

Received: 2017.02.22
Accepted: 2017.05.09
Published: 2017.08.01

Atrio-Esophageal Fistula: A Case Series and Literature Review

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Conflict of interest: None declared

Case series

Patient: Male, 72 • Male, 29 • Male, 75
Final Diagnosis: Atrio-esophageal fistula
Symptoms: Altered mental state • chest pain • fever • melena
Medication: —
Clinical Procedure: —
Specialty: Critical Care Medicine





Objective: Rare disease
Background: Percutaneous catheter radiofrequency ablation (RFA) and cryoablation of the left atrium and pulmonary vein ostia have become successful therapeutic modalities in the management of atrial fibrillation. Atrio-esophageal fistula is a rare complication. Awareness of complication risk is imperative because without prompt diagnosis and urgent surgical intervention, the outcome is often fatal. We present 3 cases of atrio-esophageal fistula following percutaneous catheter radiofrequency ablation (RFA).

Case Reports: Case 1: A 72-year old white male presented 27 days after percutaneous RFA for atrial fibrillation with fever, altered mental status, and melena. Esophagogastroduodenoscopy (EGD) revealed a 1-cm defect in the mid-esophagus. Upon thoracotomy, severe hemorrhage ensued from a concomitant injury to the left atrium. Multiple attempts to repair the left atrial perforation were unsuccessful and the patient died. Case 2: A 71-year old white male presented 29 days after percutaneous RFA for atrial fibrillation with fever and tonic-clonic seizure. Recognition of possible atrio-esophageal fistula was considered and confirmed on thoracotomy. Surgical fixation of the left atria and esophagus were performed. The patient survived and was discharged to a skilled care facility. Case 3: A 75-year old white male presented 24 days after percutaneous RFA for atrial fibrillation with chest pain. An echocardiogram revealed a large pericardial effusion and pericardiocentesis was performed. Despite aggressive measures, the patient died. The autopsy demonstrated a communicating esophageal fistula with the right pulmonary vein.

Conclusions: Clinicians tending to patients who have recently undergone atrial ablation need to be aware of atrio-esophageal fistula as a rare but highly fatal complication.

MeSH Keywords: Atrial Fibrillation • Catheter Ablation • Esophageal Fistula • Heart Atria

Full-text PDF: <https://www.amjcaserep.com/abstract/index/idArt/903966>

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Background

Atrial fibrillation is the most common clinically significant arrhythmia in the United States [1]. While pharmacologic therapy remains a cornerstone of treatment, percutaneous catheter radiofrequency ablation (RFA) and cryoablation of the left atrium and pulmonary vein ostia have become successful therapeutic modalities for medication-resistant atrial fibrillation [2,3]. As first described in 2004, atrio-esophageal fistula is a rare complication of percutaneous catheter ablation and can have a mortality rate as high as 71% [4,5]. We present 3 cases of atrio-esophageal fistula following percutaneous catheter RFA of the left atrium.

Case Report

Case 1

A 72-year old white male with a past medical history of atrial fibrillation, congestive heart failure, coronary artery disease, and ischemic cardiomyopathy with percutaneous pacemaker and defibrillator presented to the Emergency Department (ED) of a large tertiary care hospital at around midnight with acute-onset fever, chills, rigors, and confusion. Of note, he had undergone percutaneous catheter RFA of his left atrium for atrial fibrillation 27 days prior. He had been on stable doses of amiodarone 200 mg orally daily, aspirin 81 mg orally daily, atorvastatin 40 mg orally daily, metoprolol succinate 150 mg orally daily, potassium chloride 20 mEq orally daily, spironolactone 25 mg orally daily, torsemide 20 mg orally daily, and warfarin 2 mg orally daily. A social history was obtained, which revealed a prior history of tobacco use and the patient denied history of intravenous drug use. After admission to a medical floor, the patient became suddenly unresponsive, with left-sided neurologic deficits, including upper- and lower-extremity weakness. He was transferred to the Medical Intensive Care Unit (MICU).

