### CASE REPORT

# Atrioesophageal fistula: Imaging for a definitive diagnosis

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#### ABSTRACT

**Aim:** Atrial fibrillation is a common arrhythmia, with a prevalence of 37.574 million cases worldwide. Atrioesophageal fistula is a rare but potentially fatal complication of ablation of atrial fibrillation developing up to 60-days post-ablation with a prevalence of 0.07%–0.25%, and a 63% mortality. While chest Catscan (CT) s are abnormal in most of these patients (76%–93%), definitive atrioesophageal fistula is noted in only 23%–35% of cases, complicating pre-intervention diagnosis. Surgical repair of the left atrial and primary esophageal defect is essential for these patients, resulting in reduced mortality compared to nonsurgical management (33.71% vs. 94.19%).

**Methods:** Our case series and comprehensive review of the literature highlights the diagnostic and treatment challenges of atrioesophageal fistula.

**Results/Conclusions:** For symptomatic patients within 60-days post-ablation, IV contrast-enhanced helical chest CT with thin section collimation as initial imaging and axial reconstruction utilizing a 1 mm-mm detector with sagittal and coronal reformats should be completed to allow for optimal identification of abnormalities consistent with atrioesophageal fistula. Patients with neurologic symptoms with the presence of pneumocephalus, infarcts involving one or more vascular territories, or diffuse air emboli that are highly suggestive of atrioesophageal fistula, warrant a chest CT with IV contrast to evaluate the presence of AEF.

An initial unremarkable chest CT does not rule out atrioesophageal fistula and repeat chest CT with IV contrast within 1–3 days increases the likelihood of a definitive AEF diagnosis. Surgery is the only recommended management in patients with atrioesophageal fistula who are clinically stable enough to endure the procedures.

#### Introduction

Atrial fibrillation (AF) is a common arrhythmia with an estimated prevalence of 37.574 million cases worldwide [1]. The standard treatment method for symptomatic, refractory, or medically intolerant AF is catheter ablation (CA) [2]. Atrial-esophageal fistula (AEF) is a rare but potentially fatal complication of CA that can develop immediately after and up to 60 days post-CA (median 28 days) [3]. Prevalence of AEF ranges from 0.07% to 0.25%, [2,4] with mortality as high as 63% [3]. Thus, early identification and treatment are vital in improving outcomes for patients with AEF.

Symptoms of AEF are non-specific, with fever (53.73%), chest pain (29.10%), focal neurologic deficits (26.87%), and altered mental status (17.91%) the most common [3], often causing the initiation of cardiac or stroke protocols [chest/head Catscan (CT)] [5]. While initial chest CT findings are abnormal in most of these patients (76%–93%) [3,5–7], definitive AEF is noted in only 23%–35% of

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cases [fistulous tract, esophageal perforation, free air in the mediastinum, left atrium (LA), or wall of the LA] illustrating the difficulty in accurate pre-intervention diagnosis [3,5]. Head CT is only abnormal in 40%–50% of patients [3,6] noting diffuse air emboli and multi-territory infarcts [6,8].

While transthoracic echocardiogram (TTE) or transesophageal echocardiogram (TEE) may typically follow a non-diagnostic CT for evaluation of AEF, both modalities have limited use in safely obtaining a definitive diagnosis. Specifically, TTE has a high false-negative rate, with abnormalities noted in less than half of AEF cases (44%) [5] and TEE may result in clinical deterioration due to the probe dislodging septic emboli, causing brain hemorrhage and a bilateral frontal infarct [9].

Urgent surgical repair of the left atrial and primary esophageal defect is the gold standard of management for AEF [5,10,11], resulting in reduced mortality when compared to nonsurgical management (33.71% vs. 94.19%) [3]. Thus, early and accurate diagnosis to facilitate the surgical repair and improve the survival of AEF patients is essential [3,5].

Here we present a single institution case series and a comprehensive review of the literature to highlight the diagnostic and treatment challenges regarding post-CA AEF to evaluate strategies to obtain a definitive diagnosis that can result in optimal patient care and outcomes.

