Ahmed B. Elawad, MD
Creighton university
Omaha
abbelawad@gmail.com
(312) 522-6829
Case report (includes case series that include 5 or fewer patients)

Title: Another STEMI; Or a Sign Heralding Something More Sinister?

Description: A 44-year-old man presents with a massive STEMI and cardiac arrest secondary to a suspected coronary air embolism, 3 weeks after a routine ablation for atrial fibrillation.

Another STEMI; Or a Sign Heralding Something More Sinister?

Ahmed B. Elawad, MD[1], Navya Alugubelli, MD [1], Ryan Kimball[2], Paul Wilkinson[2], Hussam Abuissa, MD[1]

Institutional Affiliations: 1: Division of Cardiology, Creighton University School of Medicine, Omaha, Nebraska, United States 2: Creighton University School of Medicine, Omaha, Nebraska, United States

A 44-year-old man in his usual state of health develops sudden-onset, crushing, substernal chest pain, shortly before vomiting bright red blood and collapsing unconscious at work.

Emergency personnel arrive; patient is non-responsive, breathing agonally with bloody oral secretions and emergently intubated. Blood is suctioned via os but none via endotracheal tube. He has a pulse but is reported to be in a rapid wide complex irregular rhythm at 170 beats per minute, which soon decompensates pulseless on arrival to the ER. CPR is quickly initiated with return of circulation after 2 rounds of compressions and Epinephrine without need for defibrillation.

On evaluation in the ER, blood pressure was 95/64 mm Hg, heart rate was 120 beats per minute, and respiratory rate was 36 breaths per minute with an oxygen saturation of 92% on 100% Fi02 oxygen via ventilator. Pertinent physical exam findings included pale appearance with bloody oral secretions, sluggish pupils, and irregularly irregular pulse with distant heart sounds without murmurs. Capillary refill is delayed with cold extremities and non-palpable distant pulses.

He has a past medical history of non-ischemic TTN gene-positive dilated cardiomyopathy, heart failure with improved ejection fraction from 15% to 55-60%, and persistent atrial fibrillation for which he had recently undergone ablation 3 weeks prior at an

outside hospital and currently on anticoagulation with Apixaban. Other history included gastro-esophageal reflux disease with prior Helicobacter pylori infection, alcoholism in remission, and a remote history of anabolic steroid use.

An electrocardiogram is obtained which reveals irregularly irregular rhythm with profound ST-elevations in multiple coronary territories. **(fig.1)**

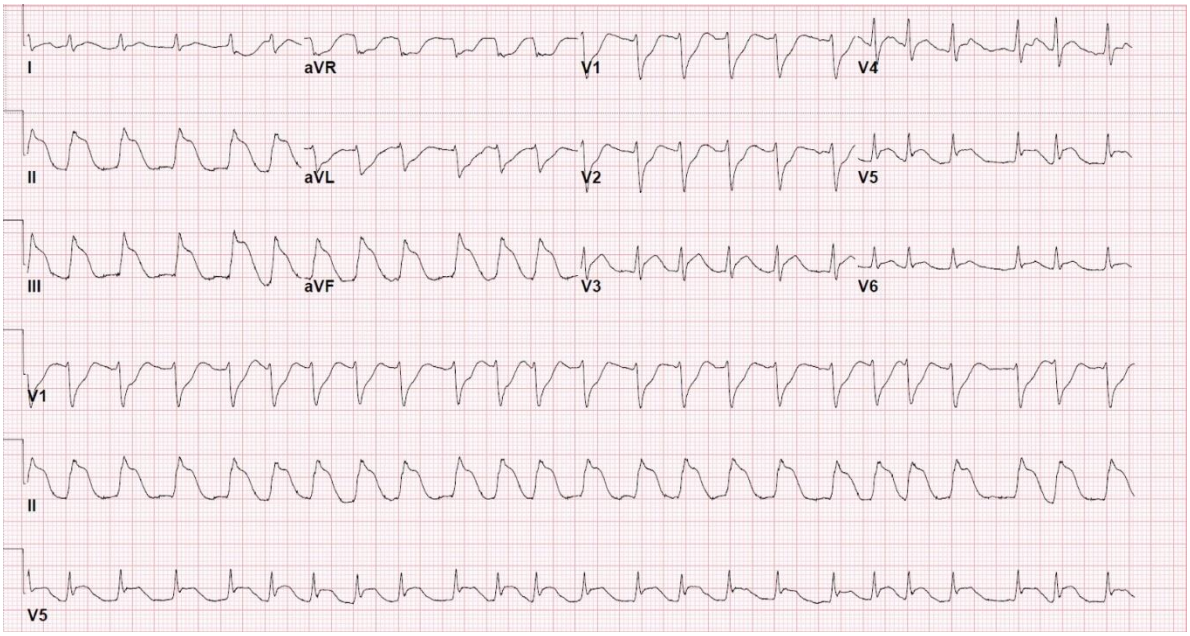

Fig.1 – initial EKG

An orogastric tube is placed and blood transfusions are initiated in addition to 4-factor prothrombin complex concentrate. The interventional cardiologist is called to activate the catheterization laboratory for an ST elevation myocardial infarction; however, noting the ongoing upper gastrointestinal bleeding, the catheterization laboratory is not immediately activated. The patient is evaluated by the gastro-enterology team and noted to have only minimal bloody output via the orogastric tube but a rapidly declining neurological exam. A CT scan of the head, chest, abdomen and pelvis was recommended prior to any invasive evaluation.

