Mechanism and Management of Aorto-Esophageal Fistulation after Thoracic Endovascular Aortic Repair

Hazem El Beyrouti,[1,2] Hendrik Treede,[2] and Nancy Halloum,[2] Oldenburg and Mainz, Germany

An aorto-esophageal fistula (AOF) is a pathological communication between the thoracic aorta and the esophagus. It can induce life-threatening hematemesis, which is unique among the other types of gastrointestinal hemorrhage in that the vomiting is bright red and represents an arterial bleed. Nevertheless, it is notable that over 70% of cases are associated with thoracic aortic aneurysms, particularly as a postoperative complication following open surgery, and arguably more so following endovascular repair. As thoracic endovascular aortic repair becomes a more common practice, it is important to remain vigilant for an increase in the incidence of AOF. A thorough consideration of potential AOF etiologies is essential, as these must be substantiated by clinical, laboratory, and imaging tests. The available management options and the likelihood of success depend largely on the diagnosis made. The stability of the fistula wall is fundamental to determine treatment options and is particularly important in case of a potentially fatal AOF. The stability of the organ wall determines the complication risk, and therefore which treatment the patient is most likely to tolerate. A new AOF classification system will allow for better diagnosis and clearer assessment of treatment options.

INTRODUCTION

Since the successful initial implantation using thoracic endovascular aortic repair (TEVAR) by Dake in Stanford in 1992, it has been proven to be a safe and effective treatment for many aortic diseases according to the guidelines, especially thanks to continuing innovation and technological development.[1,2] As TEVAR has shown lower perioperative morbidity and mortality, it has become first-line therapy for many acute and chronic aortic diseases and is also used in some off-label conditions. However, long-term TEVAR durability still needs to be established and the increasing number of situations in which TEVAR is used logically increases the number and type of associated complications. The incidence of these reported complications also seems to be underestimated due to a paucity of long-term follow-up data, but secondary intervention rates are reported to range between 16 and 32% (or reoperation—in the sense of open conversion—with a range between 0.4 and 7.9%).[3] Many complications (like endoleak and migration) are well-known, but aorto-esophageal fistula (AOF) is perhaps less so and yet is both frequent and involves high morbidity and mortality, even when detected and treated.

[1]*Department of Vascular and Endovascular Surgery, Pius-Hospital Oldenburg, Medical Campus University of Oldenburg, Oldenburg, Germany.*

[2]*Department of Cardiac and Vascular Surgery, University Medical Centre Mainz, Johannes Gutenberg University, Mainz, Germany.*

Correspondence to: Hazem El Beyrouti, MD, PhD, MHBA, Director of the Department of Vascular and Endovascular Surgery, Pius-Hospital Oldenbrug, Medical Campus University of Oldenburg, Georgstraße 12, Oldenburg 26121, Germany; E-mail: hbeyrouti@gmail.com

Ann Vasc Surg 2025; 114: 358–366
https://doi.org/10.1016/j.avsg.2024.12.015

Manuscript received: October 14, 2024; manuscript accepted: December 13, 2024; published online: 20 December 2024

CHARACTERIZATION OF AORTIC FISTULA

An aortic fistula is of a pathological communication between the aortic lumen and an adjacent organ (or organs) and results in immediate catastrophic bleeding. The aorta can fistulize into the pulmonary artery with a frequency on autopsy reported at 4% of thoracic aneurysms and into the atrium of the heart (with an incidence of 1.6% of infective endocarditis). Aorto-caval fistulization has been reported with an incidence of 2–6% in ruptured aortic abdominal aneurysm, and aorto-enteric fistulization with an incidence of 0.5–2.3%.[4] However, the most common fistulizations after TEVAR are aorto-bronchial, aorto-pulmonary and aorto-esophageal (or a combination of these).[3]

An AOF is an abnormal connection between the thoracic aorta and the esophagus and can cause severe hemorrhage, which is different from other gastrointestinal bleeding because it is bright in color, indicating arterial bleeding.[5] The AOF most commonly occurs in the middle third of the esophagus, closer to the area of the change in aortic curvature.[4] It is a serious complication with the literature suggesting that 60% of patients with AOF may die within 6 months of symptom onset.[6] Over 70% of cases are associated with thoracic aortic aneurysms (TAAs), particularly as a postoperative complication following open surgery, and arguably more so following endovascular repair.

Classification of AOF

AOF is classified as primary or secondary. While the main factors of AOF include aneurysm (54.2%), foreign body (19.2%) and esophageal cancer (17%), other etiological factors could also be radiotherapy, infection, esophageal reflux, tuberculosis, congenital abnormalities, and traumatic injuries.[6–11] The etiological factors of secondary AOF include the following: complications after esophageal surgery (open with anastomotic complications and after stenting), open with anastomotic pseudoaneurysm and TEVAR.