On exam, he was febrile with a temperature of 38.5°C, tachycardic with a pulse range of 88–135 beats per minute, and blood pressure 98–128/58–62 mm Hg. A cardiopulmonary exam revealed a tachycardic, irregular rhythm, no murmurs, and lungs clear to auscultation. On neurologic exam, he was obtunded with diffuse left upper- and left lower-extremity deficits. Labs were obtained and were mostly within normal limits with the exception of a creatinine 1.77 mg/dL [normal 0.7–1.3 mg/dL], hemoglobin 12.1 g/dL [normal 13–17 g/dL], and INR 3.9. Electrolytes, anion gap, and lactic acid were normal. A head computed tomography (CT) was performed showing hypo-densities right greater than left in the frontal sulci (Figure 1A). Blood cultures were drawn and empiric antibiotics were started for possible infective endocarditis and bacterial meningitis. A Neurology consult was requested. A CT

angiogram of the head and neck was not obtained because of his elevated creatinine and an MRI of the brain was not obtained because of his pacemaker. Further, a lumbar puncture was not obtained because of his elevated INR. In the morning, a repeat CT of the head was obtained, revealing resolution of the hypodensities, with findings suspicious for air within the venous structures. Blood cultures grew gram-positive cocci. That evening, he began having profuse melena. His hemoglobin dropped from 12.1 g/dL to 5.5 g/dL and he was intubated and placed on mechanical ventilation. Aggressive transfusion occurred with numerous blood products. Gastroenterology was consulted and performed an emergent esophagogastroduodenoscopy (EGD). Despite evidence of an upper gastrointestinal bleed with a large clot burden in his stomach, no obvious bleeding source was identified. A nasogastric tube had been placed previously and was obscuring endoscopic evaluation. Upon retraction of the scope, a 1-cm defect was identified in the mid-esophageal wall, with viewable mediastinum (Figure 2). At that time, the defect appeared to be a complication of the EGD. A CT of the chest without contrast was ordered because of elevated creatinine, revealing irregularity involving the mid-thoracic esophagus with foci of adjacent extraluminal air and inflammatory changes suspicious for perforation (Figure 3A, 3B). Cardiothoracic surgery was consulted to repair esophageal perforation thought to be secondary to EGD. Upon thoracotomy, a severe hemorrhage ensued from an apparent concomitant injury to the left atrium. Multiple attempts to repair the left atrial perforation were unsuccessful and the patient died from hemorrhagic shock. A history of previous left atrial ablation and presentation with fevers, neurologic insult with evidence of air emboli, bacteremia with *Streptococcus mitis* and *oralis*, known oral flora bacteria, gastrointestinal bleed, and concomitant injury to both the left atrium and esophagus were consistent with a diagnosis of atrio-esophageal fistula.

Case 2

A 71-year old white male with past medical history of hypertension, diabetes, and symptomatic atrial fibrillation refractory to electrical cardioversion presented to an outside hospital with symptoms of abdominal pain, nausea, and vomiting of 2-day duration. Of note, he had undergone percutaneous catheter RFA of his left atrium for atrial fibrillation 29 days prior. He had been on stable doses of sotalolol 80 mg orally twice daily, warfarin 5 mg orally nightly, metoprolol succinate 25 mg orally daily, glargine 25 units subcutaneously nightly, metformin 1000 mg orally twice daily, sitagliptin 50 mg orally daily, lisinopril 10 mg orally daily, and atorvastatin 10 mg orally nightly.

On exam, he was febrile with a temperature of 39.2°C, tachycardic with a rate of 98–118 beats per minute, and blood pressure 101–129/68–77 mm Hg. A cardiopulmonary exam revealed a tachycardic regular rhythm, no murmurs, and lungs clear to

