Catheter ablation for atrial fibrillation in a patient with unilateral left pulmonary artery agenesis: an enlarged right pulmonary vein caused arrhythmogenicity of atrial fibrillation

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A 55-year-old male with persistent atrial fibrillation (AF) and typical atrial flutter was admitted to our hospital for catheter ablation (CA). Enhanced computerized tomography (CT) before CA demonstrated a left shrunken lung and absence of the left main pulmonary artery (Panel A). Collateral flow via the coronary and bronchial arteries fed to the peripheral left pulmonary artery. All of the four pulmonary veins (PVs) were present, but the left PVs were relatively small, and the right superior and inferior PVs were enlarged because of increased pulmonary blood flow (right superior: 31.1 × 15.8 mm, right inferior: 30.3 × 17.7 mm, left superior: 16.5 × 11.8 mm, left inferior: 9.2 × 11.0 mm).

Pulmonary vein potentials in the smallest (left inferior) PV were not prominent compared with the other three PVs. After injection of isoproterenol and adenosine triphosphate disodium hydrate, premature atrial contractions from the enlarged right superior and inferior PVs were reproducibly induced (Panel B). We considered that repetitive premature atrial contractions represented arrhythmogenicity in the enlarged right PVs. We performed extensive PV isolation using 3D mapping (CARTO®3, Biosense Webster, Inc., Diamond Bar, CA, USA) and linear ablation of the cavo-tricuspid isthmus. Enlarged right PVs were easily isolated. There were no complications and no recurrence of atrial arrhythmias after 8 months follow-up using 24-h Holter monitoring every 3 months and electrocardiogram at every outpatient clinic visits.

Unilateral pulmonary artery agenesis (UPAA) is a rare congenital abnormality with an estimated prevalence ranging from 1 per 150 000 persons to 1 per 300 000 persons. Unilateral pulmonary artery agenesis is frequently complicated by other cardiovascular abnormalities, such as tetralogy of Fallot, atrial septal defect, coarctation of the aorta, right aortic arch, truncus arteriosus, and pulmonary atresia.1 In patients without concomitant cardiovascular abnormalities and symptoms, UPAA can be diagnosed by an abnormal chest radiograph and chest CT imaging, as in our patient.

Yamane et al. reported that the ostial diameter of arrhythmogenic PVs was larger than that of non-arrhythmogenic PVs.2 Some studies have shown that reconnection of a larger PV to the left atrium is associated with recurrence of AF after CA.3 These findings suggest that the size of the PV ostium is closely related to the formation of substrate for AF. Unilateral pulmonary artery agenesis increases blood
flow of the pulmonary circulation and causes enlargement of an unaffected side of PVs. Excessive stretch of the myocardium may provide arrhythmogenic substrates in PVs. This theory is supported by our finding of prominent PV potentials and frequent PV ectopy from enlarged right PVs.

In the present report, we demonstrate efficacy of CA for AF in a case of left UPAA, which led to arrhythmogenicity owing to the increased size of right PVs because of uneven blood flow.

Conflict of interest: none declared.

References