Symptoms of AOF

In 1914, Chiari described the classic clinical presentation of AOF, which was midthoracic chest pain or dysphagia followed by relatively symptomless ''lethal periods'', sentinel arterial hemorrhage, and ultimately fatal exsanguination. Although this sequence was described after the intake of foreign bodies, the clinical presentation is relatively unchanged regardless of origin.[12,13] Hematemesis, chest pain, or dysphagia were observed in 63, 58, and 42% of subjects, respectively.[6]

In addition to the above symptoms, hypovolemic shock or systemic infection was observed in 60.9% and 21.7% of 72 patients with AOF post-TEVAR, respectively.[14] New-onset fever, dizziness, and syncope are also possible symptoms.

Bleeding may be from the vasa vasorum of the aorta or from the erosive arteries of the esophagus or directly from the aorta or perhaps a combination of all three.[6] The symptom-free interval between sentinel bleeding and large-scale exsanguination is unpredictable.[9] One report indicated that up to 80% of AOF cases resulted in severe bleeding within 1 week of the onset of sentinel bleeding.[15] However, other reports of major bleeding from AOFs post-TEVAR suggest a period between one and 16 months and one report even described an AOF case 6 years after TEVAR.[9,16]

Mechanism of Secondary AOF Post-TEVAR

The development of a secondary AOF after TEVAR may be due to one or a combination of the following etiological factors:

1. Direct erosion of the esophagus due to increased radial force of the rigid stents on the esophagus, resulting in a local inflammatory response and leading to formation of stable adhesions and tissue necrosis.[17]
2. Mechanical compression from hematomas or large aneurysms (or the force of stent expansion) can cause increased pressure in the mediastinum, leading to further expansion of the pressure necrosis due to occlusion of the side branches of the aorta feeding the esophagus.[18]
3. Esophageal ischemia due to inflammation in response to hematoma resorption (as suggested by Czerny).[19]
4. Inadequate perfusion of an esophageal segment as a result of stenting the arteries directly supplying the esophagus, which may lead to the formation of ischemic necrosis.[20,21]
5. TEVAR infection: graft infections involving the esophagus have been reported in 0.5–5% of cases and may be central to the development of AOF post-TEVAR.[22] The overall rates of infection post-TEVAR were reported by Smeds to be as high as 34% (urinary tract infection in 8%, groin infection in 7% and other unspecified infection in 19%).[23]
6. Endoleak: continuing perfusion of the aneurysm can lead to increased pressure on the

aortic wall, which leads to increased pressure on the esophagus, especially when there is no outlet for this endoleak flow (as in type Ia endoleaks).
7. Underlying aortic disease such as a penetrating atherosclerotic ulcer (PAU) or vasculitis.[5]
8. Emergent TEVAR: Chiesa et al. suggest that aortic injury often includes blood extravasation and periaortic hematoma, which increases the local inflammation response and compression of surrounding organs.[17]
9. Excessive stent-graft oversizing (>20%) can lead to long-term arterial wall deterioration and aortic aneurysm enlargement.
10. Proximal bare metal stents may increase the risk of penetrating aortic wall and adjacent organ injury.[24]
11. Reaction to the foreign body of the graft.[4]

Incidence of Secondary AOF

Whereas the incidence of aorto-bronchial and aorto-pulmonary fistula is 0.5–0.6%, AOF incidence after TEVAR is more common and between 1.5 and 2.1%.[3,19,24,25] However, this incidence is likely to be underestimated due to patients being lost to follow-up. In addition, it is important to consider that overall TEVAR numbers are increasing, as endovascular procedures replace open surgical operations. Indications for index TEVAR are predominantly TAA (33–77%), type B aortic dissection (10–50% in the acute phase; 11–20% in the chronic phase), and PAU (10%).[22,24]

A systematic review revealed that the mean age of patients affected with AF is 70 years, with a male-to-female ratio of 3:1.[26] However, in another paper the male/female ratio was 50%.[22]

A fistula is present in 91% of patients diagnosed with fever.[26] The mean interval from index TEVAR to diagnosis of fever is 195 days (approximately 10 months), varying between 3 and 18.1 months.[19,22,24]

Microorganism

Blood tests and cultures are the primary diagnostic steps and fundamental for accurate diagnosis. Positive blood cultures vary between 63 and 80% of patients with AF with pathogens including *Staphylococcus aureus*, *Streptococcus anginosus*, *Lactobacillus* species, *Citrobacter freundii*, and *Escherichia coli*.[23,24] Intraoperative findings revealed the presence of Gram-positive organisms in 22% of cases, Gram-negative organisms in 13%, fungi in 5%, and polymicrobial infections in 35% of cases: no growth was observed in 30% of cases.[23]