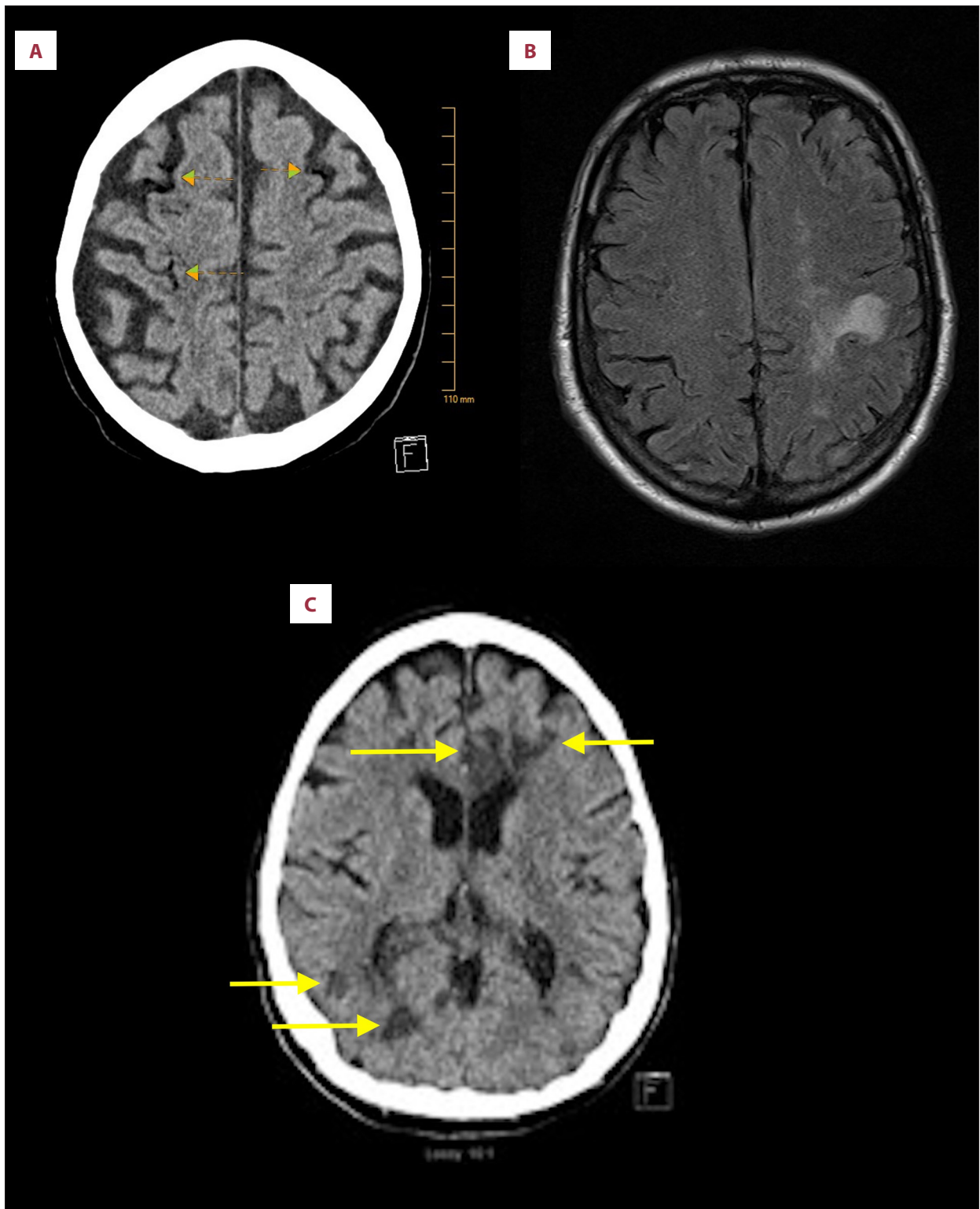


Figure 1. (A) Curvilinear foci of hypodensity seen adjacent to the right greater than left frontal sulci, representing air emboli within venous or arterial structures (yellow arrows). (B) Scattered foci of diffusion restriction in the bilateral hemispheres. (C) Multiple bilateral acute infarcts with distribution suspicious for embolic origin (yellow arrows).

