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Complications of Radiofrequency Catheter Ablation for Atrial Fibrillation

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Abstract

Radiofrequency catheter ablation (RFA) has evolved as an effective treatment modality in patients with atrial fibrillation (AF). However, complication rates associated with RFA of AF have been cited in the range of 3.5 – 5% with the majority consisting of cardiac tamponade, vascular complications, and thromboembolic phenomena. In this review, the complications of AF ablation will be discussed along with associated clinical management strategies.

Introduction

Radiofrequency catheter ablation (RFA) has evolved as an effective treatment modality in patients with atrial fibrillation (AF). Despite advances in RFA techniques and technology, and attempts to increase awareness and optimize management of procedural complications, there has not been a substantial decrease in complication rates over the last decade. A recent survey conducted by Cappato et al that included 20,000 atrial fibrillation ablation procedures performed between 2003 and 2006 identified major complications in 4.5% of procedures; [1] this is not significantly decreased from the complication rate of 4.0% reported on a survey conducted by the same group between 1995 and 2002.[2] This lack of significant improvement is at least partly due to the higher proportion of older patients and those with significant comorbidities such as cardiomyopathy.[1] Overall, complication rates associated with RFA of AF have been cited in the range of 3.5 – 5%.[3,1,4] The distribution of complications has also remained similar over the last decade, with the majority consisting of cardiac tamponade, vascular complications, and thromboembolic phenomena.[3,1,4] In this report, the incidences and outcomes of major complications associated with catheter ablation of AF as well as potential strategies for their management will be reviewed.

Cardiac Tamponade

Perhaps the most significant of the bleeding complications associated with catheter ablation of AF in terms of frequency and morbidity is pericardial tamponade. This major complication has been reported in 0.5-2.4%[3,5,6] of cases. Although outcomes associated with pericardial tamponade when recognized and treated promptly are generally favorable, it remains the leading cause of mortality during catheter ablation of AF.[7,3] Predictors of cardiac tamponade are not clear; however, prior left atrial catheter ablation may be an independent risk factor.[3]

With a recent shift in periprocedural anticoagu-

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lation strategy toward performing procedures with a therapeutic INR, there has been a growing concern for a potential increase in rates of tamponade. However, the incidence of tamponade has not been shown to be elevated with this anticoagulation strategy.[8,9] Preliminary data also suggests that the severity of this complication and outcomes when it does manifest are also not heightened in the presence of therapeutic anticoagulation.[10]

The hallmarks to management of tamponade are prompt recognition and treatment. Hypotension is often the initial finding suggesting a possible significant perforation. This can be quickly verified looking at the lack of excursion of the cardiac silhouette in the left anterior oblique view fluoroscopically. An echocardiogram is helpful to confirm the effusion and guide emergent pericardiocentesis. Once a significant effusion is recognized, heparin is usually reversed using protamine. Patients therapeutic on warfarin may be considered for reversal with fresh frozen plasma or Factor VII complex, although it is not clear whether reversal of warfarin is associated with an improvement in clinical outcome. The majority of patients are successfully treated with percutaneous drainage and close inpatient monitoring. Patients with persistent accumulation of effusion or hemodynamic compromise not responding to initial drainage may require surgery.[5] Blood transfusion may also be required and autotransfusion has been reported as a safe option.[6]

A pericardial drain is usually left to dependent drainage with the patient monitored in an intensive care setting until drain output has decreased <30 cc in 24 hours. Repeat echocardiographic evaluation is often performed after temporarily occluding the drain for 6-12 hours. Due to the unpredictable level of anticoagulation, LMWH may not be preferable during reinitiation of anticoagulation in these patients. One approach is restarting warfarin prior to discharge. Since late recurrence of tamponade can occur, clinical follow-up with a repeat echocardiogram in 1-2 weeks may be appropriate.