## Case 1

A 57-year-old male with a history of chronic heart failure and chronic kidney disease presented for CA for AF. The patient underwent percutaneous radiofrequency ablation (RFA) of the LA with the use of esophageal temperature monitoring to limit output along the posterior wall to 30 Watts. -Postablation day (PAD) 1, the patient reported pleuritic chest pain; an echocardiogram (ECG) showed no signs of pericardial effusion. The patient was discharged with medications for presumed pericarditis. On PAD 28, the patient presented to the emergency department (ED) with transient numbress of the left upper extremity along with lower back pain, confusion, and chest pain. An ECG noted a new T-wave inversion in leads V2, V3, V4, and mild ST elevation in aVR. A chest CT without contrast showed loculated gas present within the posterior aspect of the LA along with particulate matter. The patient underwent a chest CT with IV contrast which showed small gas containing focus within

the posterior aspect of the LA and a small amount of adjacent air at the cardiac margin (Fig. 1a and b).

Due to concern for AEF, an emergent right thoracotomy, esophagectomy, and left atrial perforation repair were performed. Post-operative day (POD) 1, the patient reported weakness of the left upper extremity suggestive of a stroke; a head CT head contrast noted a wedge-shaped area of hypoattenuation in the right posterior cerebellum. Magnetic resonance imaging (MRI) imaging showed multifocal infarcts in the right cerebellar hemisphere (Fig. 1c and d). The patient was extubated POD 3, the chest tube was removed POD 7 and discharged POD 12. After nine months, a gastrocutaneous fistula and colonic reconstruction of the esophagus with esophageal/colon and gastro/colon anastomoses were completed. Postoperative persistent acidosis was noted; acute hepatic failure progressed to multisystem organ failure, requiring the patient to be placed on multiple vasopressors. The patient went into pulseless electrical activity POD 3 and cardiopulmonary resuscitation (CPR) was initiated. The patient's family elected to cease resuscitative efforts and the patient expired.

## Case 2

A 57-year-old male with a past medical history of hypertension, obstructive sleep apnea, and CA for AF, which utilized percutaneous RFA for AF with the use of esophageal temperature monitoring, developed a significant pericardial effusion requiring pericardial drain placement PAD 13 at an outside institution. The patient presented to our ED PAD 31 with right-sided weakness and facial droop suggestive of stroke. A CT angiogram of the neck and head with and without contrast was completed; no abnormalities were identified. A chest CT with IV contrast revealed gas between the LA and esophagus suggesting an AEF (Fig. 2a and b). The patient underwent cardiopulmonary bypass and patch closure of the left atrial fistula. Due to worsening hypotension despite receiving multiple blood products in the intensive care unit (ICU), a mediastinal exploration and insertion of an intra-aortic balloon pump were completed POD 1. Repair of the esophageal fistula with an intercostal muscle flap placed over the site of the initial esophageal injury was completed POD2; due to elevated creatinine, dialysis therapy was initiated. Due to altered mental status POD 6, A head CT was completed and revealed new bifrontal edema. (Fig. 2c). The patient was maintained on ventilator therapy and pressors for hemodynamic



**Figure 1.** Intravenous contrast enhanced chest CT. (a) Axial and (b) sagittal views demonstrating air within the LA (white arrow). Brain MRI diffusion weighted axial imaging. (c) Restricted diffusion (white arrows) in the supratentorial and (d) infratentorial (right cerebellar hemisphere) brain indicative of multifocal infarcts.



**Figure 2.** Intravenous contrast enhanced chest CT. (a) Axial and (b) coronal images demonstrating air (white arrows) extending from the esophagus into the LA. Non-contrast head CT. (c) Axial view with infarcts of the frontal (white arrow) and parietal lobes (broken white arrow) bilaterally.

support and continued on a multi-antibiotic regimen for suspected sepsis; on POD 19, attempts to wean the patient off the ventilator failed, and life support was withdrawn.

