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Fatal esophageal–pericardial fistula as a complication of radiofrequency catheter ablation

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Abstract

The clinical role of catheter ablation using radiofrequency or cryothermal energy has become an important therapy in the management of patients with recurrent or persistent tachyarrhythmia that is refractory to medical therapy. It is regarded as a safe and reliable procedure and is performed routinely in health care facilities across the country. Like all procedures, there are associated risks and benefits. Development of an esophageal–atrial fistula is a rare but often-fatal complication of radiofrequency ablation. It is the second most frequent cause of death caused by the procedure, with mortality rates in excess of 70%. Death usually occurs as a result of cerebral or myocardial air embolism, endocarditis, massive gastrointestinal bleeding, and/or septic shock. Electrophysiologists have instituted a number of safeguard techniques to diminish the risk of developing esophageal–atrial fistula. Despite these measurements, instances of fistulous development still occur. Herein, we report a case of a 74-year-old male who presented with chest pain secondary to esophageal–pericardial fistula 19 days after pulmonary vein isolation using radiofrequency energy for atrial fibrillation in order to illustrate the clinical variability and diagnostic challenges associated with this dreaded gastrointestinal complication.

Keywords

Radiofrequency catheter ablation, atrial fibrillation, atrial flutter, esophageal–pericardial fistula

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Introduction

The clinical presentation of esophageal injury is extremely variable and often vague. Presenting symptoms such as chest pain, hematemesis, heartburn, dysphagia, and odynophagia often require additional workup that can delay diagnosis. The time to symptom onset appears to be within the first 3–35 days following the ablation, with sudden-onset chest pain and fever being the most common unifying symptoms among case reports.[1,2] We report a patient who presented 19 days following radiofrequency ablation (RFA) with leukocytosis, odynophagia, and esophageal perforation despite the appropriate use of real-time temperature and contact force monitoring. His clinical course was complicated by fatal innumerable acute bilateral cerebral and cerebellar infarctions secondary to paradoxical septic emboli from a peripherally inserted central catheter (PICC) and right atrial thrombus.

Case history

A 74-year-old male was evaluated in our cardiology department for recurrent paroxysmal atrial fibrillation. The RFA was performed using the CARTO three-dimensional (3D) mapping system, Lasso catheter was used for pulmonary vein localization, and a THERMOCOOL (Biosense Webster) 3.5 mm irrigated ablation catheter was used for segmental pulmonary vein isolation (PVI), without any additional substrate linear lesions performed. The patient initially discharged after the procedure with no acute complication. He was presented to our emergency department 19 days later complaining of chest discomfort. His chest pain was initially

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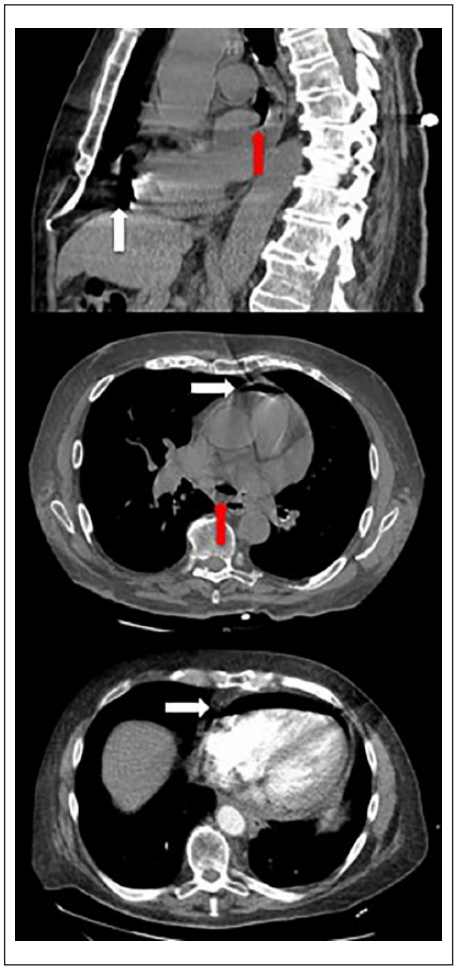

