

Multivessel spontaneous coronary artery dissection as a cause of acute coronary syndrome: An often-forgotten differential diagnosis

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A 55-year-old female patient with no history of cardiovascular disease was admitted to our hospital with acute retrosternal chest pain. Her electrocardiogram showed ST-segment elevation in leads I, aVL, and V2–V4, with ST-segment depression in leads II, III, aVF, and V6. The patient was diagnosed with ST-segment elevation myocardial infarction, and urgent coronary angiography was performed. It showed multivessel spontaneous coronary artery dissection (SCAD) type 2a of the left

anterior descending artery (LAD) and SCAD type 2b of the posterolateral branch from the right coronary artery (Figure 1A–C). Moreover, intravascular ultrasound was performed, which, aside from confirming dissection of the LAD, showed no signs of atherosclerosis. Her laboratory results demonstrated markedly elevated creatine kinase myocardial band and troponin-T levels and hypercholesterolemia.

Echocardiography showed akinesis of the apex and apical segments of all walls,

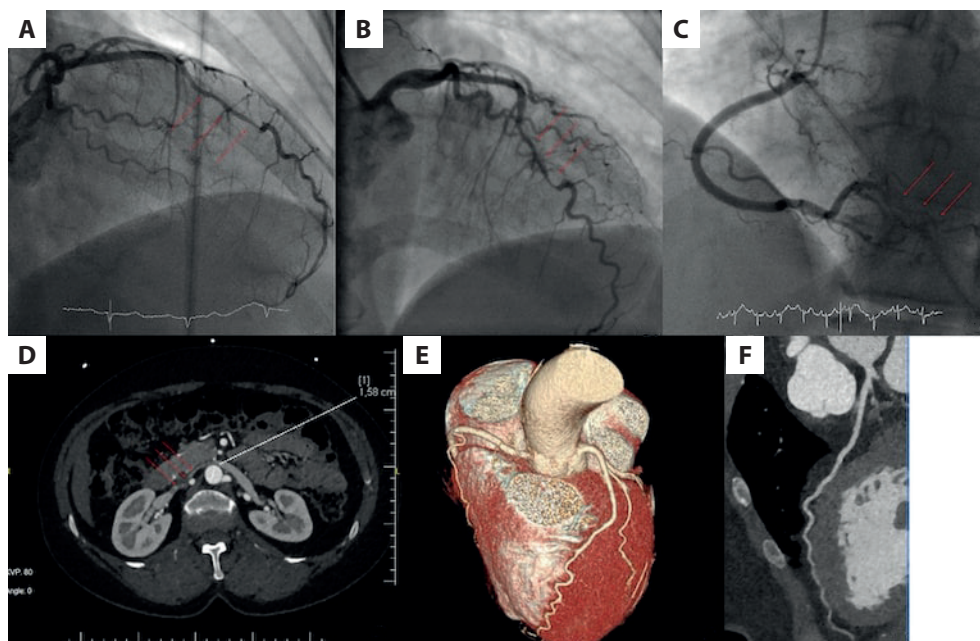


Figure 1. A–B. Coronary angiography showing spontaneous coronary artery dissection (SCAD) of the left anterior descending artery. C. Coronary angiography showing SCAD of the posterolateral branch from the right coronary artery. D. Computed tomography (CT) showing the right renal artery with morphology characteristic of fibro-muscular dysplasia. E–F. CT showing complete healing of coronary arteries

hypokinesis of medial segments of the anterior wall with hyperkinesis of basal segments, and a left ventricular ejection fraction of 45%–50%. Due to the presence of SCAD in coronary angiography and the patient's history of migraine headaches, additional imaging, including computed tomography of the cerebral, carotid, and vertebral arteries, was performed, in which dissection of intracerebral arteries was excluded, and a common origin of the left common carotid artery and the brachiocephalic trunk was revealed. Moreover, a Doppler-ultrasound examination of renal arteries showed no features of fibro-muscular dysplasia. The patient was treated conservatively with dual antiplatelet therapy, a statin, angiotensin-converting enzyme inhibitor, and beta-blocker. However, due to bradycardia, the beta-blocker was withdrawn. Control echocardiography showed an improvement in left ventricular systolic function, and she was discharged in good condition. During a follow-up visit, 3 months later, computed tomography was performed, which visualized the right renal artery with morphology characteristic of fibro-muscular dysplasia and complete healing of the coronary arteries (Figure 1D–E). Therefore, dual antiplatelet therapy was modified into single antiplatelet therapy with aspirin only.

This case shows multivessel SCAD as a cause of acute coronary syndrome. It highlights the importance of considering SCAD in the differential diagnosis of chest pain, particularly in middle-aged women and patients with few conventional atherosclerotic risk factors. Since a spontaneous recovery of the dissected artery is observed in the vast majority of patients, a conservative strategy is generally favored over revascularization [1, 2]. Diagnosing SCAD with the use of coronary angiography may be in some cases challenging, therefore, intravascular ultrasound or optical coherence tomography can be helpful [3]. Moreover, imaging of extra-coronary arteriopathies is advised in SCAD

patients [2]. Although there is currently greater awareness of this condition, SCAD remains underdiagnosed and poorly understood, with few studies evaluating management strategies, assessing the presence and impact of predisposing and precipitating causes, or assessing its effects on short and long-term cardiovascular prognosis [1, 2, 4].

Supplementary material

Supplementary material is available at https://journals.viamedica.pl/kardiologia_polska.

Article information

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