### Case 3

A 77-year-old man with a history of hypertension underwent percutaneous RFA for AF without the use of esophageal temperature monitoring, presented to the ED on PAD 21 with reported left arm weakness, chest pain, and altered mental status. On arrival, the patient was unresponsive and vomited, requiring rapid sequence intubation. The patient developed ventricular tachycardia requiring synchronized cardioversion with successful restoration to normal sinus rhythm. Due to concern for a stroke, a head CT without contrast was obtained and showed evidence of low attenuation in the cerebral sulci thought to reflect fat or pneumocephalus. A head CT angiogram was suspicious for intravascular air over the sulci of the left frontal lobe (Fig. 3a and b). TTE revealed a reduced EF of 45%–50% compared to his previous of 55%–60%. A brain MRI demonstrated an atypical, restricted diffusion pattern affecting the gyri

with patches of a signal abnormality in both cerebral hemispheres. Metabolic encephalopathy versus infarct was suspected. On PAD 23, the patient could follow simple commands and was successfully extubated. Due to persistent sepsis, a chest CT with and without contrast (intravenous contrast and oral contrast) was completed PAD 26; air and oral contrast adjacent to the left pulmonary vein consistent with esophageal perforation was identified (Fig. 3c and d). Surgical closure of the atrial esophageal fistula was completed on PAD 27 with an intercostal muscle flap. A total esophagectomy was performed with the creation of a cervical esophagostomy and percutaneous endoscopic gastrostomy (PEG) tube placement. The patient was extubated on POD 2 in the ICU; however, on POD 8, he developed hypotension and altered mental status requiring intubation and multiple pressors. The patient developed bradycardia and became pulseless, requiring CPR. Return of spontaneous circulation was obtained multiple times during CPR but subsequently lost. Bedside ultrasound showed pericardial tamponade, and pericardiocentesis was performed. Despite multiple interventions, the patient expired POD 9.

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**Figure 3.** CT angiogram brain with intravenous contrast. (a) Axial and (b) coronal images demonstrating intravascular air (white arrow) within the left frontal lobe. The coronal image is demonstrated in a modified window/level setting to increase the conspicuity of air. Chest CT with oral contrast only. (c) Axial and (d) coronal images demonstrating air and oral contrast (white arrow) within the LA adjacent to the left superior pulmonary vein.

#### Case 4

A 46-year-old female with a history of RFA for AF with esophageal temperature monitoring, presented with abdominal pain, fever, nausea, and vomiting PAD 15 to an outside hospital; a chest CT revealed gas posterior to the heart and anterior to the esophagus. During imaging, the patient developed hematemesis, required intubation, and an emergent esophagogastroduodenoscopy (EGD), which noted lesions of the mid-esophagus thought to resemble perforation; the patient was transferred to our institution. She arrived intubated and hypotensive, requiring pressor stabilization. A chest CT angiogram with and without IV contrast demonstrated extraluminal gas adjacent to the right inferior pulmonary vein and contrast extravasation towards the esophagus (Fig. 4a and b). A right thoracotomy and repair of an AEF were initiated; however, a fistula from the esophagus to the right inferior pulmonary vein requiring resection of the inferior pulmonary vein and right lower lobectomy was identified intra-operatively. A cardiopulmonary bypass was initiated, and the atrial defect was repaired with a patch graft. The esophagus was dissected off the pericardium,

and intercostal muscle was placed over the esophageal repair site. On POD 2, the patient had left-sided hemiparesis; a head CT without contrast showed subarachnoid blood in the right frontal region with possible infarction in the right parieto-occipital junction (Fig. 4c). A brain MRI brain confirmed infarcts in the supratentorial and infratentorial regions (Fig. 4d and e); a tracheostomy was placed. She continued to have tachycardia and respiratory insufficiency, and on POD 9, chest CT with and without IV contrast still noted a distal esophageal fistula within the right lower thorax/mediastinum with leakage of esophageal administered contrast. In the operating room (OR), an esophageal perforation was discovered underneath the intercostal muscle flap. The distal esophagus was resected during the esophagectomy, and the patient underwent the creation of a cervical esophagostomy; gastrostomy and feeding jejunostomy tubes were placed. The patient tolerated the procedure well and remained intubated with chest tubes in the ICU. She was started on tube feeds, and it was noted that she had high output from her chest tube secondary to a chyle leak. On POD 31, the patient was discharged to a long-term



