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CASE REPORT

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Association between epicardial adipose tissue and coronary artery vasospasm during pulmonary vein isolation

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Abstract

Coronary artery vasospasms (CAVs) during pulmonary vein isolation have been reported, but the mechanism remains unclear. We experienced a rare case of CAVs caused by radiofrequency (RF) applications to sites with massive epicardial adipose tissue (EAT) attached. Because EAT contains ganglionated plexuses, RF application may have caused an autonomic nervous system imbalance, which thereby provoked severe CAVs.

KEYWORDS
atrial fibrillation, catheter ablation, coronary artery vasospasm, epicardial adipose tissue

1 | INTRODUCTION

Coronary artery vasospasms (CAVs) during pulmonary vein isolation (PVI) have been reported a rare complication[1]; however, the mechanism is unclear. The epicardial adipose tissue (EAT) of the left atrium (LA) has been reported to contain a ganglionated plexus (GP) and associated with the severity of atrial fibrillation (AF). [2] We report on a case of CAV caused by radiofrequency (RF) applications to LA sites with massive EAT attached.

2 | CASE REPORTS

A 64-year-old man with drug-resistant paroxysmal AF was referred to our center for catheter ablation. He had no history of coronary artery disease, or ischemia-related symptoms, and no coronary risk factors. Before catheter ablation, we evaluated EAT surrounding LA using an enhanced computed tomography scan. We used with dual-source 64-slice multidetector computed tomography scanner and transferred the images to a NavX mapping system (Ensite-Verismo, St.Jude Medical).

A point-by-point RF application was initiated at the posterosuperior site of the right superior pulmonary vein (PV). Thirty seconds after initiating the RF application at the anterior site of the right inferior PV, he developed a sinus bradycardia (heart rate about 40 bpm) and his systolic blood pressure rapidly dropped to 80 mm Hg, and a ST elevation occurred in the inferior leads of the 12-lead electrocardiogram (Figure 1A). Urgent coronary angiography revealed diffuse and severe stenosis of the right coronary artery (Figure 1B). We diagnosed CAVs because stenosis was immediately improved by a nitrate injection (Figure 1C). An investigation of the regional distribution of EAT revealed that the RF application site that provoked CAV was attached to a large section of EAT (Figure 2). After a nitrate injection, CAVs improved and his systolic blood pressure and heart rate were recovered. We have performed PVI procedure carefully under the continuous infusion of the nicorandil. An acetylcholine provocative test conducted later also caused CAV, and the patient was prescribed permanent vasodilator intake to prevent the recurrence of CAVs.

3 | DISCUSSION

CAVs during PVI have been reported to be a rare complication, but the pathophysiological mechanism remains unclear.[3] An air embolism can reduce the blood flow in the coronary artery; however, in this case coronary angiography did not reveal any embolisms. We

