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# Radiologic Manifestations of Pulmonary Vein Ablation Complications: A Pictorial Review

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# Radiologic Manifestations of Pulmonary Vein Ablation Complications: A Pictorial Review

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**Abstract:** In patients with atrial fibrillation refractory to drug therapy and cardioversion, pulmonary vein ablation is an alternative treatment that eradicates arrhythmogenic activity originating in the muscles of the pulmonary veins. While this procedure has a low incidence of significant complications, iatrogenic injuries are possible. Through multimodality pictorial examples utilizing computed tomography, nuclear medicine, fluoroscopy, and chest radiographs, the complications associated with pulmonary vein ablation will be reviewed. Examples of pulmonary vein stenosis, right phrenic nerve injury with associated diaphragmatic paralysis, atrioesophageal fistula, and pericardioesophageal fistula will be illustrated.

**Key Words:** pulmonary vein ablation, atrioesophageal fistula, phrenic nerve injury, pericardioesophageal fistula, pulmonary vein stenosis

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Atrial fibrillation is the most common cardiac arrhythmia and a significant source of morbidity and mortality in the United States. Defined as uncoordinated atrial activation that results in impaired mechanical atrial function, it ultimately originates from ectopic myocardial beats that either possess enhanced automaticity or an aberrant reentry circuit.[1] While it was first believed that the ectopic beats originated from within the atrial myocardium, research in 1998 demonstrated that the myocardial sleeves encasing the pulmonary veins were in fact an important source of ectopic beats, particularly in paroxysmal atrial fibrillation.[2] The sequelae of atrial fibrillation at a cellular level are severe; the longer atrial fibrillation goes untreated, the more likely atrial remodeling is to occur, which leads to scarring, irreversible atrial enlargement, impaired cardiac function, and an increased likelihood of stroke.[1]

The main clinical strategy for managing patients with atrial fibrillation is that of cardioversion by way of rate or rhythm control—namely utilizing medications to decrease the ventricular response rate and thereby preventing tachycardia-related cardiomyopathy. In patients with sustained paroxysmal atrial fibrillation refractory to pharmacologic treatment or electrical cardioversion, catheter ablation is considered an effective second-line therapy. The pulmonary vein ablation procedure specifically targets and eradicates the arrhythmogenic activity within the muscle sleeves of the pulmonary veins through application of either cryothermal energy or radiofrequency energy.[3] Radiofrequency energy yields myocardial ablation by conducting an alternating electrical current through myocardial tissue, thereby increasing tissue resistivity and generating heat, which conducts passively to deeper tissue layers. Tissues exposed to temperatures of > 50° C undergo irreversible coagulation necrosis. Cryoablation is a process that yields tissue injury by facilitating ice crystal formation within a cell, which disrupts the cell membrane, and thereby disrupts cellular metabolism and electrical activity. This is achieved by delivering liquid nitrous oxide under pressure through a catheter to its tip, where it changes to gas and cools the surrounding tissue.[4]

During both procedures electrode catheters are positioned at the site of the His bundle and distal coronary sinus to detect aberrant electrical activity. Subsequently, following transseptal puncture, mapping and ablation catheters are placed into pulmonary veins. Following anatomic mapping of the pulmonary veins, either radiofrequency energy or cryotherapy is applied 1 to 2 cm around each ostium of the right and left pulmonary veins. During radiofrequency ablation, a maximal power output of 70 W is applied for 20 to 40 seconds until the amplitude of aberrant activity displayed on the electrogram drops by 80%.[5] Notably, sometimes radiofrequency energy is also applied to the posterior wall of the left atrium. In cryoablation, freezing cycles at a temperature of −80°C are used for ablation, with confirmation of electrical isolation upon electrogram demonstration of sudden loss of electrical activity within the pulmonary veins compared with the left atrium.[6]

Catheter ablation is relatively effective, particularly in patients with paroxysmal atrial fibrillation, as opposed to persistent/longstanding atrial fibrillation. A worldwide survey reviewing outcomes of ablation cases performed between 2003 and 2006 demonstrated an 80% success rate after an average of 1.3 ablation procedures per patient.[4] Although widely regarded as a safe procedure, it can be associated with significant complications. The simple application of radiofrequency or cryotherapy could result in an excessive inflammatory response that may lead to extensive local scarring, and, ultimately, pulmonary vein stenosis. Furthermore, the close proximity of the esophagus, pericardium, right phrenic nerve, and left atrium to the pulmonary veins render them susceptible to direct injury (Fig. 1). Multiple studies have reported a 2% to 5% periprocedural complication rate, the majority of which are vascular in nature. The relatively more common injuries observed after pulmonary vein ablation include pulmonary vein stenosis, right phrenic nerve injury, and complications related to the site of vascular access. Rarer complications include esophageal injuries including both atrioesophageal fistula and pericardioesophageal fistula. Additional rare complications include tamponade, thromboembolic events, and lung or bronchial injury.