**Figure 4.** Intravenous contrast enhanced chest CT. (a) Axial image demonstrating intravenous contrast extravasation into the esophagus (white arrow) (b) pneumomediastinum (white arrow 2) adjacent to the right inferior pulmonary vein. Non-contrast head CT. (c) Axial view with subarachnoid hemorrhage (white arrow) in right frontal lobe. Brain MRI diffusion weighted axial imaging. (d) Restricted diffusion (white arrows) in the bilateral supratentorial and (e) infratentorial brain indicative of multifocal infarcts.

acute care facility with a trach collar and g-tube. An esophageal reconstruction with a substernal gastric conduit and left sternocleidomastoid muscle flap placed over the anastomosis was completed after seven months, which was well tolerated; the patient was discharged home POD 7.

### Case 5

A 74-year-old male with a history of follicular cell thyroid cancer/right thyroidectomy, hypertension, renal cell carcinoma/right nephrectomy presented to the ED with retrosternal chest pain, indigestion, and double vision. The patient was PAD 9 of an RFA of his LA for AF during which rapid esophageal heating was noted by the esophageal temperature probe and high flow cooling irrigation of the catheter tip was performed; prednisone for intermittent chest pain that had begun PAD 7. A TTE, chest x-ray, and EKG did not show any acute abnormalities. A chest CT with and without IV and oral contrast demonstrated a moderate volume of pneumomediastinum, and small pericardial effusion; no definitive extraluminal esophageal contrast was noted. A head CT without contrast

did not identify any acute intracranial abnormalities. A chest CT without contrast PAD 10 showed a subtle presence of contrast between the esophageal lumen and left para esophageal space consistent with esophageal perforation (Fig. 5a-c). An isolated esophageal perforation without evidence of atrial injury was identified, prompting an esophagectomy with the subsequent creation of a cervical esophagostomy; a jejunostomy, gastrostomy, and chest tube placement were also completed. The patient was discharged POD 11. An esophageal reconstruction with colonic interposition with cervical esophagus-colonic anastomosis, gastro-colonic anastomosis was completed after ten months; due to a posterior splenic laceration, a splenectomy was completed. The patient tolerated the procedure well and was discharged home POD 9.

All data are summarized in Table 1.

## Discussion

Esophageal perforation and AEF are rare but potentially fatal complications that can occur secondary



**Figure 5.** Chest CT with oral contrast only. (a) Axial (b) Sagittal and (c) Coronal images demonstrating oral contrast extravasation (white arrow) from the esophagus (E) posterior to the LA. Note is also made of pneumomediastinum (broken white arrows).

Total patients <i>n</i> = 5	AEF and Esoph <i>n</i> = 5 <sup>a</sup>	AEF only <i>n</i> = 4 <sup>a</sup>	
Demographics			
Age, mean (range)	62.8 (46–77)	59.25 (46–77)	
Sex (male) <i>, n</i> (%)	4 (80%)	3 (75%)	
Symptom presentation			
Onset Time, mean (range), days	21.4 (9–31)	24.5 (15–31)	
Fever, <i>n</i> (%)	3 (60%)	3 (75%)	
Chest Pain, n (%)	3 (60%)	2 (50%)	
AMS, n (%)	2 (40%)	2 (50%)	
Focal neurologic deficits, n (%)	3 (60%)	3 (75%)	
Hematemesis, n (%)	1 (20%)	1 (25%)	
Dysphagia and indigestion	1 (20%)		
Initial surgery	<i>n</i> = 5	<i>n</i> = 4	
Esophagectomy, n (%)	3 (60%)	2 (50%)	
Esophagostomy, n (%)	3 (60%)	1 (25%)	
Cardiopulmonary Bypass, n (%)	2 (40%)	2 (50%)	
Intercostal Muscle flap, n (%)	2 (40%)	2 (50%)	
Repeat surgery	<i>n</i> = 2	<i>n</i> = 2	
Esophagectomy, n (%)	1 (50%)	1 (50%)	
Esophagostomy, <i>n</i> (%)	1 (50%)	1 (50%)	
Intercostal muscle flap, n (%)	1 (50%)	1 (50%)	
Overall intra-hospital mortality, n (%)	2 (40%)	2 (50%)	