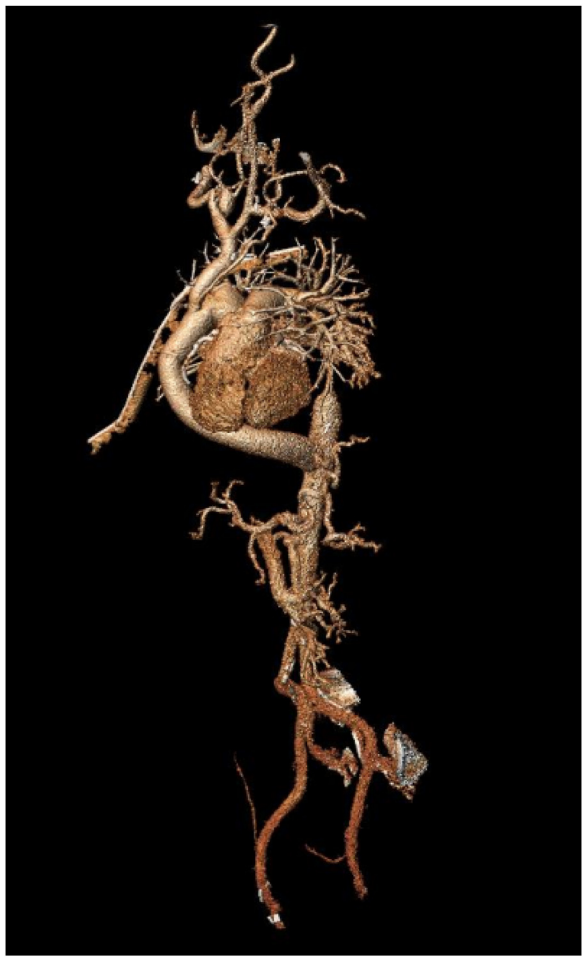


Fig. 1. Postop 3D rendering of a patient who underwent an extra-anatomical ascending to descending aortic bypass with a Dacron graft. ''View: LAO: 64; Cranial 7.'' Postop, postoperative; 3D, three-dimensional.

Microorganisms were identified in 43.2% of TEVAR patients with AOF and in 31.2% of patients with associated inflammation: the isolated pathogens were highly virulent, including methicillin-resistant *Staphylococcus aureus*, *Streptococcus spp.*, Gram-negative species such as *Pseudomonas* and *Klebsiella* and *Coxiella burnetti* in some cases.[14,27]

Experience

Since 2014, 5 cases of AOF post-TEVAR have been observed at this center (2 sites): the initial disease was PAU in 2 patients (50%) and thoracoabdominal aneurysm in the other 2 (50%). One patient underwent an extra-anatomic ascending to descending aortic bypass with a Dacron graft (Fig. 1), while the second patient underwent in situ reconstruction

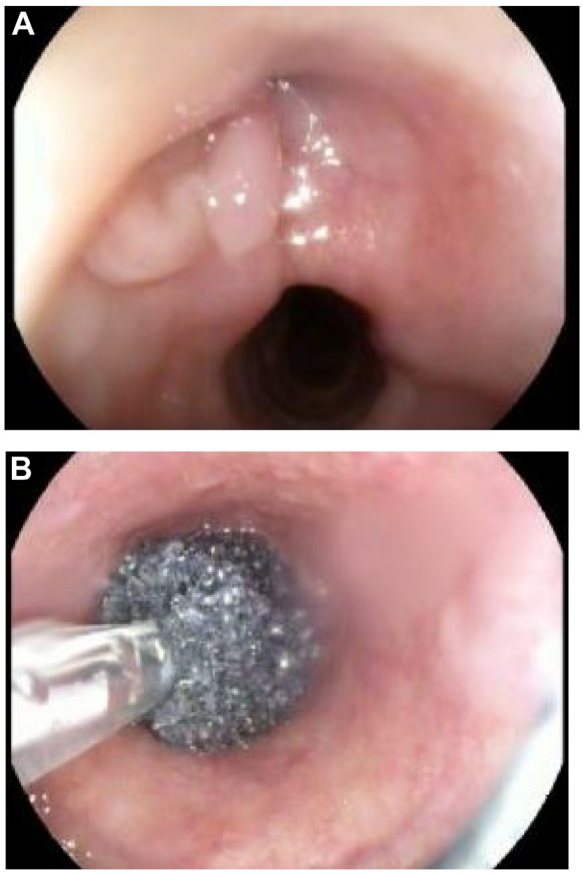


Fig. 2. (A) Endoscopy showing the aorto-esophageal fistula (AOF). **(B)** A vacuum sponge is placed in the esophagus in front of the fistula.

with a pericardial tube, accompanied by subtotal esophagostomy and gastrostomy. One patient rejected open surgery and was treated conservatively, including endoscopic vacuum therapy (Fig. 2), application of fibrin glue, and antibiotics. In the context of multimorbid diseases, the other 2 patients were treated with antibiotics in a palliative approach. All patients died within 1 year (80% 1-year mortality), and one patient who underwent conservative endoscopic closure with fibrin glue died after 31 months.