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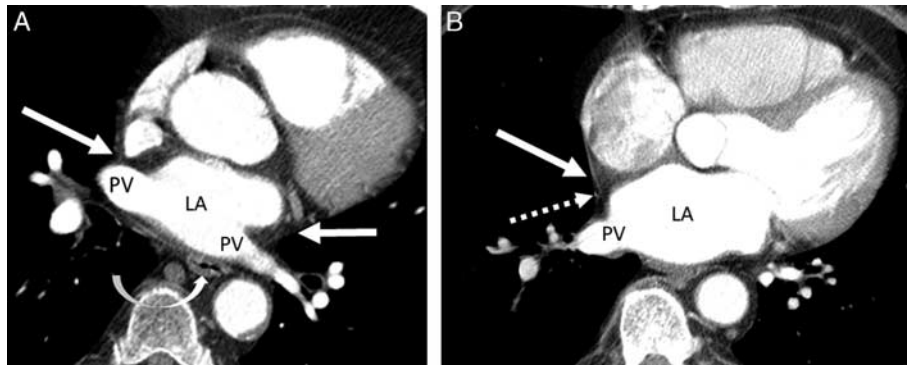


**FIGURE 1.** Axial contrast-enhanced CT images from preablation CT scans displays proximity of structures to the pulmonary vein (PV) ostia. The esophagus (curved arrow, A) commonly courses along the posterior aspect of the left superior PV, left atrial (LA) posterior wall, and left inferior PV. The right phrenic nerve (dashed arrow, B) is in close proximity to the anterior aspect of the right superior PV ostium. Pericardial reflections (solid arrows, A and B) insert into close proximity to the left and right PVs.

## PULMONARY VEIN STENOSIS

Radiofrequency ablation is a main cause of pulmonary vein stenosis, and it is postulated to result from a vascular and inflammatory response to radiofrequency energy that induces scarring and collagen deposition.[7] The published incidence of pulmonary vein stenosis following catheter ablation ranges from 0% to 38%, with an expert consensus review from the Heart Rhythm Society in 2007 reporting an overall incidence of <10%. While there have been case reports of pulmonary vein stenosis following cryoablation, it is much more rare. The symptoms of pulmonary vein stenosis can manifest as early as 1 to 2 days after the ablation.[8] Symptoms include dyspnea, chest pain, hemoptysis, cough, recurrent lung infections, and symptoms of pulmonary hypertension.[4] Some studies suggest a direct correlation with increased radiofrequency energy and the degree of pulmonary vein stenosis, namely patients who have undergone procedures with $>30$ W of ablation energy have a higher propensity to develop severe stenosis.[9] While the majority of the cases of pulmonary vein stenosis are mild stenosis without clinical consequence, a minority of these cases results in severe stenosis. A severe stenosis is defined as a $>70\%$ diameter reduction of a pulmonary vein. Severe stenosis can lead to pulmonary infarction, pulmonary venoocclusive disease, or pulmonary arterial hypertension, which may require intervention consisting of balloon dilatation and stenting.[10] On transesophageal echocardiography, pulmonary vein stenosis is identified as an increased peak systolic velocity within the pulmonary veins or a narrowed diameter of the veins. A contrast-enhanced computed tomography (CT) of the chest demonstrates a narrowed caliber of the involved pulmonary vein (Fig. 2). Should pulmonary venous hypertension or pulmonary venous infarct occur, CT thorax may demonstrate interlobular septal thickening and ground-glass opacities in the distribution of the stenotic vessel. Chest radiograph or CT thorax may show heterogeneous or patchy airspace opacity corresponding to the infarcted parenchyma.[11] A nuclear medicine ventilation perfusion study may allow for confirmation of pulmonary vein stenosis by demonstrating differential perfusion within the lungs (Fig. 3).