Table 1.	AEF and	esophageal	perforation	patient	characteristics.
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<sup>a</sup> Overall, five patients were noted with AEF or an esophageal perforation; the summary of the entire cohort is noted in the first column. Four patients had a true atrioesophageal fistula (one patient had an esophageal perforation but no fistula); the summary of this group is noted in the second column.

to CA for AEF [3,12–14]. Isolated esophageal perforation is an even less frequent complication, as most esophageal perforations present with fistula formation [13]. Therefore, the standard of care posits that early diagnosis and surgical treatment of AEF are necessary to prevent mortality [3].

To better understand these entities, a systemic review was completed noting 190 AEF patients with a

Authors	Liu et al. [3]	Della Rocca et al. [7]	Ha et al. [6]	Han et al. [5]	Barbhaiya et al.ª [12]
Description	Systematic review	Systematic review	Systematic Review	Systematic Review	Global Survey
Number of patients	190	257	126	120	31
Age, mean (range)	59.29 (24–79)	59.1 (24–85)	59 (27–85)	59 (27–85)	58 (50–67)
Sex (male), n (%)	141 (74.21)	169 (75.1)	89 (71)	87 (73)	25 (81)
Symptom Presentation					
Onset time (Range), days	21 (0–60)	21	21 (1–66)	21 (0–60)	19.3 (1–59)
Fever, n (%)	155 (81.58)	100 (40.7%)		87 (73%)	12 (39%)
Chest Pain, n (%)	64 (33.68%)	100 (40.7%)		42 (35%)	16 (55%)
AMS, n (%)	47 (24.74%)	40 (16.3%)		23 (19%)	
Focal neurologic deficits, n (%)	66 (34.74%)	122 (49.6%)		53 (44%)	
GI bleeding, n (%)	61 (33.68%)	31 (13.1%)		49 (41%)	5 (15%)
Abnormal initial chest CT, n (%)	115 (91.24)	150 (95.8%))	97 (87%)	88 (90.7%)	
Abnormal initial brain CT, n (%)	44 (60.27%)	85 (74.9%)	28 (51%)	36 (78%)	
Surgical Intervention, n (%)	89 (46.8%)	120 (51.7%)		70 (58.8%)	16 (57%)ª
Mortality in patients undergoing surgery, n (%)	30 (33.71%)	77 (35.3%)		23 (33%)	8 (50%)
Overall mortality, n (%)	120 (63.16%)	140 (59.3%)		65 (55%)	16 (52%)

Table 2.	Selective summary	y of the	literature.
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<sup>a</sup> Includes the 28 patients with full details available; grey boxes indicate that the report did not provide this information.

mean age of 59.29 and predominantly male (141/190, 74.215%; Table 2) [3]; our cases reveal similar patterns with the average age at presentation 59.25 and 75% male (n = 3). The average age of our patients with esophageal perforation with and without AEF is 62.8, 80% of which were male (n = 4; Table 1).

Patients with AEF commonly present with non-specific symptoms complicating early diagnosis and identification. The most common reported symptoms include fever (81.6%), hemiparesis (34.7%), chest pain (33.7%), gastrointestinal bleeding (32.1%), and altered mental status (24.7%) [3], similar to symptoms noted in our case series. The median time from CA to symptomatic AEF has been reported to be 21 days, with all cases occurring between 0 and 60 days [5]; in our case series, the mean presentation was 24.5 days (15–31 days; Table 1) post-ablation. Patients with neurological symptoms presented later than other patients (27.5 vs. 16 days) [3], which is also reflected in our series (26.7 vs 12 days). Thus, AEF should be in the differential for all patients within 60 days of a CA who present with fever, chest pain, altered mental status, and focal neurologic deficits.