Peripheral Vascular Complications

The most common peripheral vascular complications associated with catheter ablation are hematomas, pseudoaneurysms, and arteriovenous fistulas which are cumulatively reported in about 1-2% of cases.[3,11,1,4] Several studies have revealed that risk of vascular complications may be increased with female patients, older age, use of clopidogrel, and less experienced operators.[3,11,8,4] About half of pseudoaneurysms and fistulas ultimately require surgical intervention.[3]

Recent changes in anticoagulation protocols to perform RFA with a therapeutic INR have shown no increase in overall vascular complication incidence. In fact, minor bleeding complications may actually be decreased when the need for bridging with heparin or LMWH are avoided.[8]

Cerebrovascular Events

Recent studies have shown an overall incidence of cerebrovascular events including stroke and transient ischemic attack (TIA) ranging from 0.20% to 0.94%.[3,11,1,12] Several factors including chronicity of AF, left atrial size ≥ 4.5 cm, dislodgment of existing thrombus, and ablation-related char formation and tissue destruction can promote thrombogenicity.[13,14] Patients with CHADS2 scores ≥ 2 are five times more likely to experience a thromboembolic event when compared to those with a score of 0.[13]

Prior to the ablation procedure, patients should be maintained on anticoagulation if indicated by the American College of Cardiology/ American Heart Association/ European Society of Cardiology 2006 guidelines.[15] It is imperative that all patients receive anticoagulation for at least a period of 8 weeks post procedure regardless of their CHADS2 score due to the increased risk of thrombogenicity secondary to disruption of endothelial integrity during RF ablation and periprocedural cardioversion if performed.

During the ablation procedure, patients must be periodically assessed for evidence of neurologic abnormalities including asymmetric facial expression, slurred speech, and an inability to move extremities upon command. Asymptomatic "silent" cerebral embolism has been reported in >10% of patients after left atrial catheter ablation using diffusion-weighted magnetic resonance imaging despite rigorous periprocedural anticoagulation regimens.[16,17,18] The mechanism and clinical signifi-

cance of these silent emboli and how best to prevent them remain to be determined. Maintenance of ACT > 300 seconds and continuous infusion through the sheaths during the procedure have been suggested to reduce the incidence of thromboembolic events.[19]

Di Biase et al. performed a prospective trial to evaluate whether anticoagulation strategy had an effect on the overall rate of cerebrovascular events.[8] Patients who had a therapeutic INR during the ablation procedure had a lower rate of periprocedural stroke compared to patients who had discontinued warfarin prior to the procedure (0.9% vs 0.0%) despite having a higher proportion of chronic AF and increased CHADS2 scores. No increase in pericardial effusions or other bleeding complications were noted.

Those patients who show evidence of cerebrovascular complications should immediately be evaluated to determine if tissue plasminogen activator (TPA) is appropriate. If TPA is administered, clinicians should be aware that the risk of vascular complications is significantly increased.

Although the overall risk of cerebrovascular events is low, additional modification of ablation strategies is necessary to further reduce this complication rate. Strict maintenance of ACT >300 seconds, careful sheath management, minimizing char formation by discontinuing radiofrequency energy in the setting of abrupt impedance rises, and possibly performing RFA on anticoagulation may further decrease the risk of embolic events.[14]

Pulmonary Vein Stenosis

The incidence of pulmonary vein (PV) stenosis has significantly decreased in recent years due to improvements in ablation technique. With antral PV isolation, current studies cite an incidence of PV stenosis <0.01% to 0.04%[3,1,12]as compared to 5% when ostial PV isolation was routinely utilized.[20]Inflammation and smooth muscle constriction secondary to RFA are thought to be mediators of post procedure PV stenosis.[21] Location of ablation lesions in the PV ostia and increasing size of lesions are the primary risk factors in the development of PV stenosis.[14]

Late diagnosis of PV stenosis can be especially challenging as most primary care physicians are unaware of this complication. Patients may present with nonspecific symptoms such as dyspnea, chest pain, wheezing, and hemoptysis. Patients are

Table Potential Causes of Mortality Associated with Radiofrequency Atrial Fibrillation Ablation

Cardiac tamponade
Atrioesophageal fistula
Peripheral embolism
Stroke
Massive pneumonia
Myocardial Infarction
Intractable torsades de pointes
Septicemia
Sudden respiratory arrest
Extrapericardial pulmonary vein perforation
Occlusion of bilateral pulmonary veins
Hemothorax
Anaphylaxis
Asphyxia from tracheal compression secondary to subclavian hematoma
Intracranial bleeding
Acute respiratory distress syndrome
Esophageal perforation from intraoperative transesophageal echocardiography

often subjected to a multitude of unnecessary tests leading to misdiagnosis including pulmonary embolism, pneumonia, and lung carcinoma.[22] If a clinician suspects PV stenosis post ablation, multidetector computed tomography and magnetic resonance imaging are often used to establish a diagnosis. If patients are found to have severe stenosis with symptoms, most centers recommend angioplasty with or without stenting.

