The pulmonary vein that stumped us

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A 76-year-old man with hypertension, gout, and symptomatic paroxysmal atrial fibrillation (AF) resistant to dofetilide presented to our institution for catheter-based ablation. Sixteen years prior to the procedure in 2004, the patient had small cell lung cancer of the left lung treated with chemotherapy and radiation. He developed symptomatic atrial arrhythmias in 2010 and had a cavotricuspid isthmus ablation in 2011 and had been on dofetilide 250 μg twice daily since 2015. Additional antiarrhythmic options were limited by relative sinus bradycardia and QT prolongation with higher doses of dofetilide. In the electrophysiology laboratory, left atrial (LA) electroanatomical mapping (Figure 1A and B) as well as contrast injections (Figure 1C) revealed complete occlusion of the proximal left superior pulmonary vein (LSPV). The authors were initially reluctant to commit to the isolation of the LSPV stump in order to avoid any possible thrombotic risk related to the isolation of such an appendage-like structure. Therefore, following the isolation of the right pulmonary veins (PV), a spline-based multi-electrode catheter (Pentaray, Biosense Webster) was positioned into the LSPV stump, and an open irrigated radiofrequency ablation catheter (STSF, Biosense Webster) was positioned in the left inferior PV. Upon infusion of isoproterenol up to 4 mcg/min, multiple paroxysmal runs of triggered firing were appreciated from the posterior aspect of the LSPV stump (Figure 1D). To eliminate the possibility that these may have been mechanically triggered from the multi-spline catheter, the catheter was repositioned elsewhere but AF paroxysms continued with an identical triggering activation sequence on decapolar recordings in the coronary sinus. Ultimately, AF sustained, and we reasoned that the LSPV stump was unlikely to be meaningfully contractile and likely represented a clinically relevant trigger source. Therefore, ablation was commenced around the left PV antra. Just prior to completing encirclement, the rhythm converted to sinus. The patient’s episodes of AF had been lengthening leading up to the procedure, edging towards persistent durations (>7 days), and therefore, the posterior wall of the LA was also isolated to provide additional substrate control. Following ablation, isoproterenol was reinfused and uptitrated to 20 mcg/min, but no arrhythmias remained inducible. He has not had a recurrence to date, now 2 months post-ablation.

It is now well appreciated that the PV muscle sleeves play a prominent role in the initiation and maintenance of AF,1 and their isolation constitutes the cornerstone of AF ablation. Disruption of these sleeves or the associated neuromuscular anatomy might be predicted to

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reduce their triggering role. However, Kanmanthareddy et al.\textsuperscript{2} studied 15 patients with a history of pneumonectomy and found that PV stumps after pneumonectomy continued to be electrically active, with 60% acting as triggering sites for AF. Our findings agree with theirs but also are unique in that the patient described in our case example had no history of pneumonectomy.

At our centre, pre-procedural imaging often presents a financial and logistical constraint, and particularly as three-dimensional mapping systems have advanced, we do not typically acquire such studies before AF ablation. Thus, the anatomical findings of this case were discovered intraprocedurally. Though not clear, it would have altered the ablation strategy in this case, in efforts to improve procedural planning, it may prove useful to selectively pursue imaging in advance for cases with a history of thoracic pathology. In summary, we present a case of triggered AF from a PV stump from prior cancer in the absence of pneumonectomy. Our findings agree with prior data that fully intact PV muscle sleeves are not necessary for the PV anatomy to trigger AF.

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\textbf{References}