Diagnostic imaging for AEF relies initially on chest CT [3,5,7], with fistulous tract, esophageal perforation, and free air in the mediastinum, LA, or wall of the LA as the most common abnormal findings (Figs. 1, 3, and 5; Table 3) [3,6]; narrow irregular pulmonary venous thickening, thickening of the left atrial wall, posterior fat pad induration, esophageal thickening, and pericardial or pleural effusions have also been reported [5,15,16]. While 76%–93% of AEF patients were noted to have an abnormal chest CT [3,5–7], all of the patients in our series had identified free air; oral or intravenous contrast extravasation (40%), and fistulous tract (20%) were also observed (Table 3). Given these data, however, a definitive diagnosis is only detected in 23%-35% of AEF patients on chest CT [3,5] regardless of contrast use [3,5,6,17]. In our experience, both oral and intravenous contrast aided in the diagnosis of AEF and esophageal perforation in Cases 3, 4, and 5 and allowed for urgent treatment (Figs. 3a and b, 4a, and 5a). Identification of oral contrast outside the lumen of the esophagus and intravenous contrast extravasation from the LA are helpful findings in the identification of AEF, but unfortunately, not always present.

Diagnostic challenges with chest CT include differentiating AEF from other causes of pneumomediastinum, pneumopericardium, or cardiac tamponade, as well as definitively identifying AEF. Initial chest CTs in patients with AEF can be normal (13%) [6], while subsequent chest CTs may identify esophageal perforation [18]; detection of abnormal imaging findings increases from 83.94% to 91.24% with repeat imaging [3], particularly within 4–6

Total patients n = 5	AEF and Esoph <i>n</i> = 5*	AEF only <i>n</i> = 4*
Initial CT of the Chest, n	<i>n</i> = 5	<i>n</i> = 4
Free air, n (%)	5 (100%)	4 (100%)
AEF tract, <i>n</i> (%)	1 (20%)	1 (25%)
Esophageal perforation (contrast extravasation), n (%)	1 (20%)	1 (25%)
Repeat CT Chest, n (%)	<i>n</i> = 1 <sup>+</sup>	<i>n</i> = 2
Free air, <i>n</i> (%)	1	
Esophageal perforation (contrast extravasation), n (%)	1	
Initial CT of the Head, n (%)	<i>n</i> = 2	<i>n</i> = 2
Pneumocephalus, n (%)	1	1 (50%)
Subarachnoid hemorrhage, n (%)	1	1 (50%)
Repeat CT Head, n (%)	<i>n</i> = 3	<i>n</i> = 3
Focal Infarct, n (%)	1 (33%)	1 (33%)
Diffuse Cerebral edema, n (%)	1 (33%)	1 (33%)
Pneumocephalus/intravascular air, n (%)	1 (33%)	1 (33%)
MRI brain, n (%)	<i>n</i> = 3	<i>n</i> = 2
Multi-territorial Infarcts, n (%)	3 (100%)	2 (100%)

\*Overall, five patients were noted with AEF or an esophageal perforation; the summary of the entire cohort is noted in the first column. Four patients had a true atrioesophageal fistula (one patient had an esophageal perforation but no fistula); the summary of this group is noted in the second column; †patient had both abnormalities noted.

days after the onset of symptoms [3,6]. In conjunction with a chest CT, or patients with neurologic symptoms, a head CT is the second most common imaging modality for diagnosis of AEF; however, abnormalities have been noted in only 40%-50% of patients [3,6]. The most common abnormal findings on head CT are diffuse air emboli (56.90)% and multi-territory infarcts (60.34%) [6-8,19]. In our series, head CT only noted abnormalities in 40% of patients (n = 2). The CT brain imaging findings in Case 3 included pneumocephalus; the brain CT in Case 4 showed subarachnoid hemorrhage and an ischemic infarct. Differentiating pneumocephalus from AEF versus mechanical trauma, meningitis, or fat can be challenging; intracranial fat has a much higher density (-90 HU) compared to air (-1,000 HU) but can appear hypodense on CT scans and mimic pneumocephalus [20]. Localizing the air embolism to a specific compartment is essential to determine etiology, as intravenous gas is most commonly seen in the cavernous sinuses, inferior petrosal sinuses, or potentially extracranial veins [21] and cerebral arterial gas suggests an AEF or right to left shunting [21].