## RIGHT PHRENIC NERVE INJURY

Direct thermal injury to the right phrenic nerve can be seen in either radiofrequency or cryoablation, although it is most commonly seen following cryoballoon ablation of the right-sided pulmonary veins. While phrenic nerve injury can be seen in up to

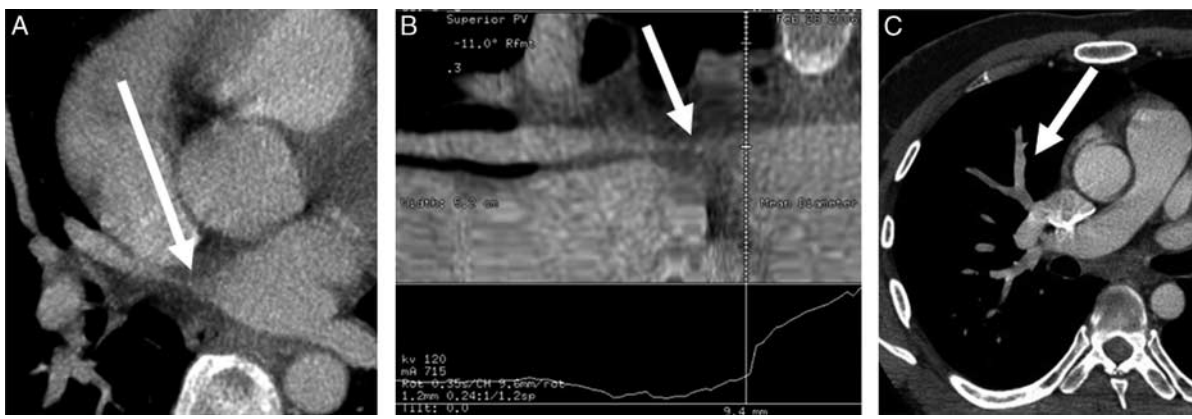


**FIGURE 2.** A 48 year-old man status postpulmonary vein ablation. Axial contrast-enhanced CT (A) demonstrates near-complete occlusion of right superior pulmonary vein (arrow) confirmed by catheterization. Reconstructed vessel probe image demonstrates the severe stenosis (B, arrow). There is prestenotic dilatation of the right superior pulmonary vein (C, arrow).

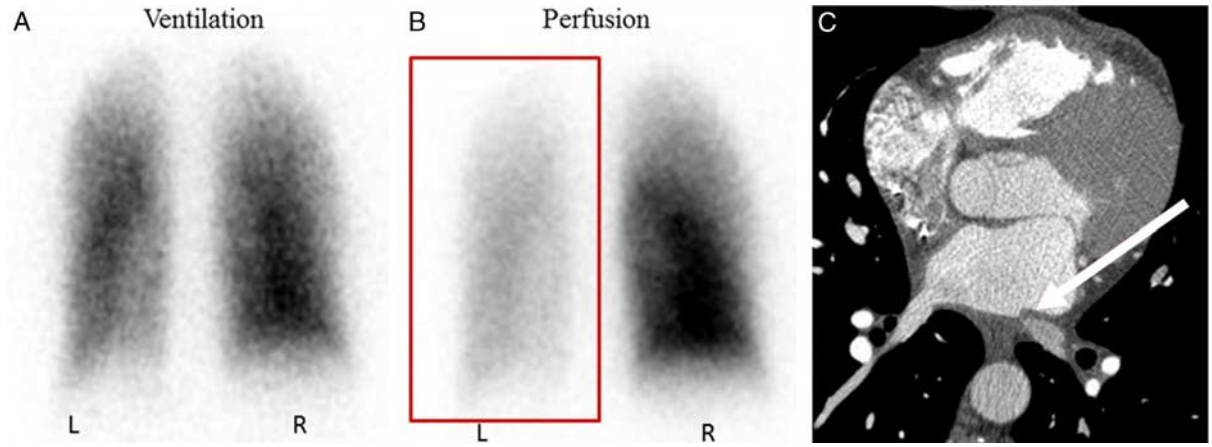


**FIGURE 3.** Posterior ventilation (A) and perfusion (B) images from a nuclear medicine ventilation perfusion scan of a dyspneic 39-year-old man with atrial fibrillation status postradiofrequency ablation demonstrate markedly decreased perfusion of the left lung compared with the right (red box), quantified as 15% left and 85% right. Axial contrast-enhanced CT of the chest (C) shows severe stenosis of the left inferior pulmonary vein (white arrow). Angioplasty was unsuccessful, and stenosis was treated with stent placement. Image courtesy of Dr E. Kazerooni.