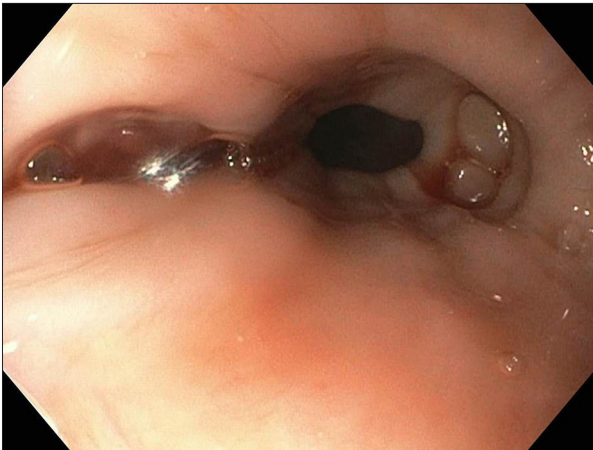


Figure 2. Esophagogastroduodenoscopy image depicting an approximate one centimeter defect in the mid-esophagus with viewable mediastinum representing the atrio-esophageal fistula.

auscultation. Initially, no neurologic abnormalities were present. Labs were obtained with hemoglobin at 12.2 g/dL [normal 13–17 g/dL] and an INR of 3.6. Electrolytes, creatinine, anion gap, and lactic acid were normal. Chest x-ray demonstrated infiltrates and he was started on antibiotics and admitted for a presumptive pneumonia. Shortly after admission, his troponin I increased from reported normal to 0.80 ng/mL [normal 0.0–0.78 ng/mL]. An electrocardiogram revealed sinus rhythm with a right bundle branch block of unknown duration. Of note, blood cultures were positive for gram-positive rods. The decision was made to transfer the patient to the cardiology

service at a tertiary care hospital for presumed non-ST elevation myocardial infarction. Prior to transfer, the patient had a witnessed generalized tonic-clonic seizure and was intubated for hypoxic respiratory failure. Head CT without contrast at the outside hospital was negative for intra-cranial pathology. Intravenous antiepileptics were started and he was transferred to the Medical Intensive Care Unit (MICU) at the tertiary care hospital. Upon arrival, a CT chest without contrast was obtained given concern for atrio-esophageal fistula. A 1.4-cm collection of air between the esophagus and the left atrium was found with mediastinal stranding (Figure 4A, 4B). A cardiothoracic surgery consult was requested for probable diagnosis of an atrio-esophageal fistula. A collaborative decision was made for timely transfer of the patient to an academic university teaching hospital for necessary surgical intervention.

Upon arrival at the university hospital, he was taken immediately to the operating room. Aggressive reversal of his elevated INR with fresh frozen plasma was initiated. An EGD during thoracotomy was performed and revealed an eschar of the esophagus at approximately 35 cm from the incisors. No further endoscopic manipulation of the esophagus was performed. A right-sided thoracotomy was performed that revealed an adherent fistula between the left atrium and mid-esophagus. Surgical exploration revealed an open defect of both atrium and esophagus. Bleeding was controlled with tamponade and the patient was placed on cardiac bypass with repairs in both the atrial and esophageal defects. After successful repair, he was transferred to the Surgical Intensive Care Unit (SICU). Post-op MRI of the brain with and without contrast revealed