Table 3. Imaging.

While MRI is capable of identifying abnormalities associated with AEF such as diffuse air emboli and ischemia better than head CT (87% vs. 51%, respectively) [6], it is less frequently used, as patients arriving at the ED with neurologic symptoms are typically evaluated by CT utilizing the stroke protocol since AEF is not always considered in the initial differential diagnosis. Of note, brain MRI was abnormal in all three of our patients that utilized this imaging, with findings of restricted diffusion in both cerebral hemispheres (Case 3) and multi-territorial infarcts (Case 1 and Case 2). If AEF is suspected clinically, early MRI examination may be helpful due to its better sensitivity at detecting air emboli and infarction.

Diagnosis or suspicion of AEF necessitates urgent surgical intervention due to the associated mortality of untreated AEF [3], as surgical repair is associated with significantly reduced mortality rates versus conservative management (33% vs. 94.19%) [3,5]. Delays of even 13 hours are associated with a threefold increase in mortality [22].

Surgical management typically repairs both the left atrial and primary esophageal defect [10,11]; patients who do not receive primary esophageal repair are more likely to experience postoperative complications, including mediastinitis, need for PEG feeds, esophageal stent, or death [11]. All patients with AEF described in our series underwent surgical correction: Cases 1 and 3 had primary esophageal repair with an esophagectomy, Case 4 required a total esophagectomy due to a persistent esophageal fistula, and, while Case 2 did not initially undergo primary repair of the esophagus, delayed esophageal repair POD 2 resulted in multiple postoperative complications ultimately leading to the withdrawal of life support POD 19. Case 5 was noted to have an isolated esophageal perforation and only underwent esophagectomy and did not require fistula takedown or repair of the LA. Ultimately, even with surgical intervention (Table 2), >50% die post-operatively (Tables 1 and 2), indicating the gravity of AEF.

Endoscopic intervention alone, including esophageal stenting, remains secondary to surgical repair due to increased mortality (65%) compared to those undergoing surgical repair (33%; Table 2) and should be reserved only for patients unfit for surgical intervention [5,23]. Nonsurgical treatments instituted without a definitive diagnosis of AEF include making the patient *nil per os*, broad-spectrum antibiotics, and total parenteral nutrition [3]; however, surgical repair is the only resolution for AEF.

Isolated esophageal perforation without a fistula formation is an extremely rare complication of CA; however, surgical intervention is still essential to prevent the formation of AEF [12]. The treatment in these patients is less well defined, with limited case series showing some success with esophageal stenting without neurological injury (n = 3; 100%) [12]. Primary repair of the esophageal perforation and subsequent reinforcement with omental wrapping [24] has also been reported as an alternate technique (n = 2; 100%) [25]. In Case 5, the damage to the patient's esophagus was too extensive for stenting or primary repair, instead requiring esophagectomy.

# Conclusion

These data have suggested optimal imaging to facilitate definitive AEF diagnosis for early and optimal management. For patients within 60 days post-CA who present with fever, chest pain, gastrointestinal bleeding, or neurologic symptoms, oral or arterial contrast-enhanced helical chest CT with thin section collimation as initial imaging is warranted [3,26]. Thin-section axial reconstruction utilizing a 1 mm-mm detector with sagittal and coronal reformats allow for optimal identification of abnormalities consistent with AEF. Patients who present with neurologic symptoms may initially be worked up for the stroke, receiving an initial head CT; the presence of pneumocephalus, infarcts involving one or more vascular territories, or diffuse air emboli is highly suggestive of AEF, warranting a chest CT with IV contrast to evaluate the presence of AEF. An initial normal/unremarkable chest CT does not rule out AEF in symptomatic patients within 60 days of CA [6,7]. Repeat chest CT with IV contrast as early as 1–3 days after the initial imaging [3] increases the likelihood of a definitive AEF diagnosis. EGD, TEE, and endoscopy should not be performed in patients with suspected AEF due to reported clinical deterioration [3,5,6,9]. Surgery is the only recommended management in patients with AEF who are clinically stable enough to be brought to the OR [3,7].

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