Pulmonary Vein Remnant as a Trigger Site for Atrial Fibrillation

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Over the past 10 years, pulmonary vein (PV) isolation (PVI) has been a widely accepted therapy for atrial fibrillation (AF), but success of the procedure requires an understanding of the complexity of PV anatomy. Technology has evolved to register cardiac activation to 3-dimensional (D) computed tomography (CT) images, also allowing identification of variations in PV anatomy. A right middle PV and common left trunk have been reported as the 2 most common PV anomalies. In the case described herein, 3-D CT of the left atrium (LA) and PVs showed a rare anatomic variant, that

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AF Originating From Remnant RIPV

is, an inferior PV remnant within the common inferior PV ostium; interestingly, this PV anomaly played an important role in triggering AF. A 63-year-old man with symptomatic paroxysmal AF was referred to us for PVI. He had previously suffered traumatic pulmonary contusion for which right lower lobectomy had been performed. We performed PVI guided by an Ensite NavX system (St. Jude Medical, St. Paul, MN, USA). Briefly, a quadripolar catheter was placed in the coronary sinus (CS), and after a single trans-septal puncture, a 3.5-mm irrigated-tip catheter (NaviStar Thermo-Cool; Biosense Webster, Diamond Bar, CA, USA) and 10-pole Lasso circular mapping catheters (Biosense Webster) were advanced into the LA. Heparin was given by i.v. infusion to maintain an activated clotting time >300 s. To prevent esophageal injury, an esophageal temperature catheter was placed in the esophagus adjacent to the posterior LA. The 3-D LA/PV geometry was created with a mapping catheter and merged with pre-acquired 3-D CT. Both images showed the common inferior PV ostium at the midline of the posterior LA. Also, a blind-ended right inferior (RI) PV (remnant) that resulted from the right lower lung lobectomy was seen (yellow arrowhead; Figure 1A). Because AF was sustained, a complex fractionated atrial electrogram (CFAE) map of the LA and right atrium was created (Figure 1B). Because no PV potentials were observed in the left inferior PV, isolation of the left and right superior PVs was performed during AF (each at a maximum temperature of 41°C, power 20–30 W, and irrigation rate 17 ml/min for 30 s). The AF was not terminated, so additional ablation at the distal sites of the CS, LA roof, and superior vena cava isolation was performed according to the CFAE sites (Figure 1B). These ablations did not terminate the AF; therefore cardioversion was performed. Spontaneous AF, however, occurred, initiated by atrial ectopic beats for which the earliest activation was recorded in the CS orifice. Thus, a mapping catheter was advanced into the RIPV remnant, showing the site of earliest activation to be the orifice of the RIPV remnant (Figure 1C). (Note that this area was already identified as a CFAE site on the pre-acquired CFAE map; yellow arrow, Figure 1B). The close proximity of the esophagus to the common inferior PV ostium made isolation of the common inferior PV ostium difficult. Interestingly, the esophageal temperature probe was seen on the 3-D map and on fluoroscopy to have shifted by 5 cm from the right to the left side of the inferior pulmonary vein ostium as shown on (Upper) 3-dimensional computed tomography and (Lower) fluoroscopy. Red arrow, distal pole of the esophageal temperature catheter. AP, anteroposterior; PA, posteroanterior.

Figure 2. Shift in the position of the esophagus from (A, C) the right to (B, D) the left side of the inferior pulmonary vein ostium as shown on (Upper) 3-dimensional computed tomography and (Lower) fluoroscopy. Red arrow, distal pole of the esophageal temperature catheter. AP, anteroposterior; PA, posteroanterior.
tion, the present patient had a rare variant RIPV remnant that played an important role in triggering AF. AF remains the most common medical complication after thoracic surgery, with an incidence ranging from 10% to 20% after pulmonary lobectomy. Despite that high incidence, the mechanism of AF after lung lobectomy remains unclear. It is possible that surgical knotting of the wall of the PVs near the pericardial reflection can cause mechanical ischemia or inflammatory damage to the zone that is continuous with excitable myocardial tissue, in turn generating ectopic beats and subsequent AF.

To the best of our knowledge, there has been only 1 report of a left superior PV remnant resulting from lobectomy being the AF trigger site. The present and these reported observations suggest that a PV remnant resulting from lung lobectomy can be related to the development of AF although we could not entirely exclude the possibility that the original RIPV could have provided arrhythmogenic substrate for AF triggers. Furthermore, in the present patient, the position of the esophagus changed during the ablation procedure. Kennedy et al reported that in 7 of 42 patients (17%), the esophagus shifted at least 1 cm from its original position toward the left and right PVs during LA ablation. In the present patient with the rare PV anatomy, the esophageal location shifted dramatically from the right to the left side of the common inferior PV ostium. Real-time identification of the esophageal temperature probe on the 3-D map helped in planning the AF ablation strategy, leading to successful isolation of the RIPV without any complications.

The present experience in this case implies that, after lung lobectomy or pneumonectomy, even the PV remnant can be an AF trigger site. Detailed anatomic identification on 3-D CT combined with real-time monitoring of the esophageal temperature probe can help to ensure the success and safety of AF ablation in such complex anatomical cases.

Disclosures

None.

References