The first reported case of a transvenous left ventricular pacing lead of cardiac resynchronization therapy causing aggravation of coronary atherosclerosis

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A 64-year-old male underwent aortic valve replacement surgery in his 40s. His cardiac dysfunction continued to progress, and a cardiac resynchronization therapy-defibrillator (CRT-D) was implanted when he was 61 years old. The patient had few coronary veins of suitable size available for implantation of the left ventricular (LV) lead and, therefore, the tip of the bipolar lead was pushed and wedged into a small branch in the lateral position of the atrioventricular groove. Coronary arteriography (CAG) following CRT-D implantation did not show coronary stenosis, but retrospectively uncovered a dimple in the left circumflex coronary artery (LCx) wall underneath the LV lead.
at systole only (Panels A and B). Three years later, the patient experienced several CRT-D discharge events for ventricular tachycardia (VT) with a right bundle branch block/right-axis morphology pattern and a QS pattern in lead I (Panel D). The LV lateral wall was determined to be the VT origin. The VT was induced repeatedly by double extra-stimuli from the CRT-D device. Computer tomography revealed a configuration of the epicardial lead running upon the LCx and poor contrast enhancement of the LCx at that point (Panel E). A subsequent CAG showed severe stenosis of the LCx (Panel C), which was identified by an intravascular ultrasound as an atherosclerotic plaque, rather than a direct compression of the LV lead (Panel F). After a balloon angioplasty, the VT was no longer inducible, and CRT-D discharge has not been observed during a 9-month follow-up period.

An LV pacing lead of the CRT is commonly inserted and left in a coronary vein that runs on the epicardium and intersects with coronary arteries. There are reports that an electrode lead detained surgically on the epicardium could constrict the coronary artery during heart growth or cardiac dilatation and cause angina pectoris. However, the influence of the transvenous implantation lead on the neighboring coronary arteries has not been studied.

This case suggests that the long-term contact and pressure to the coronary artery wall can affect fluid shear stress and cause damage to the coronary artery intima, resulting in an aggravation of coronary atherosclerosis. Therefore, when the position for the implantation of an LV lead is determined or when a coronary lesion is suspected to have progressed following implantation of the CRT device, it is important to consider the influence of the implanted LV lead on coronary atherosclerosis.

Supplementary material is available at Europace online.

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References