10.8% of patients following cryoballoon ablation, the reported incidence of phrenic nerve injury following radiofrequency energy is <1%.[4] It has been postulated that the inflated balloon devices themselves not only create a broader thermal gradient, but also distort the anatomic distance between the right superior pulmonary vein endocardium and the right phrenic nerve. Furthermore, smaller balloon devices, such as those used during cryoablation, carry a higher risk for phrenic nerve injury, as they are placed more distally within the pulmonary veins.[4] The right phrenic nerve is susceptible to injury, as it runs posterior to the subclavian vein and crosses anteriorly over the root of the right lung at the origin of the pulmonary veins. Notably, left phrenic nerve injury does not occur, as the course of the left phrenic nerve is anterior and within the pericardium. Phrenic nerve injury can be asymptomatic, or manifest as dyspnea, hiccups, atelectasis, cough, and thoracic pain.[12] On conventional radiograph, a newly elevated right hemidiaphragm after the ablation should raise suspicion for right phrenic nerve injury. A confirmatory fluoroscopic sniff test would demonstrate paradoxical upward motion of the right hemidiaphragm with increased intrathoracic pressure (sniffing), as negative intrathoracic pressure causes a nonfunctioning diaphragm to move upward (Fig. 4).

## ESOPHAGEAL INJURIES

The close proximity of the esophagus to the posterior wall of the left atrium makes it highly susceptible to injury, thermal or mechanical, during the catheter ablation procedure. The complications of esophageal damage can be severe, and thus the procedure necessitates usage of several safeguards to prevent injury. The most common of these safeguards is known as modifying energy delivery, essentially lowering power delivery on the posterior wall of the left atrium to <25 W during radiofrequency ablation. Despite this precaution, injury to the esophagus may still occur if ablation duration is prolonged or contact force significant. As such, an additionally used strategy requires movement of the ablation catheter every 10 to 20 seconds along the atrial wall. A second safeguard against esophageal injury is that of esophageal visualization; by utilizing multidetector CT or topographic tagging with an electroanatomical mapping system, operators can either avoid the esophagus altogether or use lower power and/or alternative energy to ablate lesions over the esophagus. A third safeguard involves use of luminal esophageal temperature monitoring to identify dangerous heating of the esophagus. While there is no consensus with regard to the exact esophageal temperature at which ablation can be

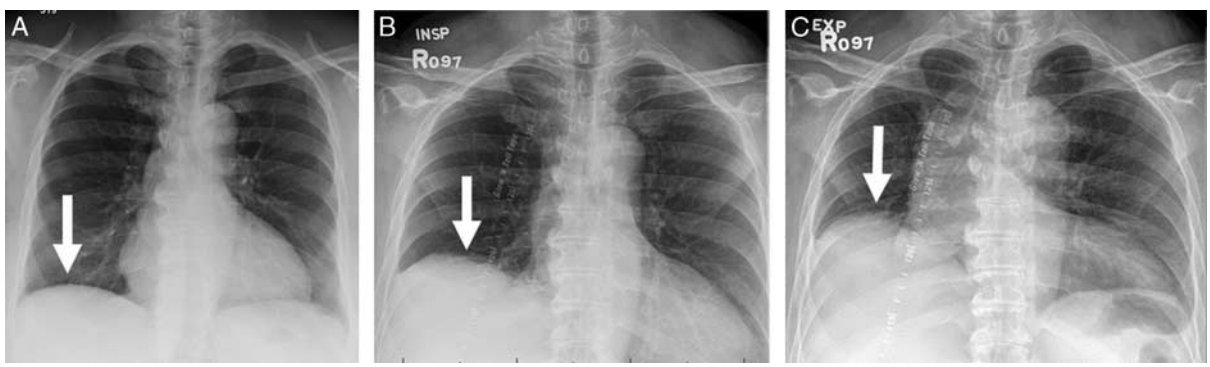


**FIGURE 4.** Baseline chest radiograph (A) of a 62-year-old woman with a history of paroxysmal atrial fibrillation demonstrating relative symmetric position of the hemidiaphragms. Two weeks after radiofrequency ablation, the patient presented with new-onset dyspnea. Inspiratory (B) and expiratory (C) chest radiographs demonstrate new right hemidiaphragm elevation (white arrows). A fluoroscopic sniff test confirmed paradoxical motion of the right hemidiaphragm, diagnostic of right phrenic nerve injury and diaphragmatic paralysis.