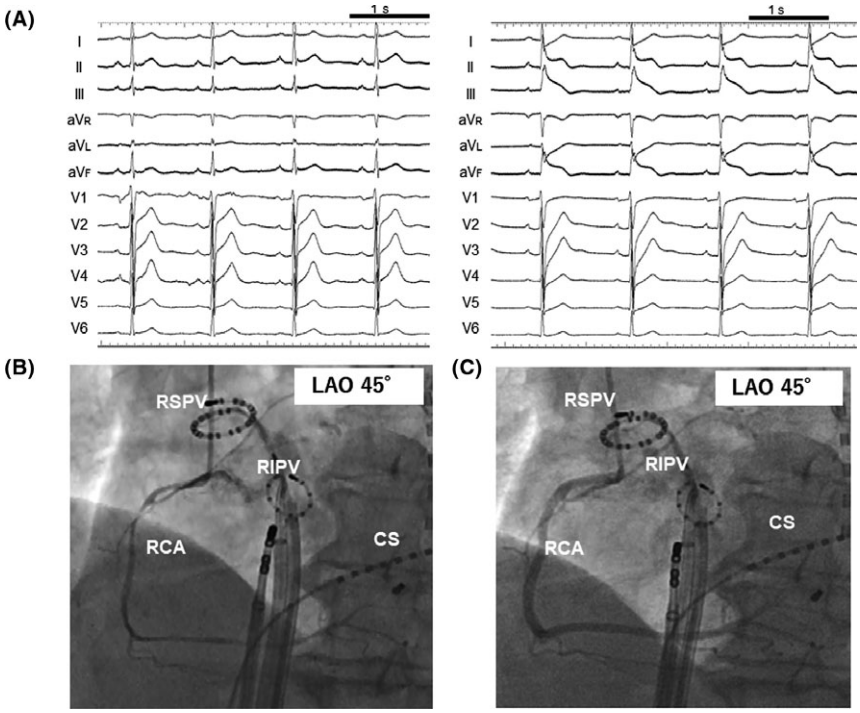

FIGURE 1 A, Left, baseline 12-lead electrocardiogram. Right, after RF application at the anterior site of RIPV, the ST segment elevation occurred in inferior leads. RIPV, right inferior pulmonary vein. B, Coronary angiography revealed a severe stenosis of RCA. C, Nitrate administration resulted in an immediate improvement of the coronary blood flow. RCA, right coronary artery; LAO, left anterior oblique

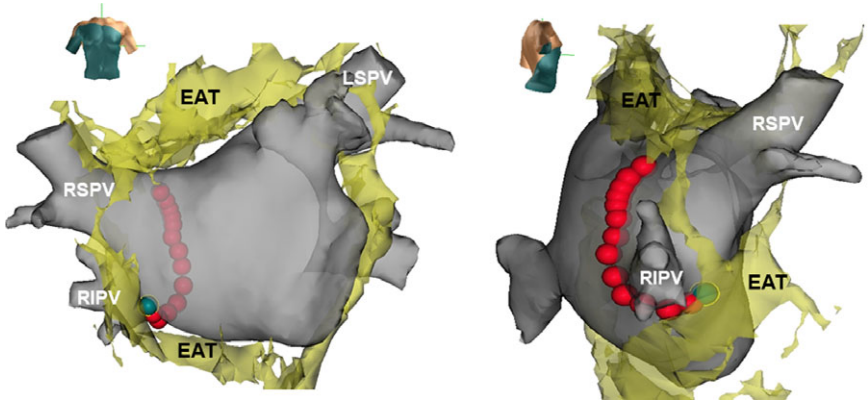

FIGURE 2 The RF application site attached to large amounts of EAT provoked CAVs. The red dots depict the ablation line of PVI. The blue dot represents the ablation site at which RF application provoked CAVs. EAT, epicardial adipose tissue; RSPV, right superior pulmonary vein; RIPV, right inferior pulmonary vein

diagnosed CAVs because the coronary blood flow was immediately improved by a nitrate injection.

This case suggested two clinically important issues about CAVs during PVI. First, the modulation of autonomic nervous system might be one mechanism of CAVs during PVI. Takahashi et al[2] have reported that LA-EAT overlays more than 90% of the five major anatomical GP areas, including vagal response sites identified by high-frequency stimulation. Endocardial RF applications to EAT sites affected the epicardial GPs through thermal injury, which caused an autonomic nervous system imbalance. Hishikari et al[4] reported that severe coronary artery vasospasm during PVI procedure. When they performed RF application at the posteroinferior aspect of the right inferior PV, a severe CAVs of the right coronary artery occurred. The ablation sites causing CAVs were presumed areas of the inferior right GP located. In our case, the patient's heart rate and blood pressure were immediately dropped by vagal effect promptly after the application to EAT site. There were several reports about the relationship between coronary artery vasospasm and autonomic nervous system. Lanza et al[5] also reported that the autonomic changes associated with spontaneous coronary spasm in patients with variant angina. Drastic changes and imbalances in autonomic nervous system activity due to RF ablation at EAT sites contained epicardial GPs may have caused CAVs.

Second, careful monitoring should be performed using a 12-lead electrocardiogram during RF application to EAT attached site for an early identification of any ischemic changes. Particularly because vagal reactions may be the initial signs of the autonomic nervous system imbalance, careful monitoring of the ST segment in the 12-lead electrocardiogram was useful for the early identification. RF applications to EAT attached sites did not provoke CAVs in all patients. The acetylcholine provocative test also caused CAVs of the right coronary artery in this patient, which suggests that the patient had a predisposition for CAV provoked by PVI.

4 | CONCLUSION

This case study suggests that careful 12-lead electrocardiogram monitoring should be performed to facilitate the early identification and management of any ischemic changes or vagal reactions during RF application to LA sites with EAT attached.

CONFLICT OF INTEREST

Authors declare no conflict of interests for this article.

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