Ventilation-perfusion Mismatch Resulting From Iatrogenic Pulmonary Vein Stenosis After Radiofrequency Ablation: A Case Report

Ka Kit Wong, MB, BS, Simon M. Gruenewald, MB, ChB, George Larcos, MB, BS

Centre for Biomedical Imaging and Research, Department of Nuclear Medicine and Ultrasound, University of Sydney, Westmead Hospital, Sydney 2145, New South Wales, Australia

ABSTRACT

A lobar ventilation-perfusion mismatch is reported in a patient with right superior pulmonary vein stenosis, developing after radiofrequency ablation for atrial fibrillation. Radionuclide lung perfusion tomography was performed to assist with clinical management. Serial planar lung scintigraphy demonstrated no improvement in the perfusion abnormality despite interventional stenting. Iatrogenic pulmonary vein stenosis may potentially mimic pulmonary embolic disease.

Introduction

Catheter-directed radiofrequency ablation is an increasingly popular and effective treatment for atrial fibrillation (AF). The aim of radiofrequency ablation is to isolate ectopic foci of electrical activity in the muscular sleeves surrounding the pulmonary veins, which have been demonstrated to be the triggers for AF. Rarely, severe pulmonary vein stenosis (PVS) can occur as a complication and this may lead to venous occlusion and infarction. Milder degrees of PVS have less physiologic impact. Lung scintigraphy has been found to demonstrate reduced or absent perfusion with intact ventilation, and has been used to aid diagnosis and management of this condition.

Case Report

A 63-year-old man was referred to our department with stenosis of his right superior pulmonary vein secondary to radiofrequency ablation for AF. The patient had a longstanding history of paroxysmal AF treated with sotalol. He had undergone 3 catheter procedures in total due to recurrence of his AF each time. At the first procedure 16 months earlier, ectopic electrical foci adjacent to his left inferior and right superior pulmonary veins were isolated. Following circumferential ablation to the right superior pulmonary vein, a 40% to 50% stenosis was noted. Five months later during the second procedure both his left inferior and superior pulmonary veins were treated and sinus rhythm was restored. After another 11 months during his third catheter ablation procedure balloon venoplasty to the right superior PVS resulted in a completely patent vessel

immediately thereafter (Figure 1). He was referred to our department 2 weeks later for assessment of lung perfusion.

On physical examination, he had a normal heart rate in sinus rhythm, clear lungs on auscultation, and normal heart sounds. He was asymptomatic and all his laboratory values were within normal limits. A 99m-Tc-macroaggregated albumin perfusion lung scan was performed with a dual-head gamma camera set to a 140 keV energy peak with a 20% window. The single-photon emission computed tomography (SPECT) acquisition involved 360-degree rotation; 64 steps; 30 seconds/step, and reconstruction was performed with FBP on a 128 × 128 matrix. There was moderate reduction in perfusion to the right upper lobe (Figure 2).

Three weeks later the patient returned for a follow-up ventilation perfusion lung scan to assess for any improvement in perfusion to the right upper lobe. There was redemonstration of reduced perfusion to the right upper lobe in a pattern of lobar ventilation-perfusion mismatch (Figure 3). Guided by the scintigraphic finding of persistent abnormal perfusion, the patient proceeded to interventional stenting of his right superior pulmonary vein. Although the patient was asymptomatic, there was concern that the reduction in right upper lobe perfusion indicated failure of the earlier venoplasty procedure and an increased risk of progression to pulmonary venous infarction.

Long-term lung scintigraphy at 16 months and 20 months following the stent placement has been performed for this patient. The right upper lobe perfusion defect became more severe and fixed in appearance between the 2 later studies (Figure 4). With clinical follow-up out to 2 years there was

Figure 1. (A) Pre-venoplasty catheter image demonstrates stenosis of the origin of the right superior pulmonary vein (arrow) and (B) post-venoplasty, the luminal defect is no longer visualized. Abbreviations: LA, left atrium; RSPV, right superior pulmonary vein.

Figure 2. Two weeks post-venoplasty 99m-Tc-MAA SPECT images show a moderate reduction in perfusion to most of the right upper lobe on coronal views and comparison between right and left lungs on the sagittal views.

Figure 3. Follow-up lung scintigraphy 3 weeks later. Selected right posterior oblique (RPO) view on a planar 99m-Tc-technegas/99m-Tc-MAA lung scintigraphy demonstrates subtle ventilation-perfusion mismatching of the apex of the right lung with normal ventilation.

no evidence for thromboembolic disease, with the patient remaining in sinus rhythm and in good health.