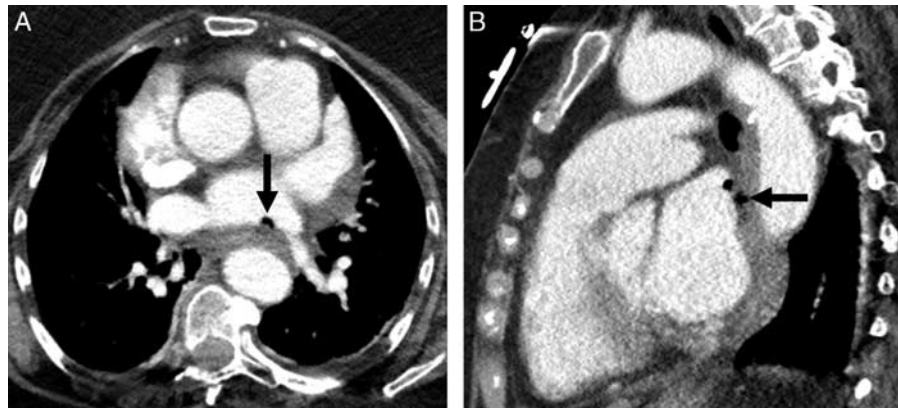


**FIGURE 5.** A 70-year-old woman presented with stroke-like symptoms ~3 weeks after catheter ablation for atrial fibrillation. Contrast-enhanced axial (A) and sagittal (B) images from CT of the thorax demonstrate loss of fat plane between the esophagus and left atrium with a few pockets of air in the dependent atrium abutting the esophagus (black arrows), strongly suggestive of an atrioesophageal fistula.

terminated, some studies have recommended cessation of radiofrequency ablation with a 2°C rise in luminal esophageal temperature.[4] Esophageal ulcerations, perforations, and atrioesophageal fistulas have all been reported after ablation. Post-ablation esophageal ulcers immediately posterior to the left atrium have a reported incidence of up to 60% in one study,[4] all of which were confirmed with endoscopy. Notably, these were usually healed on follow-up endoscopy 2 to 3 weeks later.

Atrioesophageal fistula is the most severe of esophageal injuries, and has a high mortality rate, which approaches 100%. The overall incidence of atrioesophageal fistula is <1%.[13] Clinical manifestations of an atrioesophageal fistula typically present within 2 to 4 weeks after the ablation procedure.

Patients most often have a clinical presentation of stroke and sepsis, as ingested material enters the left atrium via the fistula and is embolized to the brain and other organs. Radiologists must be vigilant as a CT head may be the first ordered study in such a patient, potentially demonstrating multiple embolic infarcts within the brain, some of which may contain air emboli. The presence of air emboli should trigger a review of the recent past medical history for pulmonary vein ablation, as that history frequently may not be provided in the setting of a neurological presentation. A CT of the thorax can demonstrate a near-pathognomonic finding of small bubbles of air within the dependent left atrium adjacent to the esophagus (Fig. 5). Less commonly, an air-filled communication between the atrium and esophagus will be seen (Fig. 6).

Serous pericardium, the innermost layer of pericardium, is reflected onto the heart as epicardium and has attachments to the pulmonary veins, SVC, aorta, and pulmonary trunk. As such, direct injury to the pericardium and esophagus during the ablation procedure could result in a fistulous tract between the 2. Termed pericardioesophageal fistula, this is a rare complication with the first reported case in 2010.[14] This condition usually manifests 2 to 4 weeks after surgery with nonspecific complaints, including epigastric and substernal chest pain. Chest radiograph in these patients may demonstrate an air-fluid level within the pericardium, with diagnostic confirmation established after demonstration of passage of contrast material from the esophagus into the pericardium on fluoroscopy.[15] Findings of pericarditis or pericardial effusion may be other indications of a pericardioesophageal fistula. The presence of pneumopericardium on CT in the setting of prior pulmonary vein ablation is strongly suggestive of pericardioesophageal fistula (Fig. 7).

