Atrial-Esophageal Fistula after Atrial Radiofrequency Catheter Ablation

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Atrial-esophageal fistula is a rare but often fatal complication of catheter radiofrequency ablation. Patients occasionally have bacteremia and have been misdiagnosed with endocarditis. Infectious diseases specialists are often consulted and need to be aware of this complication. We report a case of atrial-esophageal fistula after radiofrequency ablation that illustrates the salient features of this illness.

Radiofrequency ablation for drug-refractory atrial fibrillation is being more commonly performed both by cardiac surgeons and by electrophysiologists. As a result, complications from this procedure are being more frequently reported. Since 2004, atrial-esophageal fistulas (AEF) with high mortality rates have been reported with an increasing incidence after both surgical and percutaneous radiofrequency ablations. We present a case of AEF that illustrates the salient features of this illness.

Case report. A previously healthy, 46-year-old white man with pectus deformity of the chest and a 10-year history of atrial fibrillation presented with fever, rigors, and near syncope. One month earlier, he had undergone an atrial radiofrequency ablation for symptomatic atrial fibrillation refractory to medical management. A wide area circumferential ablation encircling the 4 pulmonary ostia was performed.

Three days after the ablation, he had a single episode of fever (temperature, 39°C) associated with a rigor. Subsequently, all symptoms resolved. Two weeks later, he underwent a routine dental cleaning, and 10 days later developed fever, violent shaking chills, and a near-syncopal episode for which he sought medical care at a local hospital. He was febrile (temperature, 40°C) and hypotensive (blood pressure, 80/60 mm Hg), with an irregular heart rate of 120 beats/min. He was noted to be confused, with myoclonic jerking of both lower extremities and bilateral positive Babinski signs. Atrial fibrillation was confirmed by electrocardiography. His leukocyte count was 2500 cells/µL, with neutrophilic predominance. Findings of chest radiograph were unremarkable, and a transthoracic echocardiogram showed no thrombus or vegetation. He was given empirical treatment with piperacillin/tazobactam and levofloxacin, which resulted in resolution of his fever and improvement in his mentation. Findings of computed tomography (CT) imaging of his head were unremarkable, as were findings of magnetic resonance (MR) imaging of his cervical spine. Two sets of blood cultures revealed growth of Streptococcus mitis, Streptococcus sanguis, and a nonenterococcal group D Streptococcus species. He was transferred to our institution for further care.

At admission, the patient was afebrile, and physical examination revealed persistent atrial fibrillation. Laboratory data were unremarkable, and therapy with ceftriaxone was initiated. Findings of a transthoracic echocardiogram and CT scans of the chest were unremarkable. Blood cultures remained negative, and the patient was discharged 4 days later to complete a 4-week course of ceftriaxone treatment for presumed endocarditis.

The following day, his temperature increased to 39°C, and shortly thereafter he developed sudden-onset right-sided hemiparesis. He was brought to the emergency department, at which time he was afebrile, but his blood leukocyte count was 19,000 cells/µL, with 92% neutrophilic predominance. MR imaging of the brain revealed multifocal infarcts. A CT scan of the chest showed a small 8-mm dependent air density in the left atrium but an esophagus with unremarkable appearance (Figure 1). He was emergently taken to cardiothoracic surgery, which revealed a fistula between the mid esophagus and the mid portion of the left atrium that was surgically repaired. His blood cultures remained sterile. He completed a 4-week course of ceftriaxone treatment and made a full recovery with no residual neurological deficits.

Discussion. Tamponade, presumably due to perforation into the pericardial space, has been the most commonly reported complication of percutaneous radiofrequency ablation, with incidence ranging from 0.6% to 1.3% [1–4]. Reports of AEF first appeared in the surgical literature in 2001 [5] and in the medical literature in 2004 [6]. Since then, there have been 49 reported cases of AEF occurring as complications of both surgical and percutaneous radiofrequency ablations [1, 5–22]. The incidence of AEF following percutaneous ablation has ranged from 0.01% to 0.2% [1, 6–10, 21], whereas it has been
Figure 1. Computed tomography scan showing an 8-mm pocket of air (black arrow) in the dependent portion of the left atrium adjacent to the esophagus.

as high as 1%–1.5% for patients undergoing surgical ablations [8, 10, 11], mirroring the rate of tamponade.

The clinical presentation frequently seen (Table 1) and the complexities of establishing the diagnosis of AEF are evident in our patient. The time from ablation to onset of symptoms has ranged from 2 to 41 days. Fever is one of the most common symptoms seen in patients with AEF, occurring as early as the third postprocedure day. Among those patients for whom symptoms were reported, fever was noted in 75% of the patients with AEF [1, 6, 8, 10–18, 20, 21–23]. However, fever is non-specific and can occur in patients undergoing ablations simply as a result of the inflammatory nature of the procedure. In one retrospective review, 8% of patients who underwent ablations developed fever within 24 h after the procedure [24]. As a result, early fever following ablations is often attributed to pericarditis or post-pericardiotomy syndrome, which was the initial diagnosis for several patients who were subsequently identified as having an AEF.