Total PV occlusion can be the result of a slow and insidious progression from a previous insignificant narrowing; therefore balloon angioplasty with or without stenting should be considered for any vein with a cumulative stenosis index (CSI) ≥ 75% regardless of symptomology (CSI = sum of percent stenosis of the unilateral veins divided by the total number of ipsilateral veins).[23] Because dilation of total PV occlusion is less successful, clinicians may consider angioplasty of severely stenosed PV regardless of symptomology in order to avoid progression to vein occlusion.[23]

Changes in ablation strategy have significantly reduced the incidence of PV stenosis in recent years. However, PV stenosis is a debilitating complication that must always be considered during the ablation procedure. Electrophysiologists must ensure that ablation lesions are delivered on the atrial side of the pulmonary veins avoiding the PV ostia. If severe flow limitations are noted in the setting of symptoms, balloon angioplasty with or without stenting should be performed to alleviate symptoms. Currently, there is no consensus regarding treatment of severe PV stenosis without symptoms; although stenting should be considered in order to avoid total occlusion. Many patients with single PV occlusion are asymptomatic and should undergo surveillance; however, patients with concomitant ipsilateral stenosis and a CSI ≥75% require intervention to restore flow in order to prevent associated lung disease.

Phrenic Nerve Injury

Phrenic nerve (PN) injury is a rare complication (<0.01%) of RFA due to its close proximity to common ablation sites.[3,11] The right PN courses laterally and slightly posteriorly to the superior vena cava and right atrium and descends anteriorly to the right superior PV, while the left PN tracks closely to the left atrial appendage.[24] The right PN has an especially close relationship to the right superior PV and is the most commonly injured phrenic nerve during RFA.[25]

High output pacing and monitoring of fluoroscopy during RFA are imperative to avoid permanent PN injury. However, PN injury secondary to RFA appears to have a benign prognosis and patients

usually experience improvement in function over time (1-2 years).[26]Phrenic nerve injury is uncommon when ablation is performed outside the PV.

Atrio-Esophageal Fistula

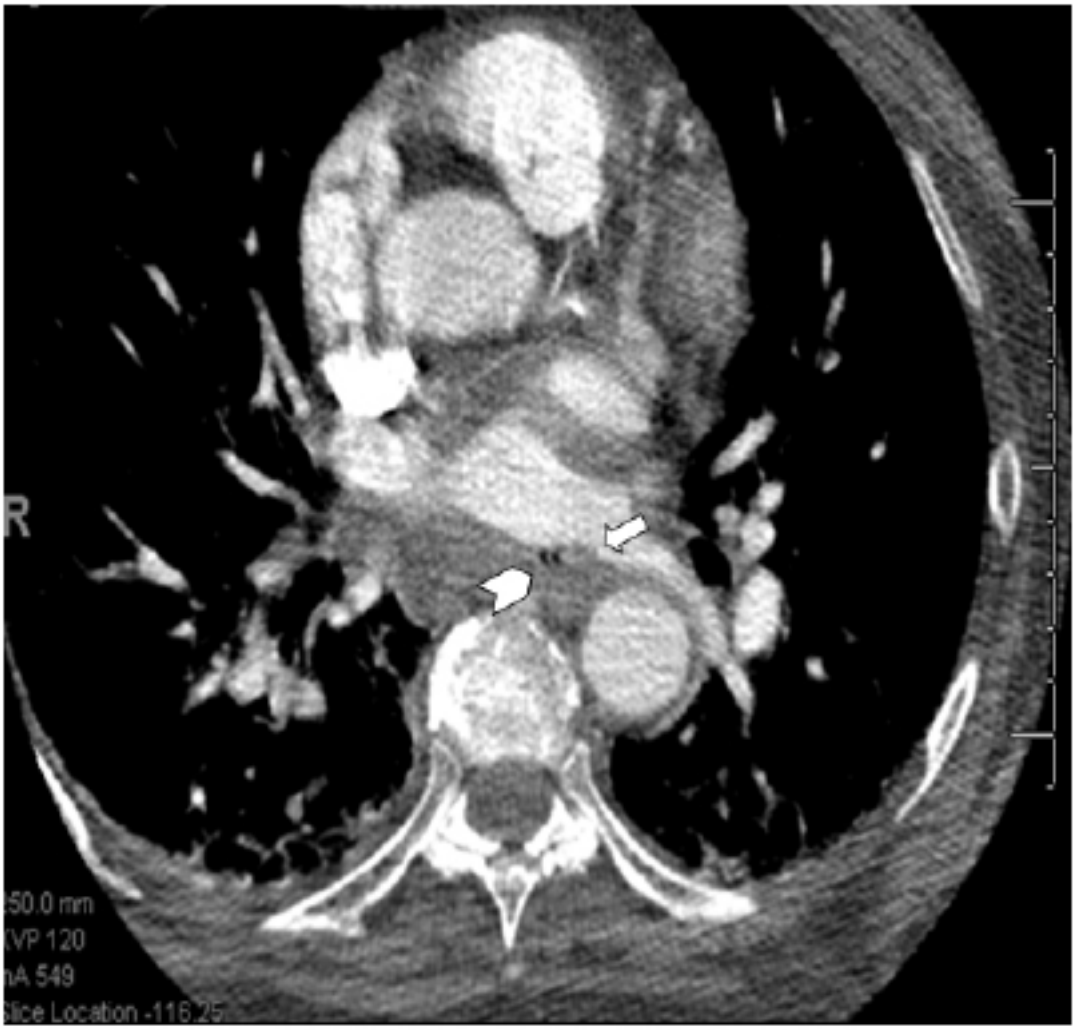
Atrio-esophageal fistula (AEF) is an infrequent (<0.3%) however often fatal complication of RFA.[3,1,27] Due to the associated devastating consequences and mortality rate >80%, great care must be taken to avoid this catastrophic complication.[27]