Figure 1. Computed tomography revealed air adjacent to the mid-esophagus (red arrow) and in the anterior pericardial space (white arrow) with a small amount of pericardial fluid consistent with esophageal perforation.

mild; however, 3 h later, he had abrupt onset of severe, substernal chest pain complicated by hemodynamic instability. His vital signs on presentation were temperature 99.3 F, pulse 54 bpm, respiratory rate 22 bpm, blood pressure (BP) 78/45 mmHg, and oxygen saturation 95% on room air. An emergent bedside echocardiogram was performed, which revealed a moderate-size pericardial effusion with no evidence of cardiac tamponade physiology. The patient was taken for a stat computed tomography (CT) scan of the thorax and abdomen after stabilizing him hemodynamically; which revealed features consistent with esophageal perforation (Figure 1). The patient was started on clindamycin and given fresh frozen plasma due to a supra-therapeutic international normalized ratio (INR).

A multidisciplinary team evaluated the patient, and a decision was to immediately transfer him to the operating room where he underwent a right thoracotomy with exploration of the mediastinum, drainage of the pericardial effusion, and an intraoperative esophagogastroduodenoscopy (EGD), which was equivocal. A nasogastric tube was placed in the esophagus, and under fluoroscopic visualization, barium was injected with no active extravasation of contrast noticed as well.

The pericardium was opened anterior to the phrenic nerve, and approximately 200–300cc of purulent fluid mixed with air bubbles escaped through the opening of the pericardium. At this point, it was felt that the most likely mechanism to explain our patient's clinical condition was thermal injury to the mid-esophagus due to the recent RFA within the left atrium. The resultant perforation and esophageal–pericardial fistula formation allowed esophageal contents (air and fluid) to escape into the pericardial space and cause a purulent pericarditis to develop. According to the cardiothoracic surgeon, the perforation had subsequently sealed and was no longer open and active. Surgical clips were placed over the area of suspected perforation.

Postoperatively, the patient was managed in the cardiovascular intensive care unit through a multidisciplinary approach with general supportive measures, intravenous (IV) antibiotics and chest tube drainage. His postoperative course was complicated by atrial fibrillation with rapid ventricular response (RVR) and periods of hemodynamic instability. While thoracotomy fluid remained culture negative, pericardial biopsies grew *Peptostreptococcus* and *Streptococcus oralis*. He was eventually discharged with a right upper extremity PICC for continuation of antibiotic therapy with IV ceftriaxone 2 g daily based on proven culture sensitivities.

Eight days following his discharge (18 days post thoracotomy), the patient was brought back to the emergency department unresponsive. A magnetic resonance imaging (MRI) of his brain revealed innumerable acute bilateral cerebral and cerebellar infarctions suggesting possible septic emboli (Figure 2). The patient was intubated, started on broad-spectrum antibiotics, vasopressors, and supportive care measures, which yielded only marginal clinical improvement. The patient was transferred to hospice care where he succumbed to multi-organ failure. Autopsy later revealed a right atrial thrombus leading us to believe the septic emboli were dislodged from an infected PICC line that had been previously withdrawn 1 h prior to his clinical deterioration.

Discussion

The clinical role of catheter ablation using radiofrequency (RFA) or cryothermal energy has become an important therapy in the management of patients with various types of tachyarrhythmia. The indications for this modality generally revolve around the treatment of a recurrent or persistent symptomatic arrhythmia that has been refractory to medical therapy. The overall incidence of peri-procedural complications following catheter ablation is approximately 3%, which includes but not limited to death (0.1%–0.3%), heart block (1%–2%), thromboembolism (<1%), cardiac trauma (1%–2%), and vascular access complications (2%–4%).[3]