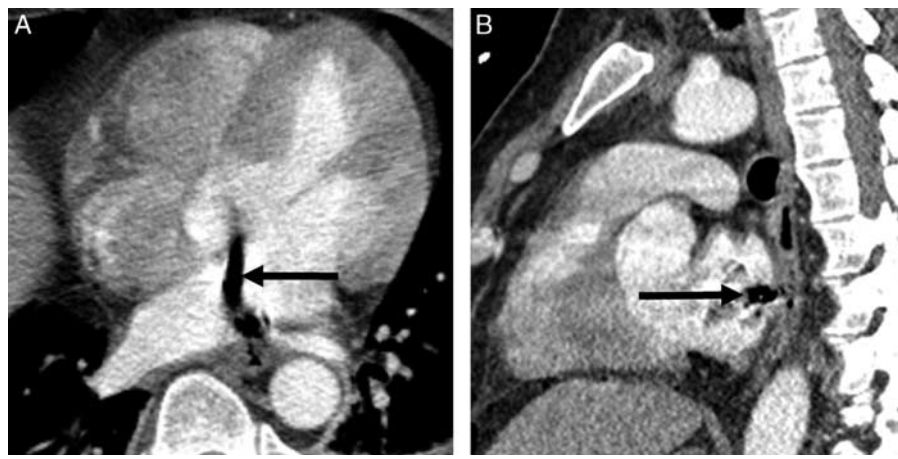


**FIGURE 6.** A 42-year-old man with atrial fibrillation status-post left atrial radiofrequency ablation presented with bilateral upper extremity weakness and dysphagia 2 weeks after the procedure. Contrast-enhanced axial (A) and sagittal (B) images from a CT thorax demonstrate linear collection of air extending from the esophagus to the left atrium (black arrows), consistent with an atrioesophageal fistula.

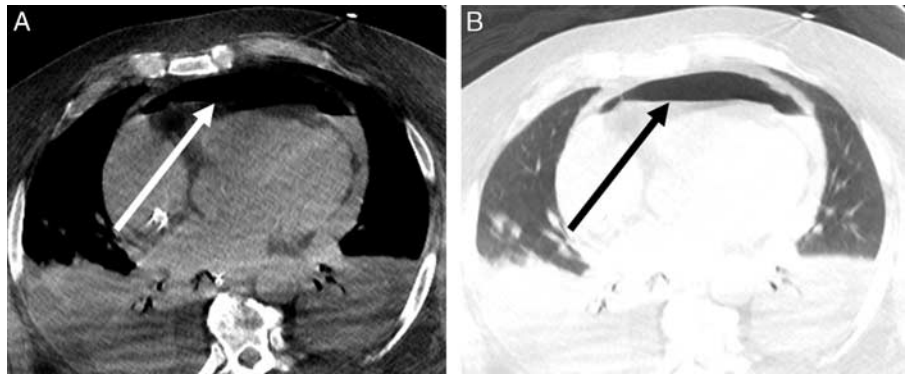


**FIGURE 7.** A 50-year-old man who became hypotensive during his ablation procedure and bedside echocardiogram demonstrated cardiac tamponade. Axial image from noncontrast CT chest on soft tissue (A) and lung window (B) after his procedure shows hydropneumopericardium (arrows). A pericardioesophageal fistula was subsequently confirmed.