The CT scan of the head revealed bilateral loss of gray-white differentiation and scattered gas throughout the left cerebral hemisphere **(fig. 2).** Chest CT revealed an ominous sign; air in the left atrium – consistent with a diagnosis of atrioesophageal fistula (AEF). Repeat EKG 1 hour later showed complete resolution of ST-elevations. Despite aggressive management, the patient expired shortly thereafter.

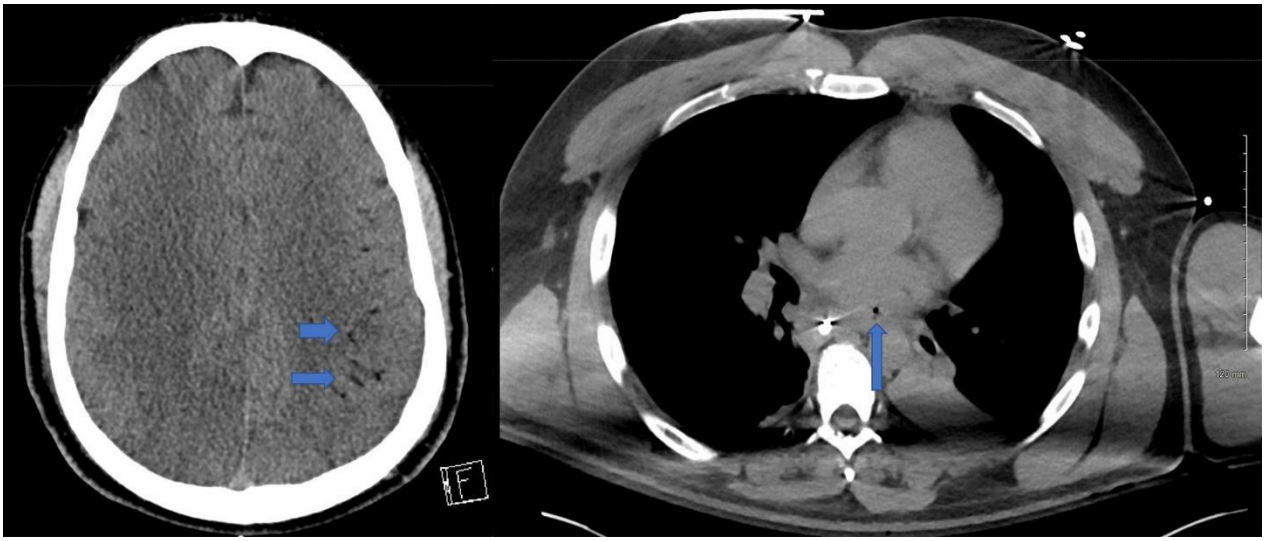

Fig. 2 – CT scan head and chest – demonstrating scattered air within cerebellar hemisphere and air within the left atrium

Discussion

Atrioesophageal fistula is one of the most harrowing and catastrophic complications of AF ablation and has a reported incidence of 0.1-0.25%[1]. The number of ablation procedures for AF has been rapidly increasing, with more than 50,000 ablations undertaken per year in the United States. The estimated global incidence of new patients with AF is approximately 5 million per year, with the highest incidence rates seen in North America[2]. This upward trend of AF, coupled with expanding indications for catheter ablation procedures, highlights the absolute

importance of early recognition of AEF as a rare, yet possible complication that is almost invariably associated with certain mortality if not promptly recognized and intervened[3].

A retrospective study by Tan et al, investigating causes of early mortality following ablation estimated that 13% of early mortality was associated with AEF[4]. Aggregated survey data by Chirag et al, including 191,215 ablations reported a total of 31 esophageal perforations or fistulas; 20 of those were AEF[5].

There are many proposed mechanisms of AEF formation, and it likely occurs as a complex interplay of multiple factors including the close-proximity of the thin-walled posterior left atrium, especially in long-standing AF, and the esophagus – setting the stage for thermal injury to esophageal microvasculature and small vagal nerve branches. This results in ulceration and esophageal dysmotility with increased reflux and further injury to a susceptible mucosa. This inciting thermal injury could not occur if not for the ubiquitously close anatomical approximation between atrium and esophagus. As such, fistula formation usually originates from the esophageal side towards the left atrium and then typically functions as one-way valve allowing air to enter from esophagus into left atrium, and less likely to allow blood from atrium into esophagus - although both can be seen as demonstrated in our patient[6]. While all modalities of ablation can lead to AEF, it has been observed most frequently with radiofrequency energy tissue ablation, especially robotic, with high temperature and power use, and with general anesthesia[1].