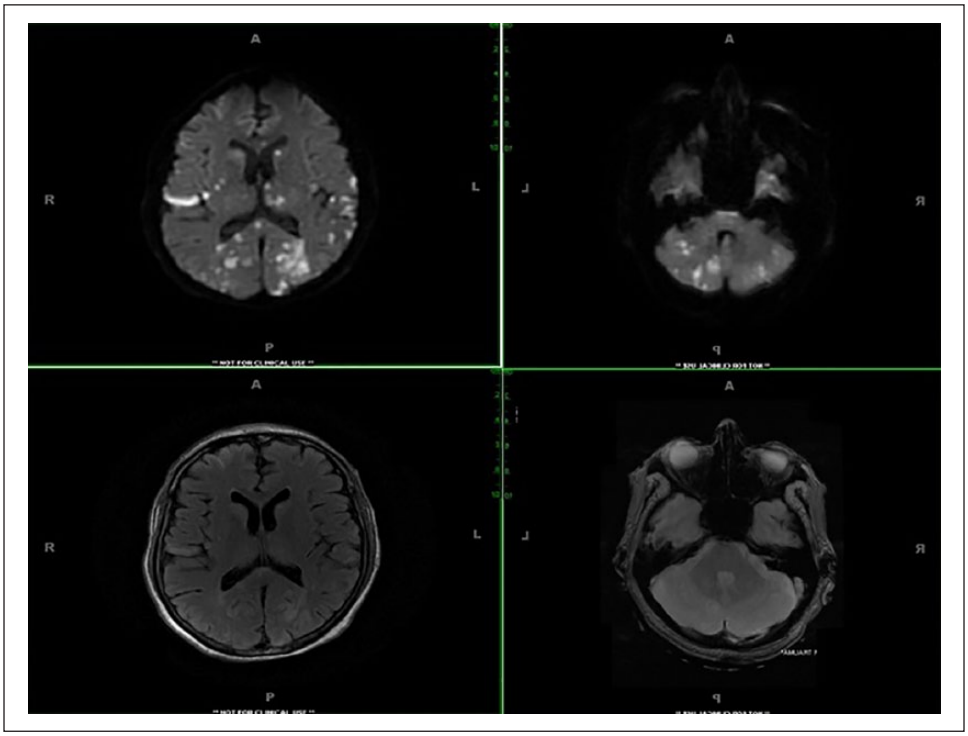

Figure 2. Diffusion-weighted sequence MRI reveals innumerable diffusion defects involving bilateral cerebrum and cerebellum.

Esophageal–atrial fistula is considered the second most frequent cause of death that may be caused by RFA with mortality rates in excess of 70%. A high degree of suspicion must be maintained for esophageal injury after RAF, particularly in patients with classic symptoms reported 3–35 days following the procedure. Computed tomography (CT) scan of the chest demonstrating the presence of pneumomediastinum or intra-atrial air is the most reliable diagnostic test, especially in unstable patients.[2]

The search to identify predisposing factors related to esophageal–atrial (and pericardial) fistulas after RFA has prompted a close examination of anatomic variability within the heart and the esophagus in relationship to the lesions that are created. It is important to note that while the temperature, catheter tip contact force, and RFA energy duration can be objectively measured in real time, fistulous lesions still occur within the confines of scientifically acceptable ranges. The depth of the lesions created remains unpredictable, and may be more inherent to the patient's anatomy overall.[1]

General discussion of RFA focuses on the posterior left atrial wall as a highly vulnerable region to RF energy. However, fistulous complications are documented when PVI is performed without targeting the posterior left atrial wall with linear or crossing ablation lines, as was the case with our patient.[4,5] The proximity of the esophagus to the posterior aspect of the left atrium is well documented with the length of the esophagus in contact with the posterior left atrial wall roughly 30–53 mm and the width between 9 and 15 mm.[6] Owing to displacement by the aortic arch, normal variants of the esophagus may place its course closer to the left or the right pulmonary veins. Cadaveric studies have shown that the left atrial wall is thinnest near the pulmonary vein orifices, lending credence to the idea that PVI alone may confer a more dangerous energy transfer to a variant esophagus coursing near these structures.[6,7] Currently, there is no real-time application for measuring left atrial wall thickness to guide ablation, and it remains to be seen if incorporating this measurement could reliably ensure the depth and safety of ablative lesions.[8]