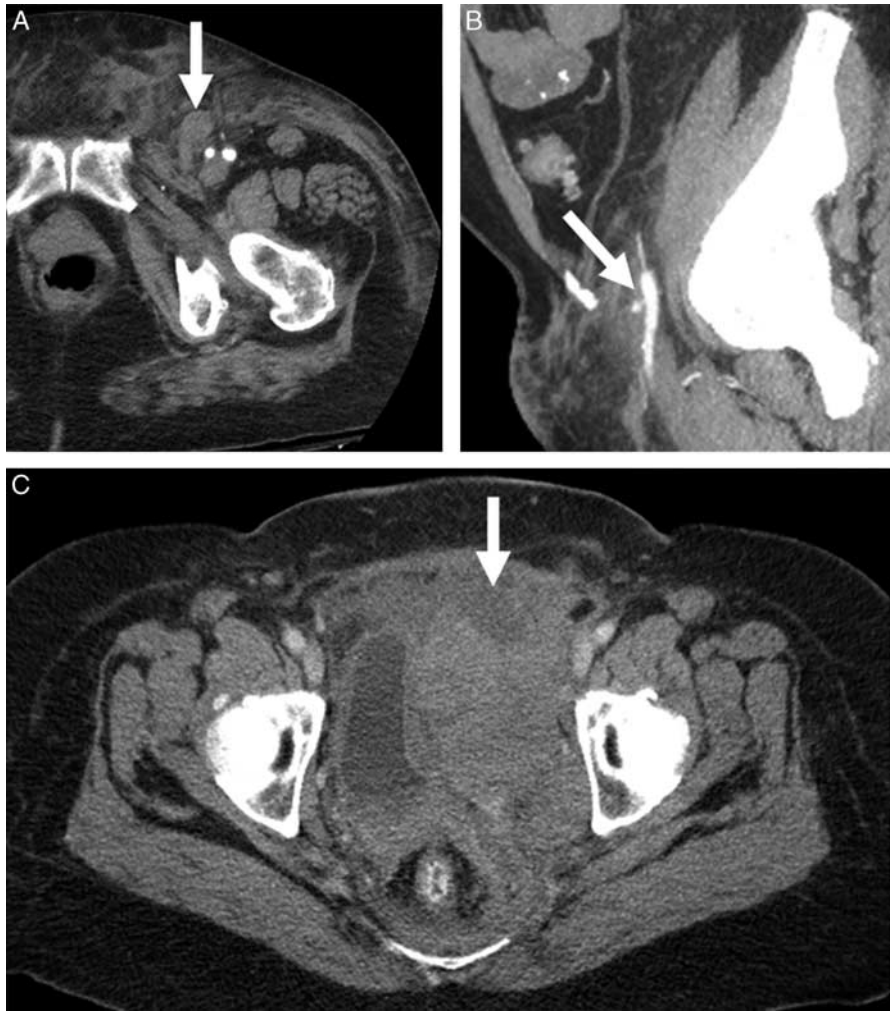


**FIGURE 8.** Images from 3 different patients demonstrating local vascular complications related to femoral arterial access. Axial image from contrast-enhanced CT angiogram of the pelvis (A) demonstrates a focal hematoma superficial to the left femoral vessels (white arrow). Sagittal image from contrast-enhanced CT angiogram of the pelvis (B) demonstrates a focal pseudoaneurysm arising off of the anterior aspect of the left superficial femoral artery (white arrow). Axial image from a contrast-enhanced CT of the pelvis (C) displays a heterogeneous high-attenuation left pelvic hematoma (white arrow).

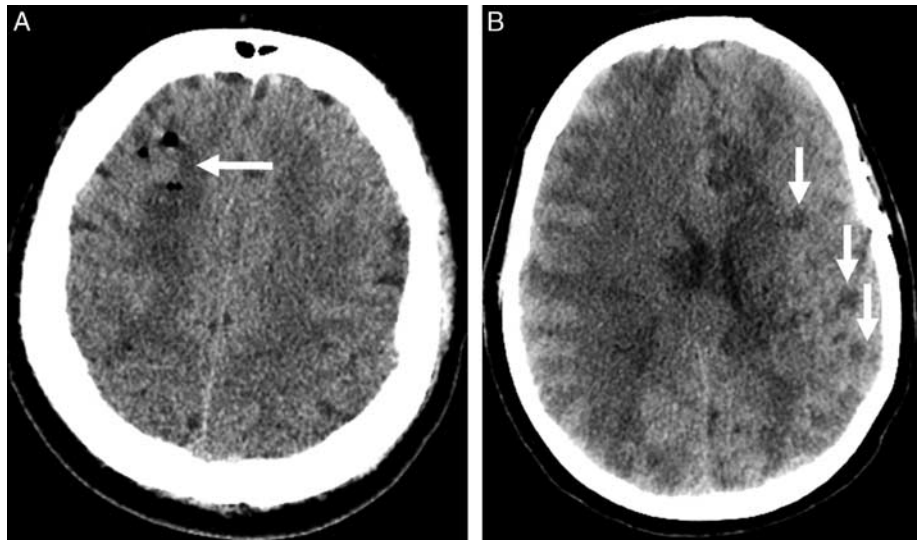


**FIGURE 9.** Axial noncontrast CT scan of the brain in 2 different patients demonstrating embolic complications of pulmonary vein ablation. In a patient with atrioesophageal fistula after ablation (A) a cluster of gas bubbles (white arrow) is present from air emboli. A different patient (B) recently status postablation demonstrates multiple rounded infarcts in the left cerebral hemisphere (white arrows), as well as larger infarct in the right cerebral hemisphere from embolic phenomena.

