CASE REPORT

A rare complication following radiofrequency ablation

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SUMMARY

Atrial-oesophageal fistula (AOF) formation is a rare but often fatal complication post radio frequency ablation (RFA). Mortality ranges from 67% to 100%, with a rapid progression from symptom onset to death. We report a case of a healthy man in his early 40s who presented with a Glasgow Coma Scale of 5/15, clinical evidence of sepsis and *Streptococcus viridans* bacteraemia, 14 days following uncomplicated RFA for atrial fibrillation. Establishing a diagnosis of AOF can be difficult, as patients may have bacteraemia, but are consequently misdiagnosed with infective endocarditis, as in this case. One should have a high-index of suspicion for AOF in patients presenting with the aforementioned constellation of symptoms following ablation. There are no established predictors of mortality, but prompt detection, emergent operative intervention and prolonged antibiotic therapy are vital for survival.

BACKGROUND

An atrial-oesophageal fistula (AOF) is a rare but known complication of cardiac radiofrequency ablation (RFA) procedures,¹ first reported in 2001.² The incidence ranges from 0.01% to 0.2%,¹ with an associated mortality of 67–100%.³ This high mortality associated with AOF relates to its late presentation, the lack of awareness among clinicians and its complicated management.⁴ Thermal injury to the oesophagus is a known complication of RFA and is secondary to the close vicinity of the posterior left atrial wall to the oesophagus. Autopsy studies have confirmed a distance often as small as 5 mm.⁵ As much as 47% of patients demonstrate macroscopic oesophageal wall injury (erythema, tissue necrosis and ulceration) following ablation, but most exhibit no sequelae.⁶

CASE PRESENTATION

A man in his early 40s presented to the emergency department unresponsive following an episode of faeculent vomiting. Upon admission, his Glasgow Coma Scale was 5/15 and he was immediately intubated and ventilated. He was febrile at 38.3°C, with a pulse of 140 bpm and a blood pressure of 124/62. His only medical history was that 14 days back he underwent pulmonary vein isolation for paroxysmal atrial fibrillation, which took place without incident. He had been well until this sudden deterioration.

INVESTIGATIONS

C reactive protein was 226 and he was treated for sepsis, possibly of abdominal origin.

Electrocardiography demonstrated a regular narrow complex tachycardia and his troponin-I assay returned at 1.53. A CT scan of his brain demonstrated a small peripheral right frontoparietal infarct, which was considered too small to account for his obtunded state. Transthoracic echocardiography revealed good left ventricular systolic function with delayed relaxation of the left ventricle. An abnormal structure was noted in the inferior vena cava (IVC), thought to be either vegetation or a clot. *Streptococcus viridans* grew in two sets of blood culture bottles and he was treated for several days with amoxicillin and gentamycin for suspected subacute bacterial endocarditis, without any clinical improvement.

He subsequently developed haematemesis but an oesophagogastroduodenoscopy did not show any pathology. The cardiologist involved questioned the diagnosis of infective endocarditis and a CT pulmonary angiogram was organised to delineate the IVC structure more clearly. A repeat CT brain was also organised. No evidence of a thrombus was seen in the IVC, but a 13 mm collection of air was noted within the left atrium (LA) (figure 1). Air was also seen in several areas of the brain along with multiple areas of haemorrhagic infarction, the largest measuring 15 mm in diameter (figure 2). These were sequelae of an atrial-oesophageal fistula, post ablation.

OUTCOME AND FOLLOW-UP

He was transferred to a specialist centre for surgical intervention, but unfortunately did not survive.

DISCUSSION

AOF formation typically occurs 2–41 days postablation.³ Clinical presentation includes fever, haematemesis, odynophagia and manifestations of cerebrovascular events secondary to air or food embolism.⁷ Neurological impairment typically manifests in the second to third week post ablation as in this case. Survivors often suffer permanent disability.³ Risk factors for oesophageal damage during RFA include intraluminal temperature rise to greater than 40°C, power more than 30 W, a reduced LA-to-oesophagus distance, and the use of general anaesthesia.⁸

The isolation of *S viridans* species in the present case led to the preliminary diagnosis of infective endocarditis; however, the incidence of infective endocarditis post ablation is low, only 0.2% at one large centre.⁹ Interestingly, bacteraemia is a rare manifestation of AOF, typically caused by oropharyngeal organisms.³ These organisms have most

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Figure 1 CT chest: 13 mm collection of air in posterior left atrium (black arrow).

likely disseminated from the proximal gastrointestinal tract through the AOF, as mucosal damage originates in the oesophagus and progresses to the atrium¹⁰ and intestinal mucosal tissue is more susceptible to ablation-induced thermal injury than muscle tissue, as demonstrated in animal studies.¹¹

Non-specific indicators of AOF include elevated troponin and inflammatory markers.^{7, 4} Transthoracic echocardiography has an important role in excluding endocarditis. Thoracic or cardiac CT remains the most reliable tool in AOF diagnosis, demonstrating pneumomediastinum or intraatrial air.⁴ One can see the merit in serial imaging as the admission CT brain was drastically different from the scan performed several days later. Gastroscopy and transoesophageal echocardiography are contraindicated as air insufflation can result in lethal systemic air embolism.⁶ Proposed measures during RFA to reduce the risk of AOF formation include intraluminal oesophageal temperature monitoring, reduction of power and duration of RF energy at the posterior left atrial wall, and tagging and real-time

Figure 2 CT brain: multiple collections of air and small haemorrhages (white arrows).

visualisation of the oesophagus to minimise RF application at points where it lies close to the posterior left atrial wall.¹² The treatment for AOF is surgical, with only one reported case of oesophageal stenting.¹³ Oesophageal resection with end-to-end anastomoses and primary pericardial patching are usually required.¹⁴ In patients presenting with the aforementioned constellation of symptoms following ablation, one should have a high-suspicion for AOF as an early treatment may prove life-saving.

Learning points

- ▶ Atrial-oesophageal fistula (AOF) is a rare but frequently fatal complication of radiofrequency ablation (RFA), with rapid progression from symptom onset to death.
- ▶ One should have a high suspicion for AOF in patients presenting with new neurological deficit following RFA.
- ▶ Prompt detection and emergent surgical intervention is crucial for survival.

Competing interests None.

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