CASE REPORT

Collateral pulmonary vein after catheter ablation therapy for atrial fibrillation

KYOKO NAGAI, MD, PhD, AKIO KOTAKE, MD, YOSHIRO HORI, MD, PhD, NOBUYUKI TAKEYAMA, MD, PhD, ELIKO TANAKA, MD, PhD, YUKI TASHIRO, MD, TOSHI HASHIMOTO, MD, PhD, DAISUKE WAKATSUKI, MD, PhD and HIROSHI SUZUKI, MD, PhD

Department of Radiology, Showa University Fujigaoka Hospital, Yokohama, Japan
The Cardiovascular Division of Internal Medicine, Showa University Fujigaoka Hospital, Yokohama, Japan
The Cardiovascular Division of Internal Medicine, Showa University Fujigaoka Hospital, Yokohama, Japan

Address correspondence to: Mrs Kyoko Nagai
E-mail: iaganokoyk@med.showa-u.ac.jp

A patient with previous catheter ablation therapy for atrial fibrillation was examined for an abnormal shadow on a chest radiograph. ECG-gated multidetector CT clearly showed the left upper pulmonary vein connected with the left inferior pulmonary vein. We hypothesize an intrapulmonary venous connection as a collateral.

A 44-year-old asymptomatic Japanese male received catheter ablation therapy twice after suffering from atrial fibrillation (AF). The first ablation therapy was performed 3 years ago, while the second was performed 2 years, 3 months after. There was no stenosis of any pulmonary vein (PV) and lung field abnormalities before the first catheter ablation therapy (Figure 1). The patient had no medical history except for catheter ablation therapy for AF.

He underwent ECG-gated multidetector CT 3 years, 11 months after the second catheter ablation therapy. CT revealed left upper PV stenosis and an anomalous intrapulmonary venous connection between the left upper PV and the left lower PV. The anomalous tortuous vein began from V4, ran caudally along the interlobar pleural surface and finally drained into the lower PV. Nodular ground glass opacities newly appeared at the apicoposterior segment of the LUL. However, the peripheral opacities and interstitial septal thickening in the LUL disappeared (Figure 3).

The patient was diagnosed with collateral pulmonary vein secondary to PV stenosis after catheter ablation therapy for AF.

The initial differential diagnosis for peripheral consolidation after catheter ablation therapy was pulmonary venous infarction, alveolar hemorrhage and nonspecific pneumonia. The differential diagnosis for anomalous intrapulmonary venous connection was pulmonary arteriovenous malformation, hypogenenic lung (Scimitar) syndrome, an anomalous unilateral single PV (the term meandering PV has also been used to refer to this condition), and collateral pulmonary vein.

The patient has remained well at subsequent clinic follow-up.

ABSTRACT

CASE PRESENTATION

INVESTIGATIONS

DIFFERENTIAL DIAGNOSIS

OUTCOME AND FOLLOW-UP
DISCUSSION

It is well-known that PV stenosis and pulmonary venous hypertension or venous infarction are relatively uncommon complications of radiofrequency ablation therapy for AF.1,2

An anomalous intrapulmonary venous connection is rare after acquired PV stenosis.4

In the present study, ECG-gated multidetector CT clearly revealed left upper PV stenosis and an anomalous intrapulmonary venous connection. An anomalous intrapulmonary venous connection was distinguished from pulmonary arteriovenous malformation and hypogenic lung syndrome (Scimitar syndrome). Arteriovenous malformation and hypogenic lung syndrome may require surgery or embolization to correct the shunt. In contrast, an anomalous intrapulmonary venous connection has no vascular shunt. This case was managed conservatively because the patient presented with mild symptoms such as hemoptysis and cough once and then became asymptomatic. An anomalous unilateral single PV is a rare congenital pulmonary venous abnormality where a single vein enters ipsilaterally into the left atrium after receiving from all the PVs.5 An anomalous unilateral single PV requires no treatment because no vascular shunt is produced.5,6

Lung parenchyma showed interstitial septal thickening, parenchymal bands, peripheral consolidation in the anterior and lingular segment of the LUL was considered pulmonary venous hypertension, venous infarction and alveolar hemorrhage,1–4 1 year and 3 months after the second catheter ablation therapy (Figure 2).

The newly appeared nodular ground glass opacities at the apicoposterior segment LUL, including the peripheral opacities and interstitial septal thickening in the LUL, disappeared, 3 years, 11 months after the second catheter ablation therapy (Figure 3). A follow-up CT 5 years after the second catheter ablation therapy confirmed an almost complete resolution of the lung parenchymal abnormalities and the anomalous pulmonary venous connection was more obvious (Figure 4). The CT findings of the lung parenchyma and the anomalous PV changed over time, and the anomalous tortuous vein may compensate for intrapulmonary venous drainage.

Anatomically, PVs and bronchial veins are interconnected through already existing bronchial venous plexuses. These
plexuses are located in the bronchial wall and the peribronchovascular connective tissues. In this case, left upper PV stenosis was the cause of high PV pressure in the LUL. We speculated that left upper PV and the bronchial venous plexuses were interconnected, while bronchial venous plexuses and the other left lower PV were also interconnected. The PV stenosis gradually led to an anomalous intrapulmonary venous connection as an intrapulmonary collateral.

A collateral pulmonary vein after catheter ablation therapy for atrial fibrillation is rare. Recognition of this condition is important. ECG-gated multidetector CT can clearly reveal an acquired anomalous intrapulmonary venous connection; therefore, we should prevent the use of invasive diagnostic and therapeutic procedures.

**LEARNING POINTS**

1. The presented case illustrates the emergence of an anomalous intrapulmonary venous connection after catheter ablation therapy.
2. Little is known about a collateral pulmonary vein after catheter ablation therapy, but recognition of this condition is important. The finding of a venous collateral has no vascular shunt, therefore, we should prevent the use of invasive diagnostic and therapeutic procedures.
3. The differential diagnosis for anomalous intrapulmonary venous connection was pulmonary arteriovenous malformation, hypogenetic lung (Scimitar) syndrome, an anomalous unilateral single PV (the term *meandering PV* has also been used to refer to this condition), and collateral pulmonary vein.

**DISCLOSURES**

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.
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