Diagnosis

Computed tomography (CT) allows for rapid acquisition of images with high spatial resolution and should be the basis for diagnosis, allowing identification, for example, of air bubbles, periaortic fluid accumulation, esophageal wall thickening or exsanguination of intravenous contrast into the gastrointestinal tract, accompanied by a nipple-like outpouching from the aorta into the esophagus (or vice versa).[4]

Volume-rendered three-dimensional images can be used to plan surgical and endovascular repair. Conventional angiography can offer a superior representation of arterial anatomy. Magnetic resonance angiography is useful especially in patients with a renal insufficiency.[28]

In cases where CT angiographic findings are inconclusive in differentiating between postoperative changes and infection, scintigraphy with technetium 99m hexamethylpropyleneamine oxime-labelled white blood cells (WBCs) or indium 111 (111In) oxime-labelled WBCs is recommended and, in select cases, fluorine-18-fluorodeoxyglucose (FDG).[4,29]

FDG positron emission tomography (PET) or CT is a higher resolution imaging model that has gained considerable recognition in recent years: in the event of acute fever and any other type of infection, the sensitivity and specificity are 95−80%.[30] It is the optimal imaging modality for patients who underwent surgery at least 3 months prior to antibiotic administration because postoperative inflammatory changes can result in false positives, while antibiotic use causes false negatives.[4,31]

Endoscopy is the most sensitive and specific diagnostic tool for AOF, although it is associated with a high incidence of complications, particularly in cases of clot dislocation, which can result in fatal bleeding. Conversely, endoscopy can also be employed as a means of targeted therapy.[5]

Therapeutical State-of-the-Art

Open surgical repair of AOF post-TEVAR involves three fundamental steps: stopping massive exsanguination, radical debridement, and esophageal reconstruction. This procedure may be performed as an in situ or extra-anatomic reconstruction. The aorta can be reconstructed with a wide variety of grafts, including cryopreserved homografts, biological (such as pericardial patches) or biosynthetic materials, silver prosthesis, and antibiotic-impregnated grafts: the choice should be determined after an assessment of the associated risks (rupture, reinfection, long-term durability, aneurysmal formation, availability, diameter mismatches).[32]

The aortic reconstruction may be covered with omental flaps to enhance resistance against reinfection (given the high vascularization observed by the omentum). A left-side posterolateral thoracotomy is typically performed in cases where the proximal landing zone of TEVAR is in the aortic arch. In such instances, a median sternotomy combined with an extended left anterolateral incision is necessary. It is advisable to use a heart-lung machine to ensure adequate perfusion of the supra-aortic and

visceral arteries via an antegrade and retrograde perfusion with a left heart bypass or complete cardiopulmonary bypass with deep or better moderate hypothermia. (Some surgeons prefer the clamshell incision.)

Proximity of the graft to the infected area is associated with an elevated risk of infection, which is why many surgeons opt to perform an extra-anatomic bypass: ascending to descending aortic bypass was initially described by Yonago et al. in 1969 and has since been shown to be a valuable technique in certain cases.[33]

Surgical reconstruction may be classified into 2 categories: partial, which encompasses techniques such as direct closure of aortic defects via suture or patch reconstruction, and complete reconstruction, which involves total TEVAR removal and replacement of the infected aorta. The infected area and all surrounding tissue should be subjected to aggressive debridement to reduce the risk of reinfection.

Finally, esophageal reconstruction is divided into partial and total procedures. In partial procedures, the esophagus is repaired with a direct stitch or patch. In total procedures, the esophagus is removed and a gastrostomy or jejunostomy is performed. Some surgeons suggest a cervical esophagostomy with gastrostomy.

Endovascular repair may be preferred for patients with multiple comorbidities and at a high risk for open surgical repair, especially those who are unstable; a re-TEVAR is a procedure that is performed by a significant number of surgeons worldwide. It should be noted that in this case, the infected endograft, aorta and esophagus will remain in situ and so lifelong course of antibiotics is recommended.