## ADDITIONAL COMPLICATIONS

### Cardiac Perforation and Tamponade

The incidence of myocardial perforation and tamponade following pulmonary vein ablation has been estimated to be 0.5% to 4%, although a study in 2005 by the Mayo Clinic Study reports an incidence as high as 2.1%.[16] The most common causes of cardiac perforation and subsequent tamponade during catheter ablation result from misdirected transseptal punctures (exit through the right or left atria), direct mechanical trauma, and overheating during radiofrequency energy delivery.[4] Cardiac tamponade manifests as either an abrupt drop or gradual decrease in blood pressure. Notably, an intracardiac echocardiogram will demonstrate an effusion before the onset of hemodynamic instability. Tamponade can also occur 1 hour or more following the ablation procedure. Cardiac tamponade is managed immediately by subxiphoid percutaneous drainage and reversal of anticoagulation. The actual perforated myocardium, however, rarely requires surgical intervention. Of note, it is estimated that 66.7% of patients with myocardial perforation develop early recurrence of atrial fibrillation.[17] Notably, in the same Mayo Clinic study, 10 of 15 patients who had their ablation procedure complicated by myocardial perforation developed early recurrent atrial fibrillation, despite having their procedures completed. This is likely secondary to the myocardial and pericardial inflammatory reaction associated with perforation, which results in cellular dysfunction and thus enhanced arrhythmogenicity.[16,17]

Vascular complications, primarily involving the femoral access site, are common, with a published incidence ranging from 0% to 13%. Groin hematomas are the most frequently encountered complication, although cases of arterial pseudoaneurysms, femoral arteriovenous fistulas, and retroperitoneal hemorrhage have been reported (Fig. 8). Intuitively, these complications are linked to the number and size of the catheters used, as well as the patients' anticoagulation status. Vascular injuries in the vicinity of the ablation site have also been reported to occur. Such possible complications include hemothorax, subclavian hematoma, and extrapericardial pulmonary vein perforation.

The incidence of thromboembolic events after ablation, be they from air emboli or thromboemboli, have a reported occurrence of 0% to 7%.[4] These events typically occur within 24 hours of the ablation procedure with the high-risk period extending for up to 2 weeks after the procedure. Thrombi can occur either from stationary sheaths or originate from a left atrial appendage thrombus, and are the reason for the current aggressive preprocedural and intraprocedural prophylactic anticoagulation protocols. Air emboli usually are introduced by way of sheath or catheter placement but may be secondary to atrioesophageal fistula (Fig. 9). While intracranial emboli are most commonly reported, coronary arterial or abdominal vascular emboli are also possible. Patients presenting with altered mental status, new-onset seizures, or a focal neurological deficit should undergo emergent brain CT or magnetic resonance imaging. Notably, air emboli preferentially migrate into the right coronary artery. As such, a patient presenting with acute-onset chest pain after their ablation procedure with ECG findings of acute inferior ischemia or heart block should be presumed to have a right coronary artery infarction secondary to an air embolism until proven otherwise.[4]

Hemoptysis is an uncommon complication of pulmonary ablation with an incidence of 0% to 2.1%.[4] While hemoptysis can be a symptom of pulmonary vein stenosis, it is not always the case. Hemoptysis can simply be a manifestation of transient interruption of vascular integrity within the pulmonary capillary system, which is associated with lower freezing temperatures. Rarely, hemoptysis can be seen with bronchial erosion given the close proximity of the pulmonary veins to the bronchial tree. The first case of bronchial erosion was reported in 2011.[18] Naturally, bronchoscopy may be warranted in a patient with hemoptysis. A CT of the thorax may or may not demonstrate the bronchial erosion or fistulous connection between the bronchus or pulmonary vein; however, it could demonstrate ground-glass opacity/edema adjacent to the vein, suspicious for injury.

## CONCLUSION

Although the imaging findings may be nuanced, many of the complications of pulmonary vein ablation for atrial fibrillation have findings on radiology examinations that are near pathognomonic. As such, it is paramount that radiologists familiarize themselves with the pulmonary vein ablation procedure and potential complications to allow for early recognition, diagnosis, and potential treatment.

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