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PUS IN THE PERICARDIUM: A PERICARDIAL-OESOPHAGEAL FISTULA WITH CARDIAC TAMPONADE AFTER ABLATION FOR ATRIAL FIBRILLATION

Taha Mansoor[1], Nahal Massoudi[1], Anandbir Bath[2], Matthew Kelly[1], Santhosh Koshy[1,2], Armand Tanase[1,2]

[1] Western Michigan University Homer Stryker M.D. School of Medicine, Kalamazoo, USA
[2] Ascension Borgess Hospital, Kalamazoo, USA

Corresponding author's e-mail: taha.mansoor@wmed.edu

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ABSTRACT

Background: Pericardial/atrial oesophageal fistula (PEF/AEF) is the most feared complication of cardiac atrial fibrillation (AF) ablation. Without urgent surgical treatment, most cases of this infrequent complication rapidly prove fatal.

Case report: We present an uncommon case of a 60-year-old male who had undergone an AF ablation 2 weeks prior and presented with chest pain. Following diagnosis and treatment of ST-elevation myocardial infarction (STEMI), a small pericardial effusion was noticed on computed tomography (CT) scan. Despite an initially reassuring hospital course, rapid decompensation and expansion of his purulent pericardial effusion led to his death within 24 hours of presentation. A post-mortem review of his CT imaging revealed an inconspicuous PEF.

Conclusions: It is essential to maintain a high suspicion of PEF/AEF in patients presenting after AF ablation with suggestive symptoms, such as fever, chest pain/odynophagia, and neurological deficits. CT chest is the most common diagnostic modality and surgical correction is the treatment of choice.

KEYWORDS

Pericardial-oesophageal fistula, atrial-pericardial fistula, ablation, atrial fibrillation

LEARNING POINTS

- Pericardial/atrial oesophageal fistula is a devastating complication of atrial fibrillation ablation and early detection is the keystone of effective management.
- Urgent surgical repair is the most effective treatment to prevent mortality.
- Preventative measures include power titration, limiting energy delivery times, and avoiding overlapping ablation lines during the ablation procedure.

INTRODUCTION

In patients with atrial fibrillation (AF) ablation using either radiofrequency, cryoballoon, or pulsed-field ablations electrically isolates the pulmonary veins from the left atrium. Radiofrequency ablation uses heat to create small point lesions to block the transmission of abnormal conductive

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signals, whereas cryoballoon ablation freezes and disables heart tissue. Pulsed-field ablation is a newer technology that uses highly specific short bursts of high-voltage pulses, leading to target cell death. A pericardial/atrial oesophageal fistula (PEF/AEF) is an abnormal connection between the oesophagus and pericardium/atria that can form after AF ablation secondary to surrounding tissue damage.

CASE DESCRIPTION

A 60-year-old male presented with severe, substernal chest pain radiating to his upper back accompanied by diaphoresis. This pain had recurred following resolution the night prior. Nitroglycerin did not relieve the pain and there was no associated shortness of breath, coughing, or vomiting. Physical examination revealed an alert and oriented male in severe pain with non-laboured breathing, adequate peripheral perfusion, and absence of peripheral oedema or jugular venous distension. The patient's vital signs at presentation were within normal limits.

The patient had a medical history of AF and an aortic aneurysm. Various treatment options had been attempted for his AF including rate control, rhythm control with amiodarone as well as five direct current cardioversions and AF ablations including pulmonary venous isolation/cavo-tricuspid isthmus ablation the year prior. Most notably, the patient had a repeat radiofrequency ablation 2 weeks prior, with a maximum power of 50W for approximately 10 seconds for each application on the anterior wall of the left atrium around the superior and inferior pulmonary veins and at the carina between the superior and inferior pulmonary veins. Because of recurrent AF and repeat ablation, linear ablation was also performed to create a roofline between the left and right superior pulmonary veins as well as the left and right inferior pulmonary veins. Further ablation was done inside the posterior box for isolation. An electrocardiogram (ECG) during the hospital admission showed ST segment elevations in leads I, II, III, aVL and aVF. Primary lab investigations were unremarkable without initial troponin elevation. However, with the patient's presenting symptoms and ECG findings, there was reasonable suspicion of a ST-elevation myocardial infarction (STEMI). Subsequent coronary angiography showed a 70% stenosis with a fractional flow reserve of 0.92 in the midsegment of the left anterior descending artery (LAD). Additional findings during the catheterization procedure included normal left ventricular wall motion with an ejection fraction of 55% and the absence of signs of aortic dissection on ascending aortic angiography. A drug-eluting stent was placed across the lesion in the midsegment of the LAD. Final angiography showed thrombolysis in myocardial infarction (TIMI) 3 flow with 0% residual stenosis and a well-apposed stent without evidence of dissection or perforation. Despite percutaneous coronary intervention (PCI), the patient's chest pain remained unchanged, with suspicion of acute pericarditis or acute aortic dissection. CT angiography demonstrated a small hyperdense pericardial effusion without evidence of acute aortic syndrome. A transthoracic echocardiogram (TTE) displayed normal global systolic function with a trivial pericardial effusion but did not show evidence of tamponade so pharmacotherapy for acute pericarditis was initiated with a plan for repeat TTE the following day. The patient reported improvement in his chest pain with a gastrointestinal cocktail, providing further reassurance against a cardiac aetiology.