The esophagus is a mobile structure with fixed points in the pharynx and gastroesophageal junction. As a result, movement within the thorax is dynamic, especially with peristalsis.[28] Several approaches have been suggested to prevent esophageal injury. One approach is real-time imaging of the esophagus with barium swallow or an esophageal probe and avoidance of RF applications in the immediate proximity of the esophagus.[29]Monitoring of intraesophageal temperature and termination of RF application after a critical rise in intraluminal temperature increase has also been suggested. However, with this approach intramural temperatures cannot be measured, and the sensor probe may not be close to the ablation site. Therefore, lack of a temperature rise may give a false sense of security and fistula has been reported after RFA using an intraesophageal temperature monitoring system.[30] Others have suggested limiting power and duration of RF applications in the posterior left atrium. However, it is not clear whether critical power settings ensure safety. Despite these safeguards, >25% of patients will exhibit structural changes in the mediastinum via endoscopy.[31]Patients with persis-

tent AF, increased left atrial size, and ablation of the roof, coronary sinus, and mitral isthmus are more likely to develop esophageal ulceration.[32] The risk of atrioesophageal fistula may be higher during general anesthesia likely due to esophageal immobility.[33] Although acid reflux may develop after RFA [34], the role of prophylactic proton pump inhibitors in prevention of fistula is unknown. A simple measure may be to administer a proton pump inhibitor prior to and for 2-4 weeks after ablation.

Nonspecific symptoms may result in delayed diagnosis of AEF. Patients may present with acute pericarditis, pneumonitis, sepsis, seizures, strokes, gastrointestinal bleeding, and circulatory collapse due to the intense inflammatory response resulting from atrial and esophageal tissue necrosis.[14] Any patient presenting with a swallowing difficulty, fever, neurological deficit, or sepsis after ter RFA must immediately undergo evaluation for AEF. Imaging of the mediastinum with CT or MRI with or without barium swallow with specific attention to the presence of air in the mediastinum, and integrity of the atrioesophageal wall is very helpful for establishing the diagnosis (Figure). Esophageal instrumentation with TEE or endoscopy is often considered contraindicated due to the risk of introduction of air and potential for embolization. Treatment often involves surgical repair;

Figure : Magnetic resonance imaging of a 66 year old woman presenting with atrio-esophageal fistula four weeks after atrial fibrillation ablation.



Thick arrow indicated classical finding of posterior wall irregularity/indentation near the junction of the left superior pulmonary vein. Arrowhead demonstrates mediastinal air.

however, cases of stenting of the esophagus and keeping the patient NPO with appropriate antibiotic coverage have also been reported.[35] Surgical consultation must be obtained immediately.

Death

Death is an infrequent complication of RFA for AF occurring in 1 of 1,000 patients.[7] Operators must be aware of the causes of death (Table) and clinical decision making must be performed in order to decrease this risk whenever possible. The most common causes of mortality are tamponade with cardiac arrest (22%), atrioesophageal fistula (16%), stroke (9%), and massive pneumonia (6%).

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