Neurological deficits were identified in 69% of patients with clinical signs reported [6, 8–18]. Symptoms have included mental status changes, transient ischemic attacks, stroke syndromes with hemiparesis, and seizures. Unfortunately, the neurologic deficits tend to be a late finding, occurring in the second to third week after ablation, and often leave those who survive with permanent disabilities. Other reported symptoms included chest pain, seen in 24% of patients, odynophagia, and hema-temesis. The development of hema-temesis often prompted esophagastroduodenoscopy, which had disastrous consequences. Of the 9 patients with AEF for whom esophagastroduodenoscopy was performed, 8 had immediate deterioration in their clinical status and eventually died [8, 10–13, 20, 21, 23], usually as a direct complication of air embolization from the esophagostroduodenoscopy. On the basis of these findings, invasive esophageal procedures should be contraindicated for patients suspected of having an AEF.

Leukocytosis is found in patients with AEF [8], although our patient presented with leukopenia, presumably secondary to severe bacterial sepsis. Bacteremia has not been frequently reported. Only 4 additional patients with AEF were reported to have bacteremic infections, occurring 3–5 weeks after their ablations. Two patients had isolated streptococcal bacteremia [6, 15], and 2 patients had polymicrobial bacteremia [14, 17] with streptococcal, Micrococcus, and Candida species. The isolation of streptococcal species often led to the diagnosis of endocarditis. However, the incidence of endocarditis after radiofrequency ablation is quite low, only 0.2% at one high-volume center [1].

Animal studies have demonstrated that intestinal mucosal tissue is more susceptible to radiofrequency ablation–induced thermal injury than is muscle tissue [11, 22]. A recent report has shown that mucosal damage originates in the esophagus and progresses to the atrium [22]. All the reported cases of bacteremia have been caused by organisms that originate in the oropharynx, which supports the concept of dissemination from the proximal gastrointestinal tract through the AEF.

Echocardiography has not been a useful diagnostic tool for patients with AEF. Transthoracic echocardiography did not identify an AEF in any patients for whom the procedure was performed. One patient underwent a nondiagnostic transesopha-

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<th>Measure</th>
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<tr>
<td>No. of patients</td>
<td>49</td>
</tr>
<tr>
<td>Time to diagnosis, days</td>
<td>3–41</td>
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<tr>
<td>Symptoms, no. of patients with symptom/no. of studies reporting symptom (%)</td>
<td></td>
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<tr>
<td>Neurologic symptoms</td>
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<tr>
<td>Fever</td>
<td>27/36 (75)</td>
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<tr>
<td>Sepsis</td>
<td>12/36 (33)</td>
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<td>Chest pain</td>
<td>10/36 (28)</td>
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<td>Leukocytosis</td>
<td>9/36 (25)</td>
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<td>No. of patients with bacteremia</td>
<td></td>
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<tr>
<td>Monomicrobial</td>
<td>2</td>
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<td>Polymicrobial</td>
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<td>No. (%) of patients who died</td>
<td>33 (67)</td>
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ageal echocardiogram and, fortunately, did not develop an air embolization [17].

Thoracic or cardiac CT scanning demonstrating the presence of pneumomediastinum or intra-atrial air has been the most reliable tool for the diagnosis of AEF [8]. CT scanning ultimately established the diagnosis of AEF in 89.5% of the patients who were studied using this modality. As in our case, one patient had an unremarkable initial CT scan [14], and one patient required 3 scans to establish the diagnosis [19].

Although our patient survived with no residual neurologic deficits, the mortality rate associated with AEF has been alarmingly high. Cummings et al [14] reported a mortality rate of 100% in their series of 9 patients. The overall mortality rate from all reported cases is 67%. There are no clear predictors of mortality, but early diagnosis, prompt surgical intervention, and prolonged antibiotic therapy are crucial for survival. All patients who did not undergo surgery died, except for one patient who was successfully treated with an esophageal stent [20]. Nevertheless, 40% of those who did have surgery died.

Electrophysiologists have implemented safeguards to diminish the risk of developing AEF. These have included administration of high-dose proton pump inhibitors before and after ablation, the use of electroanatomical and CT imaging of the left atrium and esophagus, monitoring of the posterior wall of the left atrium with intracardiac echocardiography during the ablation, use of esophageal temperature probes, use of smaller irrigated ablation catheters applying less power for shorter durations, and extensive patient education regarding signs and symptoms of esophageal injury [1, 4, 7, 21, 25]. These procedures have resulted in reduced incidences of AEF at several institutions, but, given that radiofrequency ablations are being performed for more indications at more institutions, it is likely that this complication will continue to occur.

Our patient demonstrates the complexities of establishing the diagnosis of AEF for patients who have recently undergone radiofrequency ablation. Although he was febrile and had polymicrobial bacteremia at presentation, he responded rapidly to antibiotics and findings of both transthoracic echocardiogram and CT scan were unremarkable. These findings led his physicians to discount the likelihood of an AEF in the differential diagnosis. Only when he developed a cerebrovascular accident did an additional CT scan show intra-atrial air, confirming the diagnosis and prompting immediate surgical intervention.

Infectious diseases specialists must have a high index of suspicion for this diagnosis for patients who develop postablation fever, chest discomfort, unexplained neurologic deficits, and occasionally bacteremia as long as 5 weeks after the procedure. These patients require urgent evaluation with CT or MR imaging but should never undergo transesophageal echocardiography or endoscopy, because of the potential for catastrophic outcomes.

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