In some cases, a bridging re-TEVAR may be a good way to achieve stabilization prior to the next radical procedure. Some proponents of this approach recommend that the interval between procedures should not exceed 7 days. In certain instances, an esophageal stent may be placed as a standalone intervention or in conjunction with TEVAR.

Conservative management may also be an option and can entail endoscopic therapy using vacuum therapy and the application of fibrin glue 5 or endoscopic clips.[34] The administration of antibiotics represents a cornerstone of modern medicine and a broad-spectrum antibiotic should be administered, based on the results of the blood culture, with or without drainage.

DISCUSSION

In 1818, French surgeon Dubrueil reported a case of AOF in a soldier aged 28 years who had swallowed part of a bone, suffered chest pain and diarrhea several hours after intake and an exacerbating hemorrhage 5 days later: the autopsy revealed a fistula between the esophagus and the thoracic aorta.[35]

Funk was the first to report a TAA rupture in 1918 and, since then, between 9.4% and 20% of TAA ruptures are in the context of AOF.[36] Although there was a significant incidence of AOF associated with TAA, the diagnosis was made for the first time before death in 1948 where conservative therapy was attempted.[37] In 1975 and 1981, surgical repairs were attempted, but both patients died.[38,39] The first successful repair was reported in 1983 at the Methodist Hospital (Houston, Texas, US) in 2 patients.[40] There are now some large series and systematic reviews (and several case reports) that describe AOF post-TEVAR.[17,19,24,26,41,42]

Despite some controversy, conservative treatment of AOF post-TEVAR is associated with high mortality: Smeds et al., for example, treated conservatively in 5/12 patients and survival rate was only 20% after 1 year.[43,44] In large series, the conservative approach had closer to 100% mortality after 1 year.[17,23–25,45]

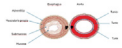
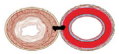
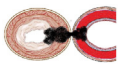
Likewise, re-TEVAR is associated with poor survival. Okita et al. performed 2/11 patients and Chiesa et al. 2/19 patients with a short-term mortality of 100%.[17,46] Czerny et al. performed esophagectomy in 7/36, Luehr 1/8, Chiesa 4/19 (one patient had concomitant bronchial repair for concomitant ABF) with a survival of 43%, 0% and 25% respectively at 12 months.[17,19,24]

Esophageal stenting was performed in 6/36 patients by Czerny et al. with a survival rate of 17% and 2/19 patients by Chiesa et al. with a survival rate of 50% at 5 months.[17,19] A combination of aortic (in situ or extra-anatomic) and esophagectomy was performed in 13/36 patients by Czerny et al., Luehr et al. 6/8 patients, Okita 9/11 patients, Kawamoto 10/10 patients, Chiesa 3/19 patients and Kotelis et al. 4/4 patients with survival rates of 46%, 44%, 68.5%, 33% and 50% in 12 months follow-up.[17,19,24,42,46,47] In our series, mortality was 100% in both the conservative and surgical groups (esophagectomy with in situ or extra-anatomic aortic bypass).

However, it is also very important to mention that secondary AOF has a higher mortality rate than primary (78% vs. 61%).[17]

Lack of standardization in the description of the invasiveness of the AOF and the corresponding treatment strategies across the majority of small and large series, including case reports, complicates interpretation of the published results and impedes a full understanding of the pathology, evaluation

Table I. A proposed classification of aorto-esophageal fistula with management strategies

Classification	Key issue	Fistula	Wall stability	Organ involvement	Tissue involvement	Surrounding tissue	Diagnostic modality	Findings	Management
Type I 	Erosion I (superficial)	No	Stable	A: aorta B: esophagus C: both	Light inflammation. The walls appear smooth and intact.	Limited: minor inflammation or edema	PET-CT (positive for signs of inflammation)	Increased metabolic activity in the aortic or esophageal walls without structural damage on CT.	• Conservative therapy and close monitoring
Type II 	Erosion II (deep)	No	Stable	A: aort B: esophagus C: both	Deep erosion involving >2 layers of one or both organs. No connection	Mild edema or inflammation, no significant structural damage	CTA ± PET-CT	Wall thickening on CT, with mild surrounding tissue edema or PET-CT positivity for localized infection/ inflammation.	• Conservative therapy • Minimally Invasive Intervention: including minimal esophageal repair (esophageal stenting) and/or endo-vascular surgery (TEVAR)
Type III 	Penetration I (superficial)	Yes	Stable	A: aorta B: esophagus C: both	Complete fistula with superficial penetration. Intact esophageal mucosa and aortic intima.	Local inflammation. There may be early signs of infection, including mediastinitis. No necrosis	CTA + Endoscopy	Contrast extravasation visible in CTA. Endoscopy confirms mucosal disruption or fistula. Surrounding tissue shows mild changes.	• Urgent minimally invasive intervention • Interdisciplinary Surgery Plan
Type IV 	Penetration II (deep)	Yes	Unstable	A: aorta B: esophagu C: both	Complete fistula and deep penetration that affects all the layers of at least organ. Collapse or rupture of one and/or both organs with jagged or discoloured areas indicating tissue breakdown.	Variable: acute cases minimal damage; chronic cases may have necrosis, abscess, mediastinitis	CTA + endoscopy	Visible fistula with structural collapse or extensive necrosis. Mediastinal abscess, necrosis, or infection.	• Hemodynamic stabilization • Intensive care support • Emergent minimally invasive intervention following Interdisci-plinary Surgery Plan