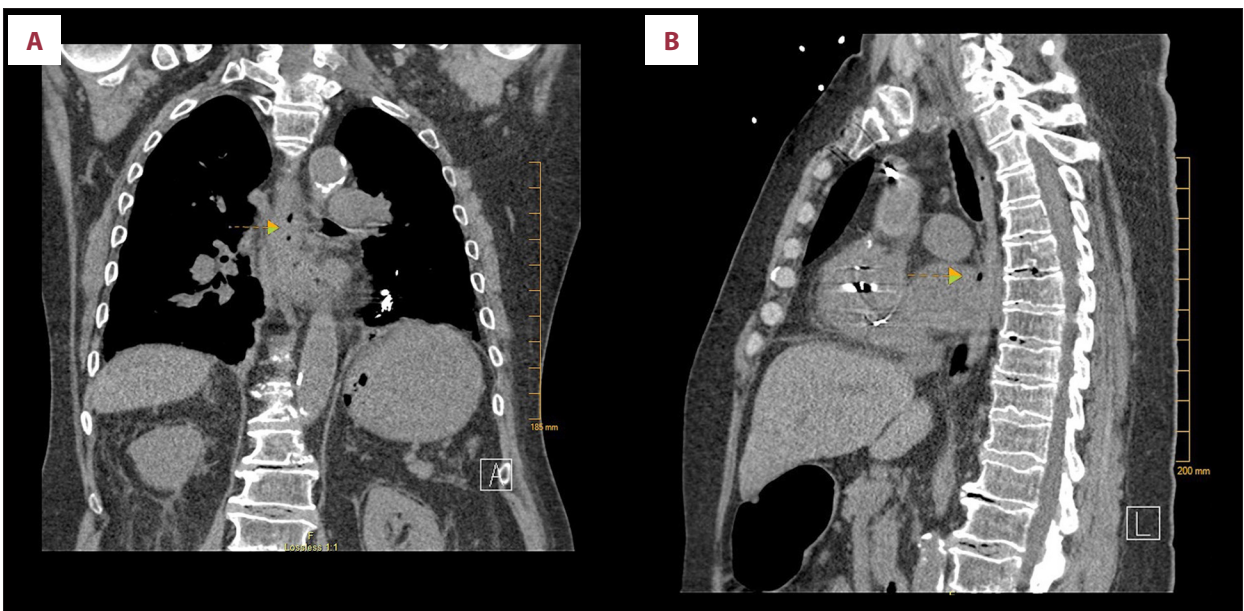


Figure 3. (A) CT Chest coronal view demonstrating a mild irregularity involving the mid-thoracic esophagus with tiny foci of adjacent extraluminal air and inflammatory change suspicious for perforation (yellow arrow). (B) CT Chest sagittal view demonstrating extraluminal anterior to the mid-esophagus and posterior to the left atrium (yellow arrow).

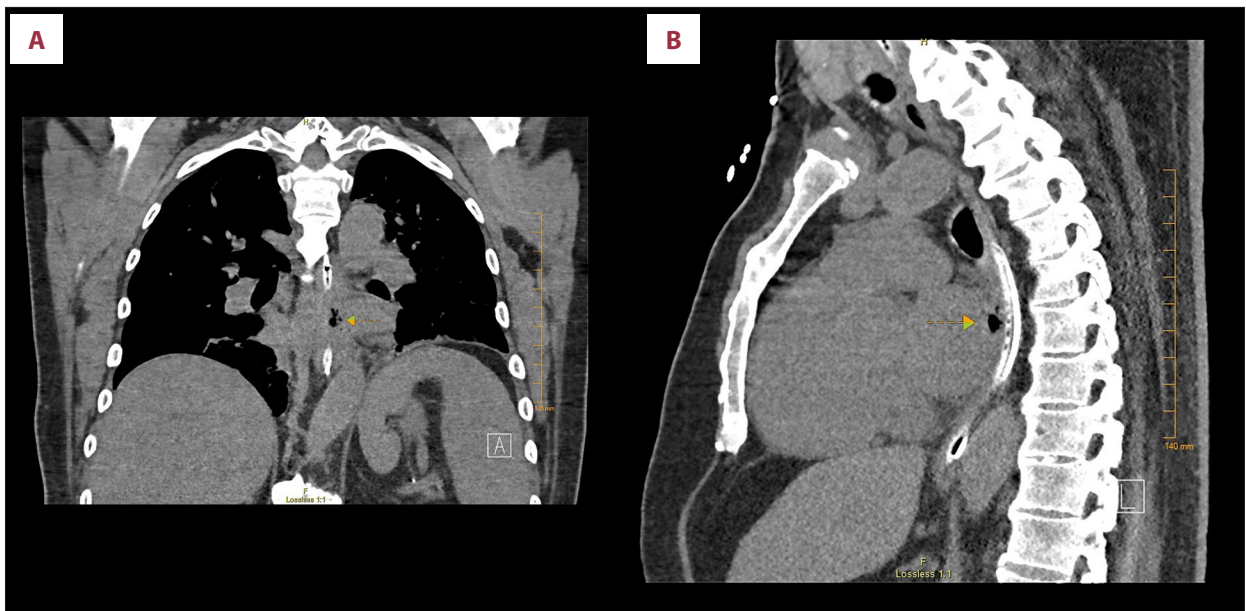


Figure 4. (A) CT Chest coronal view demonstrating mediastinal air anterior to the esophagus and posterior to the superior aspect of the left atrium (yellow arrow). (B) CT Chest sagittal view demonstrating extraluminal air in the mediastinum (yellow arrow).

scattered foci of diffusion restriction consistent with septic emboli (Figure 1B). Blood cultures grew *Lactobacillus* species, a known gastrointestinal flora. After a prolonged hospital stay of 18 days, the patient was discharged to a skilled facility for rehabilitation.

