Chronic Left Coronary Artery Stenosis After Radiofrequency Ablation of Idiopathic Premature Ventricular Contraction Originating From Left Coronary Sinus Cusp

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A 50-year-old man was referred for radiofrequency catheter ablation of idiopathic premature ventricular contraction (PVC) refractory to metoprolol. The ECG characteristics of the PVCs suggested that the PVCs originated from the left coronary sinus (LCC). Mapping was performed in the LCC using a 7F irrigated-tip ablation catheter via the right femoral artery. During clinical PVCs, the local ventricular activation was the earliest at the LCC. Radiofrequency (RF) energy application was attempted at the site (Figure [A1] and [B1]). Coronary angiography was performed before RF application, revealing a normal left coronary artery without stenosis. The distance from the catheter tip to the left main coronary artery (LMCA) ostium was \( \approx 10 \text{ mm} \) by fluoroscopy (Figure [A2] and [B2]). One application of RF energy at this site (43°C, 30 W, 17 mL/min) terminated spontaneous PVCs within 15 s and was maintained for 180 s. However, the clinical PVCs appeared during isoproterenol infusion. Another 3 applications around this point with a maximal setting of 40 W were 180 s in duration and finally eliminated the PVCs. Mean impedance drop was 12±2 Ω for the lesions. Popping or impedance rises did not occur during RF applications. No catheter dislodgment was observed during ablation, and ablation was performed only within the LCC. The patient experienced some chest discomfort during ablation. Repeat angiography after ablation showed normal left coronary artery without coronary spasm, stenosis, or occlusion. The ECG after the procedure showed normal sinus rhythm and no ST-T changes. The patient was then discharged on aspirin for 1 month. During the 3 months of follow-up, the patient remained clinically free of symptoms. Four months later, the patient was readmitted because of exertional chest pain. The chest pain had developed 3 weeks previously when he was climbing stairs, which was relieved by several minutes of rest. A 12-lead ECG recorded on admission showed sinustachycardia and remarkable ST-segment depression in leads V4 through V6 and ST-segment elevation in lead aVR. The patient’s peak troponin level was 1.7 ng/mL. His creatine kinase-MB was not increased. Coronary angiography showed 60% stenosis in the body and distal segment of LMCA and 90% stenosis in the proximal left anterior descending artery (LAD) with TIMI 3 flow (Figure [C1] and [D1]). The left circumflex artery and right coronary artery were normal. Balloon predilation with a 2.5×20 mm balloon and a 3.0×10 mm cutting balloon was performed. A 4.0×24 mm sirolimus-eluted stent was then deployed from the LAD to the LMCA with intravascular ultrasound guidance (Figure [C2] and [D2]). A final angiogram after LMCA stenting showed an optimal angiographic result. Intravascular ultrasound revealed positive remodeling characteristics in the LAD lesion, whereas the external elastic membrane cross-sectional area of LAD lesion was significantly greater than that of the LMCA lesion (Figure [E] and [F]). The therapy resulted in significant symptom relief. During the 12 months of follow-up after stenting, the patient remained clinically free of symptoms without any ischemic events. Coronary computed tomography showed stent patency at 10 months of follow-up.

Although most coronary damage in relation to RF ablation presents acutely, delayed presentations may also occur. To our knowledge, this is the first report of chronic LMCA and LAD stenoses without occlusion secondary to previous RF ablation. This study also detected the coronary plaques associated with the application of RF energy in a human subject, via intravascular ultrasound. Only 3 cases of acute LMCA occlusion caused by RF-induced thermal injury have been reported. Only 1 case report described chronic LMCA occlusion caused by vessel trauma presenting 2 years after ablation of left ventricular tachycardia. Only 1 case report described chronic ostial LMCA stenosis of after LCC atrial tachycardia. Evidence of chronic injury associated with the application of RF ablation near the coronary arteries has been observed in animal models. In a porcine model, RF ablation proximal to the coronary arteries led to chronic histopathologic changes, characterized by tunica intima and media thickening with replacement of the smooth muscle cells with extracellular matrix; however, no significant stenosis was observed up to 70 days after ablation. In the present study, significant LMCA and LAD stenoses developed 4 months after RF ablation. Intravascular ultrasound showed positive remodeling characteristics in the LAD lesion. Positive remodeling is strongly associated with plaque rupture. This is the first report documenting the progression of LMCA and LAD plaques associated with thermal

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injury in humans by angiography and intravascular ultrasound. Although the exact time of positive remodeling was not clear, the present study clearly suggests that RF ablation proximal to the coronary arteries may facilitate the development of intra-coronary atherosclerotic plaque and rupture of spontaneous plaque. As proposed by most authors, a distance of >1.0 cm from the ablation catheter tip to the left coronary ostium seems to be safe. However, repetitive RF application at high power and for a long duration in the LCC may lead to coronary injury although the distance of >1.0 cm was suggested to be adequate.

Disclosures
None

References

KEY WORDS: catheter ablation, coronary artery disease, coronary stenosis, catheter ablation, radiofrequency, ventricular premature complexes

Figure. The fluoroscopic position of the ablation site, as well as coronary angiography and intravascular ultrasound of the left coronary artery. Left anterior oblique (LAO; A1) and right anterior oblique (RAO; B1) fluoroscopic views showing the radiofrequency ablation site in the left coronary sinus. Coronary angiography performed before ablation in an LAO projection (A2) and an RAO projection (B2), showing a normal left coronary artery without stenosis. The distance from the catheter tip to the left main coronary artery (LMCA) ostium was ~10 mm. Left coronary angiography was performed once again after the ablation procedure, and ruled out coronary spasms, stenoses, and occlusions. Coronary angiography performed 4 mo after ablation in an RAO+cranial (CRA) projection (C1) and an LAO+caudal (CAU) projection (D1), showing 60% stenosis in the body and distal segment of LMCA and 90% stenosis in the proximal left anterior descending artery (LAD) with TIMI 3 (thrombolysis in myocardial infarction) flow. Repeat left coronary angiography after successful stenting without residual stenosis in an RAO+CRA projection (C2) and an LAO+CAU projection (D2). Intravascular ultrasound performed after stenting, showing positive remodeling characteristics in the LAD lesion (remodeling index=1.48). The external elastic membrane cross-sectional area (EEM CSA=23.49 mm²) of the LAD lesion (E) was significantly greater than that of the LMCA lesion (EEM CSA=15.89 mm²) (F). RI indicates remodeling index.
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