Interdisciplinary Surgery Plan: A comprehensive interdisciplinary plan (conservative therapy with follow-up monitoring or surgical reconstruction) should be developed in collaboration with specialists from gastroenterology, thoracic or general surgery (esophageal surgery specialists), and cardiac and vascular surgery (aortic specialists) in order to facilitate a surgical approach that is effective and safe.

CTA, computed tomography angiography; PET, positron emission tomography; TEVAR, thoracic endovascular aortic repair.

of treatment options, and conveying risks and benefits adequately to patients.

The stability of the fistula wall is fundamental to determine treatment options and is particularly important in case of a potentially fatal AOF. The stability of the organ wall determines the complication risk, and therefore which treatment the patient is most likely to tolerate. A stable wall means that—even in the presence of fistulization—the tissues of the diseased organs are normal with no (or minor) necrosis or defects in circularity and supports a more conservative approach.

An unstable wall points to significant deficiency in the tissue structural integrity which characteristically arise from inflammatory processes, inflammatory diseases, death of tissue or other degenerative changes. Sudden wall ruptures with serious hemorrhage or sudden wall deterioration are more common and usually calls for urgent intervention such as radical surgical intervention.

With complete wall penetration, a bridging TEVAR as a step to a lifesaving operation to avoid massive blood loss will allow for the patient to be stabilized until a radical intervention can be planned with the aortic team.

Of course, earlier EAF diagnosis can lead to more favourable results and in effect prevent death and greater awareness and knowledge of the condition can contribute to reducing risk. Complications in the perioperative period after TEVAR, especially all postsurgical endoleak or respiratory failure, guidelines restricting these patients' activities suggest that careful monitoring may improve AOF.[17]

Discussing AOF management is challenging because of the lack of standardization when describing the degree of damage. The majority of reports in the literature do not describe the correspondence between surgical approaches and the varying degrees of erosion or penetration.

For this reason, I propose a classification (Table I) to address this significant issue which incorporates patient conditions, the type and invasiveness or penetration of the AOF, and management options. What is clear is that surgical excision is the gold standard to address foreign body infection. However, an interdisciplinary group will be able to weigh this option against patient condition and ability to tolerate invasive surgery. What options are best—TEVAR, surgical replacement of the infected segment or esophageal stenting—may well emerge more clearly in future reports if this novel classification proves useful. Optimal management will involve a combination of histopathological and imaging assessments as well as a multidisciplinary team. PET-CT and CT, for example, will allow an assessment of tissue surrounding the AOF and indirectly identify different stages of infection, erosion, and fistulization. The proposed classification goes from Type I to Type IV depending on key factors such as erosion/penetration and wall instability. Types are further broken down according to organ involvement (A, aorta; B, esophagus; C, both). Finally, management options are described according to the classification staging.

Aorto-esophageal fistulization is a not uncommon complication after TEVAR but with often catastrophic consequences. The stability of the fistula wall is fundamental to determine treatment options and is particularly important in case of a potentially fatal aorto-esophageal fistulization. The stability of the organ wall determines the complication risk, and therefore which treatment the patient is most likely to tolerate. A new classification system for aorto-esophageal fistulization will allow for better diagnosis and clearer assessment of treatment options.

CREDIT AUTHORSHIP CONTRIBUTION STATEMENT

Hazem El Beyrouti: Writing – review & editing, Writing – original draft, Supervision, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Hendrik Treede:** Writing – review & editing, Writing – original draft. **Nancy Halloum:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization.