In the following hours, the patient deteriorated with concern for evolving septic shock. He demonstrated considerable respiratory distress, chest pain resurgence, and hemodynamic instability with worsening leucocytosis, lactic acidosis, and elevation of inflammatory markers. ECG demonstrated inferior lead ST elevations, and emergent TTE showed a remarkable increase in his pericardial effusion size from before.

Following subsequent transfer to the catheterization laboratory, the patient had further decompensation, leading to asystole and initiation of cardiopulmonary resuscitation. Simultaneous echocardiograms showed further increasing pericardial effusion with resulting cardiac



Figure 1. Computed tomography angiography of the chest, abdomen, and pelvis revealed small pockets of air in the pericardium raising the possibility of a fistula from the oesophagus to the pericardium.

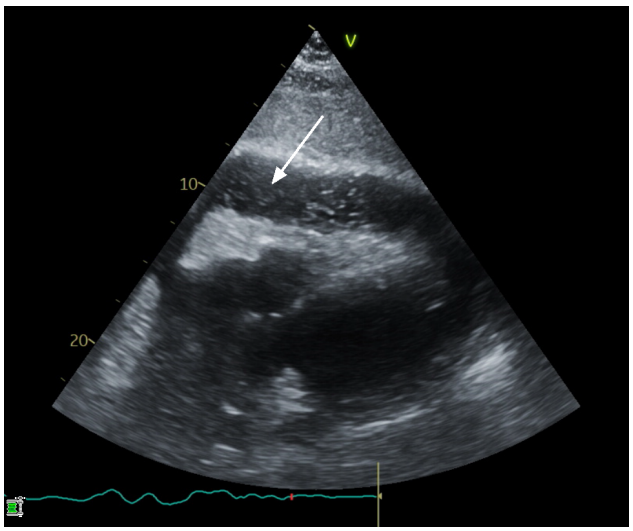


Figure 2. Pericardial space with coagulum (white arrow), likely pus, leading to cardiac tamponade.

tamponade. Pericardiocentesis resulted in 400 ml of a thick yellow pus-like drainage without blood, but despite temporary improvement, multiple resuscitation events were unsuccessful, with ensuing patient death. Post-mortem analysis of the earlier CT chest showed an inconspicuous tract spanning from the oesophagus to the heart consistent with a PEF, almost certainly secondary to the ablation 2 weeks prior *(Fig. 1 and 2)*.

DISCUSSION

We describe a case of a middle-aged male who presented with a STEMI 2 weeks after repeat AF ablation with subsequent rapid decompensation and a post-mortem diagnosis of PEF. The presenting lateral and inferior STEMI may have been secondary to air embolism as a complication of PEF, although this was not definitively diagnosed.

EAF after AF ablation has been found to occur more often after radiofrequency ablation than after cryoenergy ablation[1]. Following AF ablation, the median time to symptoms is 18 days, and fever, chest pain/odynophagia, and neurological symptoms are the key initial symptoms[1]. CT scan of the chest is the most common diagnostic modality, with indicative imaging results including oesophageal opacification and air in the left atrium[1,2]. Surgical correction of the fistula has demonstrated far superior outcomes to conservative management[1,2].

Power titration during pulmonary vein isolation, limiting energy delivery time, avoiding overlapping ablation lines, and monitoring intraluminal oesophageal temperature are suggested preventative measures[3]. Other recommendations include limiting the power use to 30W with irrigated radiofrequency and avoiding excess tissue contact by moving the ablation catheter every 15 to 20 seconds during the ablation especially when close to the left atrial posterior wall[4]. Additionally, conscious sedation instead of general anaesthesia during ablation procedures may allow for greater pain perception and prevention of an oesophageal injury[3]. Patient education about the possible signs and symptoms of an oesophageal injury is also crucial to encourage them to seek timely care if required[3]. Air embolism, a devastating complication of AEF, may be prevented with Durant's manoeuvre, and upper endoscopy is contraindicated if there is any suspicion of AEF due to the risk of air or bacterial embolization[5].

To conclude, although PEF/AEF can masquerade as other, more common diagnoses, high suspicion should be maintained for a timely diagnosis of post-ablation AF patients.

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