Discussion

Radiofrequency ablation is a relatively new treatment option for AF. The vast majority of ectopic beats that lead to AF

originate adjacent to the pulmonary veins. Targeted cautery of cardiac tissue by local application of radiofrequency energy can be used to directly destroy or electrically isolate these triggers. This leads to elimination of AF in the short-term in up to 70% of cases, although repeated procedures are often required due to new ectopic foci being identified or inadequate electrical isolation following ablation.¹ The major benefit is that the patient can avoid anticoagulation and antiarrhythmic medications.

The complication rate of PVS has yet to be fully determined for radiofrequency ablation in AF. The early experience was that severe PVS due to direct thermal injury occurred in 5% of cases however this may be decreasing with the use of lower energy ablation.² PVS can be diagnosed with various imaging modalities including helical computed tomography (CT) angiography, radionuclide lung studies, magnetic resonance (MR) perfusion imaging, and imaging strategies to monitor patients for early detection of complications have been reported.^{3–5}

The diagnosis of PVS resulting in a mismatched ventilation-perfusion defect on lung scintigraphy was based on the catheter findings of a 50% PVS with an associated

Figure 4. Long-term (20 month) progress 6-view planar radionuclide lung scan demonstrates a lobar mismatched ventilation-perfusion defect in the right upper lobe. Serial lung scintigraphy shows that the severe perfusion abnormality associated with PVS may become persistent despite interventional correction of the stenosis. In this case the upper lobe distribution is not suggestive of pulmonary embolic disease, however, in a different clinical setting this lobar pattern of mismatch can mimic pulmonary embolism and cause diagnostic difficulties.

pulmonary perfusion abnormality in the same territory, and subsequent 2 year follow-up with no clinical evidence for thromboembolic events. Helical computed tomography pulmonary angiography (CTPA) or pulmonary angiography was not performed initially as the patient had a low pre-test probability for pulmonary embolism, and only a single upper lobar perfusion defect on lung scintigraphy. Even with current technology, the sensitivity of CTPA is inadequate to conclusively exclude pulmonary embolism in a patient without an acute symptomatic presentation.⁶ Presently it is not known if CTPA is indicated in patients with known PVS and a mismatched ventilation-perfusion defect on lung scintigraphy in the same territory.

Saad et al used helical CT at 3, 6, and 12 months post-procedure and reported a complication rate for severe PVS

of 18 (5%) out of 355 patients.⁴ Abnormal radiographic findings on chest X-ray were present in 50% of these patients, mainly lung consolidation and left pleural effusions, which were misdiagnosed in 7 out of 9 patients. Helical CT demonstrated a median of 2 pulmonary veins involved, a left-sided predominance (83%), and total venous occlusion of at least 1 pulmonary vein in 7 patients. Lung scintigraphy before and after interventional angioplasty or stenting showed significant improvements in perfusion following the procedure.

The utility of radionuclide lung scintigraphy for functional assessment of PVS was reported by Nanthakumar et al in a cohort of 11 patients, 6 of whom had PVS >50% with a total of 16 pulmonary veins involved.³ They found that lung scintigraphy identified a lobar perfusion defect corresponding to every PVS >80% and a pulmonary vein to left atrial

(PV-LA) gradient of >5 mm Hg. None of the patients with PVS had evidence for arterial occlusion on anatomic imaging, suggesting that PVS was the sole cause of the perfusion abnormalities. Decreased pulmonary perfusion in PVS has also been demonstrated with MR perfusion imaging, post catheter ablation in a cohort of 110 patients, 51 of whom had PVS $>25\%$.⁵ MR perfusion imaging was highly sensitive (detected 20 of 21 perfusion defects), when compared to 39 patients who also had SPECT lung perfusion studies for comparison.