REFERENCES

1. Dake MD, Miller DC, Semba CP, et al. Transluminal placement of endovascular stent-grafts for the treatment of descending thoracic aortic aneurysms. N Engl J Med 1994;331:1729–34.
2. El Beyrouti H, Lescan M, Doemland M, et al. Early results of a low-profile stent-graft for thoracic endovascular aortic repair. PLoS One 2020;15:e0240560.
3. Coselli JS, Spiliotopoulos K, Preventza O, et al. Open aortic surgery after thoracic endovascular aortic repair. Gen Thorac Cardiovasc Surg 2016;64:441–9.
4. Gulati A, Kapoor H, Donuru A, et al. Aortic fistulas: pathophysiologic features, imaging findings, and diagnostic pitfalls. Radiographics 2021;41:1335–51.
5. Halloum N, Abdalhafez M, Thieringer F, et al. Conservative endoscopic closure with fibrin glue of an aorto-esophageal fistula secondary to endovascular repair of a contained penetrating atherosclerotic ulcer rupture. J Vasc Surg Cases Innov Tech 2023;9:101354.

6. Hollander JE, Quick G. Aortoesophageal fistula: a comprehensive review of the literature. Am J Med 1991;91:279–87.
7. Zhong XQ, Li GX. Successful management of life-threatening aortoesophageal fistula: a case report and review of the literature. World J Clin Cases 2022;10:3814–21.
8. Podbielski FJ, Rodriguez HE, Zhu RY, et al. Aortoesophageal fistula secondary to reflux esophagitis. Dig Surg 2007;24:66–7.
9. Uno K, Koike T, Takahashi S, et al. Management of aorto-esophageal fistula secondary after thoracic endovascular aortic repair: a review of literature. Clin J Gastroenterol 2017;10:393–402.
10. Lee OJ, Kim SH. Aortoesophageal fistula associated with tuberculous mediastinitis, mimicking esophageal Dieulafoy's disease. J Korean Med Sci 2002;17:266–9.
11. Angelini A, Dimopoulos K, Frescura C, et al. Fatal aortoesophageal fistula in two cases of tight vascular ring. Cardiol Young 2002;12:172–6.
12. Chiari H. Ueber Fremdkorpeverletzung des oesophagus mit aortenperforation. Berlin Klin Wochenschr 1914;51:7–9.
13. Funk EH. Aortic aneurysm with esophageal rupture. Med Clin North Am 1918;2:795–802.
14. Canaud L, Chassin-Trubert L, Abouliatim I, et al. Total arch thoracic endovascular aortic repair using double fenestrated physician-modified stent-grafts: 100 patients. J Endovasc Ther 2024;31:89–97.
15. Steffes BC, O'Leary JP. Primary aortoduodenal fistula: a case report and review of the literature. Am Surg 1980;46:121–9.
16. Kouritas VK, Dedeilias P, Sotiriou K, et al. Delayed presentation of aortoesophageal fistula after endovascular repair. Asian Cardiovasc Thorac Ann 2016;24:51–3.
17. Chiesa R, Melissano G, Marone EM, et al. Aorto-oesophageal and aortobronchial fistulae following thoracic endovascular aortic repair: a national survey. Eur J Vasc Endovasc Surg 2010;39:273–9.
18. Girdauskas E, Falk V, Kuntze T, et al. Secondary surgical procedures after endovascular stent grafting of the thoracic aorta: successful approaches to a challenging clinical problem. J Thorac Cardiovasc Surg 2008;136:1289–94.
19. Czerny M, Eggebrecht H, Sodeck G, et al. New insights regarding the incidence, presentation and treatment options of aorto-oesophageal fistulation after thoracic endovascular aortic repair: the European Registry of Endovascular Aortic Repair Complications. Eur J Cardio Thorac Surg 2014;45:452–7.
20. Marone EM, Esposito G, Kahlberg A, et al. Surgical treatment of tracheoinnominate fistula after stent-graft implantation. J Thorac Cardiovasc Surg 2007;133:1641–3.
21. VonFricken K, Karamanoukian HL, Ricci M, et al. Aortobronchial fistula after endovascular stent graft repair of the thoracic aorta. Ann Thorac Surg 2000;70:1407–9.
22. Eggebrecht H, Mehta RH, Dechene A, et al. Aortoesophageal fistula after thoracic aortic stent-graft placement: a rare but catastrophic complication of a novel emerging technique. JACC Cardiovasc Interv 2009;2:570–6.
23. Smeds MR, Duncan AA, Harlander-Locke MP, et al. Treatment and outcomes of aortic endograft infection. J Vasc Surg 2016;63:332–40.
24. Luehr M, Etz CD, Nozdrzykowski M, et al. Emergency open surgery for aorto-oesophageal and aorto-bronchial fistulae after thoracic endovascular aortic repair: a single-centre experience. Eur J Cardio Thorac Surg 2015;47:374–83.
