Idiopathic ventricular fibrillation or myocardial infarction? The impact of optical coherence tomography on therapeutic decisions

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A 47-year-old woman was referred from a district hospital for implantable cardioverter--defibrillator (ICD) insertion as secondary prevention. Cardiac arrest occurred a week earlier in the course of ventricular fibrillation and was effectively treated with defibrillation. The event was preceded by chest pain. Electrocardiography showed ST-segment elevation in inferior leads. Coronary angiography did not show any stenosis of coronary vessels (FIGURE 1A-1C). The patient's medical history revealed mitral and tricuspid valve surgery related to insufficiency 10 years earlier. No risk factors for ischemic heart disease were noted. While hospitalized, she underwent echocardiography, which showed akinesia of the basal inferior wall with maintained left ventricular ejection fraction (55%). Cardiac magnetic resonance (CMR) imaging demonstrated delayed transmural contrast enhancement in the basal and medial segments of the inferolateral wall (FIGURE 1D and 1E). Subsequently, coronary angiography with intracoronary optical coherence tomography (OCT) was performed (FIGURE 1F). It showed ruptured atheromatous plaque in the initial segment of the circumflex artery with a minor mural thrombus, which did not cause any significant lumen stenosis of the coronary vessel (FIGURE 1D). The patient was deemed eligible for intensive conservative treatment.

The insertion of ICD was waived because of myocardial infarction diagnosed based on electrocardiography (ECG) evolution, contractility disorders corresponding with ECG findings, and the transmural scar on CMR imaging. The patient was discharged home in a good general condition and was recommended to follow dual

antiplatelet therapy and to intensify cholesterol-lowering therapy. In patients with a history of sudden cardiac arrest or an incident of life--threatening cardiac arrhythmia, the implantation of a cardioverter-defibrillator is associated with arrhythmia-related mortality decreased by 50%. The reversible causes of arrhythmia such as myocarditis or dyselectrolytemia are contraindications to ICD insertion for the prevention of secondary sudden cardiac death. It is also not recommended if arrhythmia occurred within the first 48 hours following myocardial infarction.2 In our patient, despite the changes found on ECG, which were suggestive of ST--segment elevation myocardial infarction, coronary angiography did not show any abnormalities in the coronary vessels. Consequently, idiopathic ventricular fibrillation was diagnosed, and the patient was referred for ICD insertion as a secondary preventive measure against sudden cardiac arrest. However, the reported typical thoracic pain preceding cardiac arrest suggested an ischemic condition. Moreover, CMR imaging confirmed the presence of a subendocardial scar of ischemic origin in the left ventricular muscle. Myocardial infarction with nonobstructive coronary arteries is diagnosed in approximately 10% of cases in which coronary angiography does not detect any significant atherosclerotic stenoses and clinical symptoms strongly imply the ischemic origin.3 Apart from magnetic resonance imaging, intracoronary imaging (OCT, intravascular ultrasound) provides necessary diagnostic tools for suspected myocardial infarction with nonobstructive coronary arteries.4 Due to high image resolution, OCT is

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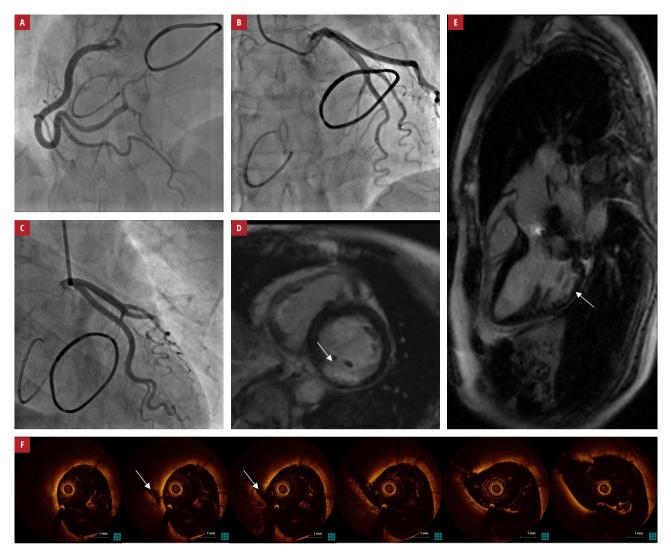


FIGURE 1 Imaging of a patient after cardiac arrest: **A–C** – coronary angiography showing coronary arteries with no significant lesions; **D**, **E** – cardiac magnetic resonance imaging demonstrating the subendocardial scar in the left ventricle (arrows); **F** – optical coherence tomography showing plaque rupture (arrows)

the gold standard modality for atheromatous plaque evaluation. The obtained results help to establish the proper diagnosis and to determine the optimal treatment.

ARTICLE INFORMATION

CONFLICT OF INTEREST None declared.

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