Case 3

A 75-year old white male with past medical history of hypertension and paroxysmal atrial fibrillation presented to an outside hospital with symptoms of chest pain and shortness of breath. Of note, he had undergone percutaneous catheter RFA of his left atrium for atrial fibrillation 21 days prior. He had been on stable doses of apixaban 5 mg orally twice daily and lisinopril 5 mg orally daily. On exam, he was afebrile with a temperature of 37.2°C, tachypneic with respiration rate 26–29 breaths per minute, tachycardic with a rate of 94–123 beats per minute, and blood pressure was 80–107/48–72 mm Hg. On admission, he was found to have runs of supraventricular tachycardia, which was believed to be the cause of his dyspnea. He was placed on intravenous amiodarone with subsequent stabilization of his arrhythmia. After 3 days of worsening oliguria, he was transferred to the Medical Intensive Care Unit (MICU) at a tertiary care hospital for Nephrology consultation.

Upon transfer, a cardiopulmonary exam revealed elevated jugular venous distension and distant heart sounds with regular rhythm and systolic murmur in the left upper sternal border. Labs were obtained with AST 495 U/L [normal: 8–48 U/L], ALT 678 U/L [normal: <41U/L], BUN 71 ug/dL [normal: 6–20 ug/dL], creatinine 4.79 mg/dL [normal: 0.8–1.3 mg/dL], potassium 5.6

mmol/L [normal: 3.5–5.1 mmol/L], INR of 7.0, and WBC 21 000 cells/mcL [normal: 4000–11 000 cells/mcL]. A chest x-ray demonstrated moderate left pleural effusion. A transthoracic echocardiogram found pericardial effusion with moderately dilated left ventricle and ejection fraction 45%. Continuous renal replacement therapy (CRRT) was initiated with diagnosis of acute tubular necrosis. He was given subcutaneous Vitamin K 10 mg and fresh frozen plasma to correct elevated INR. Pericardiocentesis was postponed at that time due to supratherapeutic INR. However, due to clinical improvement, the procedure was ultimately deferred despite resolution of his elevated INR. He was transferred to a monitored telemetry floor where CRRT was transitioned to intermittent hemodialysis, and ultimately discontinued.

On day 6 of admission, the patient developed new leukocytosis, fever, and dyspnea. A chest x-ray revealed increased left pleural effusion and a diagnostic thoracentesis was performed without complication. One hour after the procedure, the patient was found unresponsive, hypoxic, and tachycardic. He was emergently intubated and transferred back to the Medical Intensive Care Unit (MICU), where a bedside echocardiogram showed moderate-to-large pericardial effusion with early tamponade physiology. Bedside pericardiocentesis was performed, with no improvement in hemodynamics. Cardiothoracic surgery was consulted and patient was transported to the operating room. During surgical exploration, purulent material was drained from the pericardial space. There was concern for communication between esophagus and left atrium resulting from prior ablation, but this was not visualized during surgery. The pericardiocentesis catheter was found