To the best of our knowledge the finding of mismatched ventilation-perfusion on lung scintigraphy attributed to PVS was first described by Robbins et al in 1998.⁷ A radionuclide lung scan demonstrated nearly complete absence of perfusion to the entire right lung and left lower lobe with a normal pattern of ventilation due to involvement of all 4 pulmonary veins. Subsequently this finding has been confirmed by several sources in the literature, with the close correlation between perfusion deficits and pulmonary vein involvement, and the lack of arterial emboli on CT or MRI scans, arguing that arterial pulmonary embolism is not the cause of the mismatch. The appearance may range from unilateral severe or absent perfusion^{4,7,8,9} to milder degrees of impaired perfusion with lobar distributions, as in our case.^{3,5,10}

Although the pathophysiologic mechanism underlying the mismatched ventilation-perfusion has not been fully determined, similar radionuclide lung scan findings have been reported in a range of pulmonary venous system disorders, including pulmonary venous compression,^{11,12} pulmonary vein atresia,^{13,14} veno-occlusive diseases,^{15,16} and pulmonary vein occlusion related to congenital cardiac surgery.¹⁷

Conclusion

This case highlights the clinical utility of radionuclide lung scintigraphy in the diagnosis and progress assessment of PVS following radiofrequency ablation for AF. Iatrogenic PVS can cause a persistent, lobar pattern of ventilation-perfusion mismatch, potentially mimicking pulmonary embolism, and may be more commonly encountered in the future with wider use of catheter techniques for treatment of AF.

References

1. Grubb NR, Furniss S. Science, medicine, and the future: radiofrequency ablation for atrial fibrillation. *BMJ*. 2001;322:777–780.
2. Jais P, Weerasooriya R, Shah DC, et al. Ablation therapy for atrial fibrillation: past, present and future. *Cardiovasc Res*. 2002;54:337–346.
3. Nanthakumar K, Mountz JM, Plumb VJ, Epstein AE, Kay GN. Functional assessment of pulmonary vein stenosis using radionuclide ventilation perfusion imaging. *Chest*. 2004;126:645–651.
4. Saad EB, Marrouche NF, Saad CP, et al. Pulmonary vein stenosis after catheter ablation of atrial fibrillation: emergence of a new clinical syndrome. *Ann Intern Med*. 2003;138:634–639.
5. Kluge A, Dill T, Ekinici E, et al. Decreased pulmonary perfusion in pulmonary vein stenosis after radiofrequency ablation. *Chest*. 2004;126:428–437.
6. Stein PD, Fowler SE, Goodman LR, et al. Multidetector computed tomography for acute pulmonary embolism. *N Engl J Med*. 2006;354:2317–2327.
7. Robbins IM, Colvin EV, Doyle TP, et al. Pulmonary vein stenosis after catheter ablation of atrial fibrillation. *Circulation*. 1998;98:1769–1775.
8. Ravenel JG, McAdams HP. Pulmonary venous infarction after radiofrequency ablation for atrial fibrillation. *Am J Roentgenol*. 2002;178:664–666.
9. Salamon F, Hirsch R, Tur-Kaspa R, Kramer MR. Search for the complication. Clinical problem-solving case. *N Engl J Med*. 2006;354:957–963.
10. Fink C, Schmaehl A, Bock M, Tuengerthal S, Delorme S. Pulmonary vein stenosis after radiofrequency ablation for atrial fibrillation: image findings with multiphasic pulmonary magnetic resonance angiography. *Circulation*. 2003;20:e129–e130.
11. Mendelson DS, Train JS, Goldsmith SJ, Efrimidis SC. Ventilation-perfusion mismatch due to obstruction of pulmonary vein. *J Nucl Med*. 1981;22:1062–1063.
12. Hartshorne MF, Bauman JM, Cawthon MA, Huggins M. Right pulmonary vein occlusion detected by ventilation perfusion lung scanning. *Clin Nucl Med*. 1985;10:827–828.
13. Heyneman LE, Nolan RL, Harrison JK, McAdams HP. Congenital unilateral pulmonary vein atresia. Radiologic findings in three adult patients. *Am J Roentgenol*. 2001;177:681–685.
14. Beerman LB, Oh KS, Park SC, et al. Unilateral pulmonary vein atresia: clinical and radiographic spectrum. *Pediatr Cardiol*. 1983;4:105–112.
15. Holcomb BW Jr, Loyd JE, Ely EW, Johnson J, Robbins IM. Pulmonary venoocclusive disease: a case series and new observations. *Chest*. 2000;118:1671–1679.
16. Sola M, Garcia A, Picado C, et al. Segmental contour pattern in a case of pulmonary venoocclusive disease. *Clin Nucl Med*. 1993;18:679–681.
17. Houzard C, Andre M, Guilhen C, Thivolle P, Berger M. Perfusion lung scan in patients operated for transposition of the great arteries. *Clin Nucl Med*. 1989;14:268–270.