25. Czerny M, Funovics M, Ehrlich M, et al. Risk factors of mortality in different age groups after thoracic endovascular aortic repair. Ann Thorac Surg 2010;90:534–8.
26. Kahlberg A, Grandi A, Loschi D, et al. A systematic review of infected descending thoracic aortic grafts and endografts. J Vasc Surg 2019;69:1941–1951.e1.
27. Okwara CJ, Petrasek J, Gibson M, et al. Secondary aortoesophageal fistula associated with aneurysmal graft infection by Coxiella burnetii. ACG Case Rep J 2016;3:169–71.
28. Wortman JR, Landman W, Fulwadhva UP, et al. CT angiography for acute gastrointestinal bleeding: what the radiologist needs to know. Br J Radiol 2017;90:20170076.
29. Wilson JE, Galiñanes EL, Hu P, et al. Routine revascularization is unnecessary in the majority of patients requiring zone II coverage during thoracic endovascular aortic repair: a longitudinal outcomes study using United States Medicare population data. Vascular 2014;22:239–45.
30. Reinders Folmer EI, Von Meijenfeldt GCI, Van der Laan MJ, et al. Diagnostic imaging in vascular graft infection: a systematic review and meta-analysis. Eur J Vasc Endovasc Surg 2018;56:719–29.
31. Treglia G, Pascale M, Lazzeri E, et al. Diagnostic performance of 18F-FDG PET/CT in patients with spinal infection: a systematic review and a bivariate meta-analysis. Eur J Nucl Med Mol Imaging 2020;47:1287–301.
32. El Beyrouti H, Izzat MB, Kornberger A, Halloum N, Dohle K, Trinh TT, Vahl CF, Dorweiler B. Ovine biosynthetic grafts for aortoiliac reconstructions in nonsterile operative fields. Thoracic Cardiovasc Surg 2022;70:645–51.
33. Yonago RH, Iben AB, Mark JBD. Aortic bypass in the management of aortoesophageal fistula. Ann Thorac Surg 1969;7:235–7.
34. A single-center case series of endoscopically treated aorto-gastrointestinal fistula after endovascular aortic repair: surgery is still the only valid solution | visceral medicine | Karger Publishers [Internet]. Available at: https://karger.com/vis/article/37/2/134/310446/A-Single-Center-Case-Series-of-Endoscopically. Accessed September 12, 2024.
35. Dubrueil Observations sur la perforation de l'oesophage et de l'aorte thoracique par une potion d'os avale: Avec der reflexions. J Univ Sci Med 1818;9:357–63.
36. Hooper WL. Haematemesis and melaena due to rupture of a saccular aneurysm of the aorta into the oesophagus. Postgrad Med J 1962;38:297–301.
37. Baer S, Loewenberg SA. Aortic aneurysms simulating organic disease of the gastro-intestinal tract. Gastroenterology 1948;10:617–25.
38. Baron RL, Koehler RE, Gutierrez FR, et al. Clinical and radiographic manifestations of aortoesophageal fistulas. Radiology 1981;141:599–605.
39. Monro JL, Skidmore FD, Sbokos CG, et al. Intraoesophageal rupture of a thoracic aortic aneurysm. J Cardiovasc Surg 1975;16:302–7.
40. Snyder DM, Crawford ES. Successful treatment of primary aorta-esophageal fistula resulting from aortic aneurysm. J Thorac Cardiovasc Surg 1983;85:457–63.
41. Okita Y, Miki S, Ueda Y, et al. Mitral valve replacement with a collar-reinforced prosthetic valve for disrupted mitral annulus. Ann Thorac Surg 1995;59:187–9.
42. Kawamoto S, Sato M, Motoyoshi N, et al. Outcomes of a staged surgical treatment strategy for aortoesophageal fistula. Gen Thorac Cardiovasc Surg 2015;63:147–52.
43. Conservative treatment of an aortoesophagial fistula after endovascular stent grafting for a thoracic aortic aneurysm - PMC [Internet]. Available at: https://www.ncbi.nlm.nih.

gov/pmc/articles/PMC3539520/. Accessed September 12, 2024.
44. Numan F, Gulsen F, Cantasdemir M, et al. Percutaneous treatment of an infected aneurysmal sac secondary to aortoesophageal fistula with a history of stent-graft treatment for thoracic aortic aneurysm. Cardiovasc Intervent Radiol 2012;35:690–4.
45. Zhou H, Tang S, Zhong C. 348. Management of aortoesophageal fifistula secondary after thoracic endovascular aortic repair. Dis Esophagus 2024;37(Supplement_1): doae057.107.
46. Okita Y, Yamanaka K, Okada K, et al. Strategies for the treatment of aorto-oesophageal fistula. Eur J Cardio Thorac Surg 2014;46:894–900.
47. Kotelis D, Bianchini C, Kovacs B, et al. Early experience with automatic pressure-controlled cerebrospinal fluid drainage during thoracic endovascular aortic repair. J Endovasc Ther 2015;22:368–72.