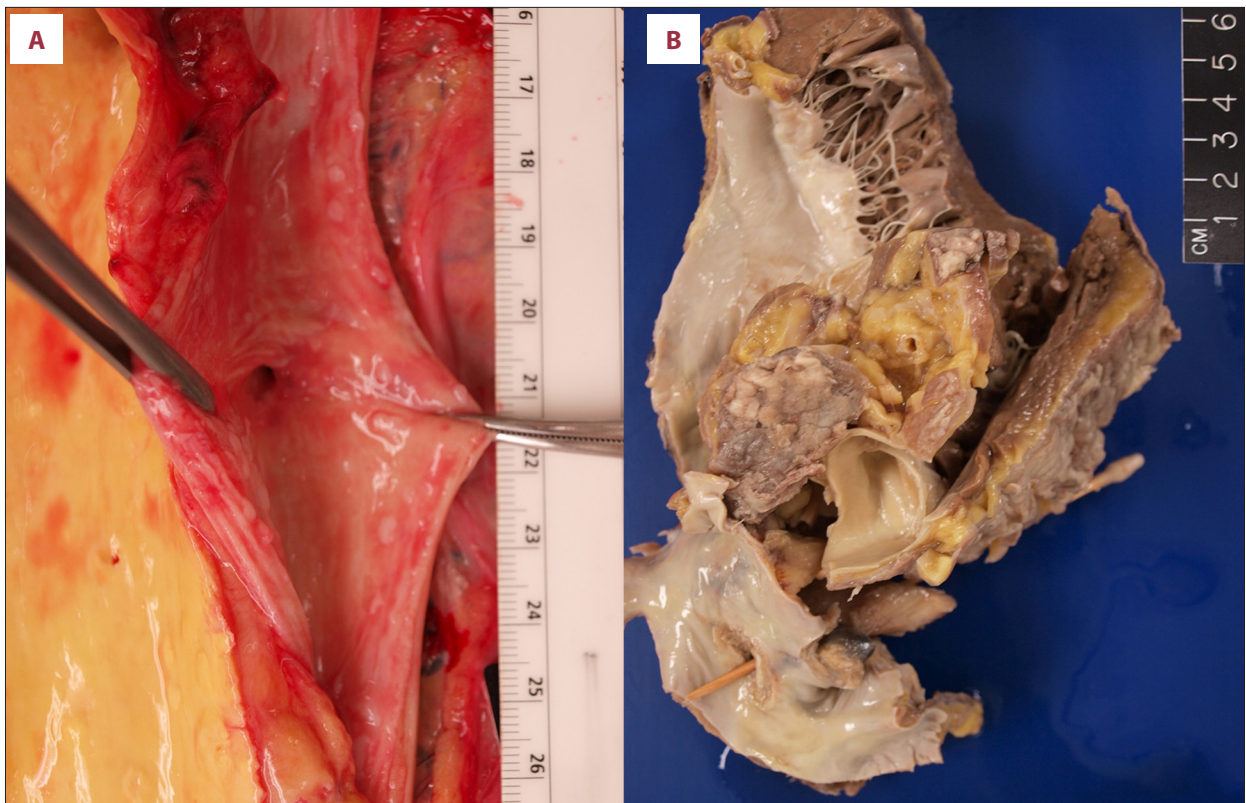


Figure 5. (A) Autopsy image demonstrating the esophageal fistula opening. (B) Formalin-fixed autopsy image demonstrating the fistula track and opening in the left atrium.

entering the right ventricle wall and removed. Broad-spectrum antibiotics were started for purulent pericardial effusion. His condition deteriorated and vasopressors were initiated. He remained unresponsive despite absence of sedation. A CT head (Figure 1C) showed multiple bilateral acute infarcts with distribution suspicious for embolic origin. After discussions with family members, the patient was transitioned to comfort care and died on day 13 of hospitalization. An autopsy was performed and showed a large communicating esophageal fistula with the right pulmonary vein, consistent with the diagnosis of atrio-esophageal fistula (Figure 5A, 5B).

Discussion

Atrial fibrillation is the most common clinically significant cardiac arrhythmia in the United States and it is estimated there will be approximately 10 million people with this diagnosis in the next 20 years [1]. While pharmacologic therapy remains the cornerstone of treatment, percutaneous catheter ablation of the left atrium and pulmonary vein ostia has become a successful therapeutic modality for medication-resistant atrial fibrillation. Between 1990 and 2005, there were an estimated 133 000 radiofrequency ablations performed in the United States for atrial fibrillation [2]. Each year, there has been a statistically

significant increase in the number of RFA performed, with a roughly 15% increase every year ($p < 0.001$) [2]. In the United States, approximately 50 000 RFA are performed annually [3]. In addition, there have been over 120 000 cases of cryoablation used in the management of atrial fibrillation worldwide since 2011 [6]. As more ablations are performed, the risk of encountering an atrio-esophageal fistula may also increase.