Thermal insult to the microvasculature of esophageal tissues is believed to be the instigating mechanism of fistula formation, with ischemic necrosis extending toward the pericardium and left atrial wall.[9,10] This concept has prompted the use of lower esophageal temperature (LET) monitoring as a strategy to mitigate excessive energy transfer during RFA. Studies have suggested that variability in probe placement, and the position of the probe within the lumen, away from the esophageal wall, makes this technique an inaccurate surrogate for the heating of the intramural tissues.[7,9] Currently, no safe method of intramural esophageal temperature monitoring exists that has been incorporated into standard practice. Data compiled by Sause et al. regarding LET monitoring caution that temperatures over 41 C may increase the incidence of esophageal ulcer formation, suggesting a theoretical upper limit to mucosal heat tolerance.[11] However, it is important to note that many ablative temperatures reported travel above this threshold, and no ideal temperature range has been firmly established in the literature. We

Table 1. Our patient's radiofrequency ablation data.

Ablation session	Time[a] (min)	Power (W)			Temperature (°C)			Impedance		Force (*g*)		
		Min	Max	Avg	Min	Max	Avg		Change	Min	Max	Avg
1	3:25	25	25	25	31	39	35.6	147	−8	0	67	10
2	1:01	25	25	25	33	38	34.8	142	−2	0	42	9
3	4:46	25	25	25	33	36	34.2	149	−6	0	57	6
4	3:11	25	25	25	33	36	34.8	131	−4	0	32	6
5	3:49	25	30	28	33	40	35.8	125	8	1	74	15
6	1:56	30	30	30	33	38	36	123	−8	1	55	13
7	1:14	30	30	30	34	40	36.6	122	7	1	51	12
8	1:23	30	30	30	35	40	36.8	135	−13	0	38	10
9	1:39	30	30	30	34	41	37.4	130	15	1	21	7
10	0:44	30	30	30	34	36	34.6	119	4	1	34	4
11	5:47	25	25	25	33	42	36.1	149	−15	0	90	15
12	7:36	25	25	25	33	38	34.8	148	−10	1	100	20
13	16:39	25	25	25	33	42	35.9	132	−13	0	63	13
14	2:28	25	25	25	32	42	35.4	144	−15	1	57	18

[a]Time represents duration of RF-on delivered at targeted sites.

recorded maximal temperatures of 41 C–42 C on 4 of the 14 ablative sessions for our patient; however, average temperature never exceeded 37.4 C during any individual RFA energy application (Table 1).

Measuring the applied force of the catheter tip allows operators to ensure consistent contact with their ablative targets. A higher proportion of incomplete ablations have been noted when a contact force of less than 10g was applied through the catheter tip for less than 90% of the ablation session.[12] With a focused intent on creating optimal lesions, operators may be applying more pressure, for longer durations than those without force sensing catheters.[13] In our patient, no power greater than 30 W was used, and the average contact force did not exceed 20*g* during any single ablative session (Table 1). Anecdotal evidence implicating increased contact force with fistula development is lacking; likely due to the low incidence rate. However, in vitro studies using canine muscle demonstrated that larger and deeper lesions were created by lower RFA power and greater contact force, as compared with lesions produced at high power but lower contact force.[14] In areas where the left atrial wall is thinnest, increased contact force even in the setting of power modulation may provide the impetus for esophageal damage.

Conclusion

Esophageal–atrial fistulas remain the most dreaded complication of RFA procedures. By implementing real-time monitoring techniques, we have developed a better understanding of anatomy and tissue tolerance to variables such as temperature and contact force. Despite these measurements, instances of fistulae development still occur. By reporting this case with objective clinical data, we hope to contribute to the global understanding of these parameters in relationship to esophageal injury and fistulous propagation.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethical approval

Our institution does not require ethical approval for reporting individual cases or case series.

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Informed consent

Written informed consent was obtained from the patients' legally authorized representative (his spouse) for his anonymized information to be published in this article.

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