompression during the initial postoperative period.

A significant percentage of patients presenting with an ABPF received the previous TEVAR in an emergent setting. In the EuREC, half of the patients underwent emergency TEVAR.[2] Most of these patients, also had a thoracic hematoma of various sizes. The presence of a hematoma may determine the risk of fistulation through both the extrinsic compression and related inflammation. However, the emergent setting may also contribute to a less optimal planning and sizing resulting in suboptimal technical

performance increasing the risk of ABPF. The group of Chiesa found that 42% of patients presenting an ABPF or an AOF had a proximal oversizing at least of 20%.[4] Other authors have supposed that excessive oversizing of stent grafts can lead to long-term degradation of the arterial wall, and consequent fistulation.[2,11,12,24] Czerny and colleagues advocate for an oversizing never exceeding the 30%.[2]

Czerny and colleagues also speculate on a possible ischemic etiology of ABPF, due to coverage of feeding bronchial arteries, which was reported as the underlying cause of fistulation in up to 12% of the cases.[2] However, a direct correlation with the length of coverage has not been found until now.[6]

Treatment Strategies

Since a direct communication between the nonsterile airways and an endovascular graft indicates a graft infection, antimicrobial therapy is always required.[15] Broad-spectrum intravenous antibiotic therapy should be started empirically, while waiting for cultures' results for a targeted antibiotic strategy and, eventually, applying antifungal agents.[15] The cultures can be based on sputum, bronchial, direct tissue or blood samples. Although there is no consensus about the optimal duration of antimicrobial treatment, it should be continued intravenously for at least 2 weeks and, thereafter, for 2–4 weeks.[15] Long-term or lifelong antimicrobial suppression may be necessary in selected cases or in patients unfit for open surgery.[15]

Although ABPF is recognized as a serious potential complication of TEVAR, the main European consensus documents and guidelines do not provide detailed, specific recommendations on its optimal surgical or endovascular management. However, the guidelines emphasize the need for management in high-volume, experienced centers and advocate an interdisciplinary approach.[1,17,25] Indeed, multidisciplinary collaboration involving vascular surgeons, cardio-thoracic surgeons, pneumologists, anaesthesiologists, radiologists and infectious disease specialists is crucial to reach the optimal patient care. In addition, postoperative care should focus on infection treatment, monitoring for signs of recurrence, and ensuring proper and lifelong follow-up imaging to assess the integrity of the repair and detect potential complications at an early stage.[15]

Traditionally, the treatment of ABPF was based on an open surgical repair, which involves the reconstruction of the aorta and a lobectomy. In the past few decades, endovascular techniques have been developed and used to treat primary fistulas, although concerns remain about their long-term effectiveness. In ABPF following a TEVAR, the deployment of a second thoracic endograft is employed in some cases as bridging procedure to stabilize patients hemodynamically unstable or with massive hemoptysis.[15] A TEVAR-only approach carries clear limitations such as potential for recurrence, inadequate sealing of the defect, and contamination of the new endograft. It is therefore intuitive that for secondary fistulas, an endovascular approach might serve only as a temporary measure, whereas a radical open procedure could be curative.[10] The surgical approach typically involves bronchial and pulmonary parenchymal repair, potentially with segmentectomy, explantation of the stent graft and all infected material, and aortic anatomical reconstruction. Materials that may be used are cryopreserved allografts, synthetic grafts, or biological xenografts.[15] The endograft is explanted and replaced (or sometimes left in situ), and coverage with an omental wrap is recommended.[15] The omental tissue, by providing a rich blood supply, enhances the immune response while draining the fluids produced by the inflammatory/infectious process. Hence, it facilitates the healing process and prevents recurrence of infections.[26,27] Alternatively, a pleural flap, the serratus muscle or even a pedicled segment of the diaphragm can be applied.[7,10,28,29] In particularly complex cases, an extra-anatomic bypass may be required.[30,31] In these cases, reinforcement of the aortic stump with bovine pericardium has also been reported.[32]

The radical approach (parenchymal repair in combination with stent graft removal and aortic reconstruction) seems to have better outcomes compared to the nonradical approaches. Czerny et al. reported an overall survival 63 vs 32% and 63 vs 21% at 1 and 2 years, respectively with radical or nonradical approach[2] and similar results have been reported by Chiesa and colleagues.[4] The effect of selection bias cannot be excluded, as patients undergoing radical surgery might present those patients in a better clinical condition and less severe fistulas. However, this underlines that mortality rates remain high even when the condition is treated in experienced centers.[4]

CONCLUSIONS

Post-TEVAR ABPF is associated with multiple risk factors, mainly patient-specific clinical and anatomical characteristics. Patient-related risk factors include chronic inflammatory conditions, underlying mediastinal oncologic pathologies, and infectious diseases. Anatomical features that may

lead to an ABPF are the presence of a large aortic aneurysm, pronounced aortic tortuosity, and an extensive periaortic hematoma. These risk factors need careful consideration during preoperative planning and may necessitate additional interventions, such as hematoma evacuation. Strategies to minimize the risk of ABPF formation can also be adopted in the planning phase. These factors include appropriate device oversizing, still guaranteeing adequate sealing zones, and limit the extent of the aortic coverage. Moreover, although it may seem like an obvious and basic consideration, maintaining sterility in the operating room is essential, as well as proper administration of antibiotic prophylaxis. Rigorous post-TEVAR follow-up and imaging surveillance are mandatory, not only for early detection and prompt management of ABPF but also for identifying endoleaks, that, if left untreated, may contribute to late fistulation. When pulmonary symptoms arise in patients with a history of a TEVAR, an ABPF should always be suspected. Radical surgical interventions have demonstrated superior outcomes compared to an endovascular approach. The latter could serve as a temporary measure to control bleeding, providing the time needed for patient optimization and optimal surgical planning, or definite solution in selective cases. Considering the extremely poor prognosis of untreated ABPF, conservative or palliative approaches may be the only reasonable treatment, when invasive and extensive procedures are contraindicated.

CREDIT AUTHORSHIP CONTRIBUTION STATEMENT

Gemmi Sufali: Conceptualization, Formal analysis, Investigation, Methodology, Writing — original draft, Writing — review & editing. **Constantijn E.V.B. Hazenberg:** Conceptualization, Investigation, Methodology, Supervision, Writing — original draft, Writing — review & editing. **Joost A. van Herwaarden:** Conceptualization, Investigation, Methodology, Supervision, Writing — review & editing. **Mauro Gargiulo:** Conceptualization, Methodology, Supervision, Writing — review & editing. **Gianluca Faggioli:** Conceptualization, Methodology, Supervision, Writing — review & editing. **Martin Teraa:** Conceptualization, Formal analysis, Investigation, Methodology, Supervision, Writing — original draft, Writing — review & editing.

None to be made.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.avsg.2024.12.061.

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