First described in 2004, atrio-esophageal fistula is a rare complication of percutaneous catheter RFA [7]. Since 2004, there have been 53 case reports published on this condition as a complication of radiofrequency ablation [8]. The incidence after RFA was initially very low (estimated $< 0.04\%$), but more recent studies demonstrate a higher incidence, estimated at 0.2–0.4% [4,5,8–13]. Early reports speculated the incidence of atrio-esophageal fistula to be higher due to missed diagnosis [4,5,9,12]. The first case report of atrio-esophageal fistula following cryoablation was published in 2012 and since then there have been 11 cases reported [6]. The risk for atrio-esophageal fistula may be less with cryoablation, with a lower reported incidence of atrio-esophageal fistula estimated at 0.01–0.014% [6,14]. Despite success rates estimated at between 60% to 80% with ablation compared to 20% to 70% with medical therapy, the depth of the ablative lesion is difficult to evaluate and control [9,15–19]. This can be compounded by

the esophagus proximity to the posterior wall of the left atrium, often separated by a thin layer of fat a few millimeters thick. This is supported by evidence that an estimated 30% to 47% of all patients who undergo radiofrequency atrial ablation have some degree of injury to the esophagus mucosal layers [11,20,21]. Thermal injury from ablation leads to ischemic necrosis of the atrium and esophageal mucosal layers, creating the fistula [12]. Several techniques have been implemented to reduce the risk of thermal injury, including electro-anatomic mapping systems, intracardiac echocardiography, decreased power delivery, decreased tissue contact pressure with frequent probe repositioning, esophageal temperature monitoring, and temperature sensors at the electrode catheter tip [22]. Currently, related data on these potential modifications are inconclusive.

It is imperative that physicians recognize the signs and symptoms of atrio-esophageal fistula. Time from ablation to symptom onset has been reported to be from 2 days to 5 weeks, with the most common occurrence at 10 to 17 days [4,11,23,24]. Most patients present with the constellation of fever and neurologic deficits, often secondary to air emboli [24–26]. Less commonly, there is septicemia with oral flora bacteremia, chest pain, and hematemesis/melena [24–26]. Presentation of the described patients was delayed, with symptoms first appearing 21 to 29 days after ablation. It is important to note that when atrio-esophageal fistula is suspected, instrumentation of the esophagus with esophagogastroduodenoscopy (EGD) or trans-esophageal echocardiogram (TEE) is contraindicated, as insufflation of the esophagus can result in the formation of air emboli causing neurologic injury and death [7,11,24,25]. In the first presented case, EGD was performed due to a life-threatening gastrointestinal bleed and occurred before the diagnosis of atrio-esophageal fistula was made. In the second case, EGD was performed during surgical repair. It is important that when atrio-esophageal fistula is suspected, insufflation of the esophagus with either EGD or TEE be

avoided. The most reliable modality for diagnosis is a CT of the chest with contrast, demonstrating extraluminal air or pneumomediastinum, air in the atrium, or the presence of intravenous contrast within the esophagus [7,11,26,27]. In the first case described, CT chest without contrast was performed because the diagnosis was not suspected. When atrio-esophageal fistula is suspected, it is recommended that a CT chest with contrast be performed as it remains the test of choice. The overall mortality of atrio-esophageal fistula has been estimated at 40–80% [8,11,12,25]. A 2016 review by Khan et al. found an estimated mortality rate of 71% [4,5]. A contributing component to the high mortality rate is failure to recognize the condition early enough [9,12,28]. The mainstay treatment is urgent cardiothoracic surgical intervention, and can include cardiopulmonary bypass with resection of necrotic tissue and patch repair [28,29]. Despite surgical intervention, the mortality rate remains high, reported at 41% after corrective intervention [30]. In this case series, the second case occurred just months after the first case and we believe rapid recognition and diagnosis, as a result of increased awareness, may have contributed to improved outcomes. The third case occurred 2 years after the first 2 cases and diagnosis may have been missed due to lack of awareness.

Conclusions

In summary, 3 cases of atrio-esophageal fistula following RFA were presented. Although rare, because of its high mortality rate, it is important that clinicians involved in the care of patients who have undergone percutaneous catheter ablation be aware of this potentially lethal complication and its clinical manifestations. When atrio-esophageal fistula is suspected, instrumentation of the esophagus with TEE or EGD should be avoided and a contrast-enhanced CT chest be obtained. Early cardiothoracic surgical intervention remains the mainstay treatment.

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