The median observed time from procedure to AEF was 21 days, with the most common period 2-4 weeks post-procedure[2]. This coincides with the time-to-presentation in our patient.

While AEF most often presents with fever (73%) and neurological symptoms (72%), it can also induce gastrointestinal (41%) and cardiac (40%) symptoms[2]. CT chest findings include

intravascular air, pericardial effusion, systemic emboli, and communication between the atria and esophagus. One retrospective study found that contrasted CT of the chest was abnormal in only 68% of confirmed AEF cases[2]. This emphasizes the importance of high index of suspicion and familiarity with this entity and recognizing the significant likelihood of a false negative initial CT.

If AEF is left untreated, it is almost universally fatal. The presence of neurological symptoms or gastrointestinal bleeding have been associated with the highest mortality rates. A previous study by Han et al, found overall mortality of AEF to be 55%, with mortality decreasing to 33% in patients who received surgical intervention, vs 65% in those undergoing esophageal stenting and 97% with medical management alone[2].

Several strategies to reduce the incidence of esophageal injury have been suggested. Luminal esophageal temperature monitoring has been shown to reduce the risk of thermal injury to the esophagus. Endoluminal temperatures of greater than 41°C have been associated with an increased risk of AEF [7]. Mechanically displacing the esophagus with an endoscope during the ablation has shown some success in preventing esophageal injury[6]. Cooling has been proposed as another method, but has thus far produced conflicting data with no definitive benefit, and possibly harm, although this data may be confounded[7]. Administering proton-pump inhibitors has also been proposed, but effectiveness has not replicated in clinical trials, possibly due to the rarity of this complication[8].

Further, air emboli can occur as a result of a direct communication between arterial circulation and esophagus. The CT scan in this case showed scattered air within the left cerebral hemisphere. Further, the profound ST elevation in multiple coronary territories with subsequent resolution on repeat ECG strongly suggests multiple air emboli into the coronary circulation.

Although this more commonly occurs during arterial instrumentation, appearance of diffuse ST elevation should raise suspicion for possible AEF in post-ablation patients.

The first goal of is to prevent air propagation, as this is almost certain of the worst outcomes. The only thus far proven way to prevent air embolization is prompt open surgical repair. Once repaired, treatment is mainly 100% oxygen and hyperbaric oxygen to promote embolus dissolution and reduce cerebral edema. Aspiration via catheter can be used as a last resort if a coronary air embolus persists.

While AEF is an exceedingly rare complication of AF ablation, it is associated with invariable mortality if not immediately recognized. Any esophageal instrumentation should be absolutely avoided to prevent propagating air embolization in cases where AEF may be suspected – especially endoscopy. There should be a heightened suspicion of AEF in all patients presenting with neurological, gastrointestinal, or cardiac symptoms within 2 months of AF ablation, and it is a condition that not only cardiologists should be familiar with, but medical and emergency physicians alike. The ability to recognize AEF immediately upon presentation is the difference, quite literally, between life or death, as without immediate surgical repair, fatality is without a doubt certain.

References:

1. Kapur S, Barbhaiya C, Deneke T, Michaud GF. Esophageal injury and atrioesophageal fistula caused by ablation for atrial fibrillation. *Circulation*. 2017;136.
2. Han HC, Ha FJ, Sanders P, et al. Atrioesophageal Fistula: Clinical Presentation, Procedural Characteristics, Diagnostic Investigations, and Treatment Outcomes. *Circ Arrhythm Electrophysiol*. 2017;10.
3. Yousuf T, Keshmiri H, Bulwa Z, et al. Management of Atrio-Esophageal Fistula Following Left Atrial Ablation. *Cardiol Res*. 2016;7.
4. Tan MC, Rattanawong P, Karikalan S, et al. Causes of Early Mortality after Catheter Ablation of Atrial Fibrillation. *Circ Arrhythm Electrophysiol*. 2023;16.
5. Barbhaiya CR, Kumar S, John RM, et al. Global survey of esophageal and gastric injury in Atrial fibrillation ablation: Incidence, time to presentation, and outcomes. *J Am Coll Cardiol*. 2015;65.
6. Leite LR, Santos SN, Maia H, et al. Luminal esophageal temperature monitoring with a deflectable esophageal temperature probe and intracardiac echocardiography may reduce esophageal injury during atrial fibrillation ablation procedures results of a pilot study. *Circ Arrhythm Electrophysiol*. 2011;4.
7. Müller P, Dietrich JW, Halbfass P, et al. Higher incidence of esophageal lesions after ablation of atrial fibrillation related to the use of esophageal temperature probes. *Heart Rhythm*. 2015;12.
8. Calkins H, Hindricks G, Cappato R, et al. 2017 HRS/EHRA/ECAS/APHRS/SOLAECE expert consensus statement on catheter and surgical ablation of atrial fibrillation. *Heart Rhythm*. 2017;14.
9. Chuang DY, Sundararajan S, Sundararajan VA, Feldman DI, Xiong W. Accidental Air Embolism. *Stroke*. 2019;50.