A young woman with recurrent spontaneous coronary artery dissection

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A 42-year-old woman was admitted to the hospital with non–ST-segment elevation myocardial infarction. Her medical history revealed that she suffered from 3 spontaneous abortions, migraine episodes, esophageal reflux, and asthma. The family history was negative for cardiovascular disease.

On admission, the patient was hemodynamically stable. Electrocardiography showed sinus rhythm at a heart rate of 73 bpm, incomplete right bundle branch block, and negative T waves in leads II, III, aVF, and V_4 through V_6. Blood tests demonstrated elevated levels of high-sensitive troponin (895 ng/l; reference range <14 ng/l), creatine kinase–myocardial band (612 U/l; reference range <170 U/l), and N-terminal fragment of the prohormone brain natriuretic peptide (137 ng/l; reference range <130 ng/l), yet normal levels of low-density lipoprotein cholesterol (2.5 mmol/l; reference range <3 mmol/l).

Echocardiography demonstrated a normal-sized, nonhypertrophic left ventricle (LV) with normal LV ejection fraction (biplane LVEF, 61%) and hypokinesia of the LV inferolateral, inferior midventricular, and inferior apical segments. Coronary angiography (CA) revealed a spontaneous dissection of the distal circumflex artery (FIGURE 1A).

Due to preserved coronary perfusion and no persistent or recurrent angina, the patient was treated conservatively with dual antiplatelet therapy (aspirin for lifetime use and ticagrelor for 12 months) and a β-blocker (bisoprolol).

The following conditions were considered as the probable causes of spontaneous coronary artery dissection (SCAD): fibromuscular dysplasia (FMD), undefined hereditary vasculopathy, and systemic inflammatory and connective tissue disorders.[1,2] A comprehensive diagnostic workup was performed and the diagnosis of FMD was established based on radiological findings (FIGURE 1B). The patient was discharged after 5 days.

A few hours later, the patient was readmitted with anterior ST-segment elevation myocardial infarction (FIGURE 1C). Emergency CA revealed a new SCAD of the middle-to-distal left anterior descending artery (with Thrombolysis in Myocardial Infarction flow grade 2/3; FIGURE 1D). The patient received conservative treatment with intravenous nitroglycerin and intravenous heparin (at the therapeutic dose) in addition to the previous pharmacotherapy.

Cardiac magnetic resonance imaging showed transmural late gadolinium enhancement with microvascular obstruction and concomitant edema in the left anterior descending artery territory and a small, old infarct scar in the circumflex artery territory; LVEF was 39% (FIGURE 1E and 1F; Supplementary material, *Video S1*).

After an interdisciplinary consultation, the patient was anticoagulated with phenprocoumon for 6 months and concomitantly received aspirin (lifelong use), bisoprolol, lisinopril, rosuvastatin, and pantoprazole. Besides anemia due to recurrent menorrhagia, no other clinical problems occurred during the 2-year follow-up. Follow-up echocardiography showed a dilated LV with slightly reduced LVEF (biplane, 45%).

Spontaneous coronary artery dissection accounts for about 1/3 of acute myocardial infarction cases in women under 50 years of age.[1,2] The prognosis following SCAD seems to be good, as the dissection usually heals completely over a few months, although a significant recurrence

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Received: May 16, 2020.
Revision accepted: July 13, 2020.
Published online: July 24, 2020.
Kardiol Pol. 2020; 78 (10): 1059-1061
doi:10.33963/KP.15526

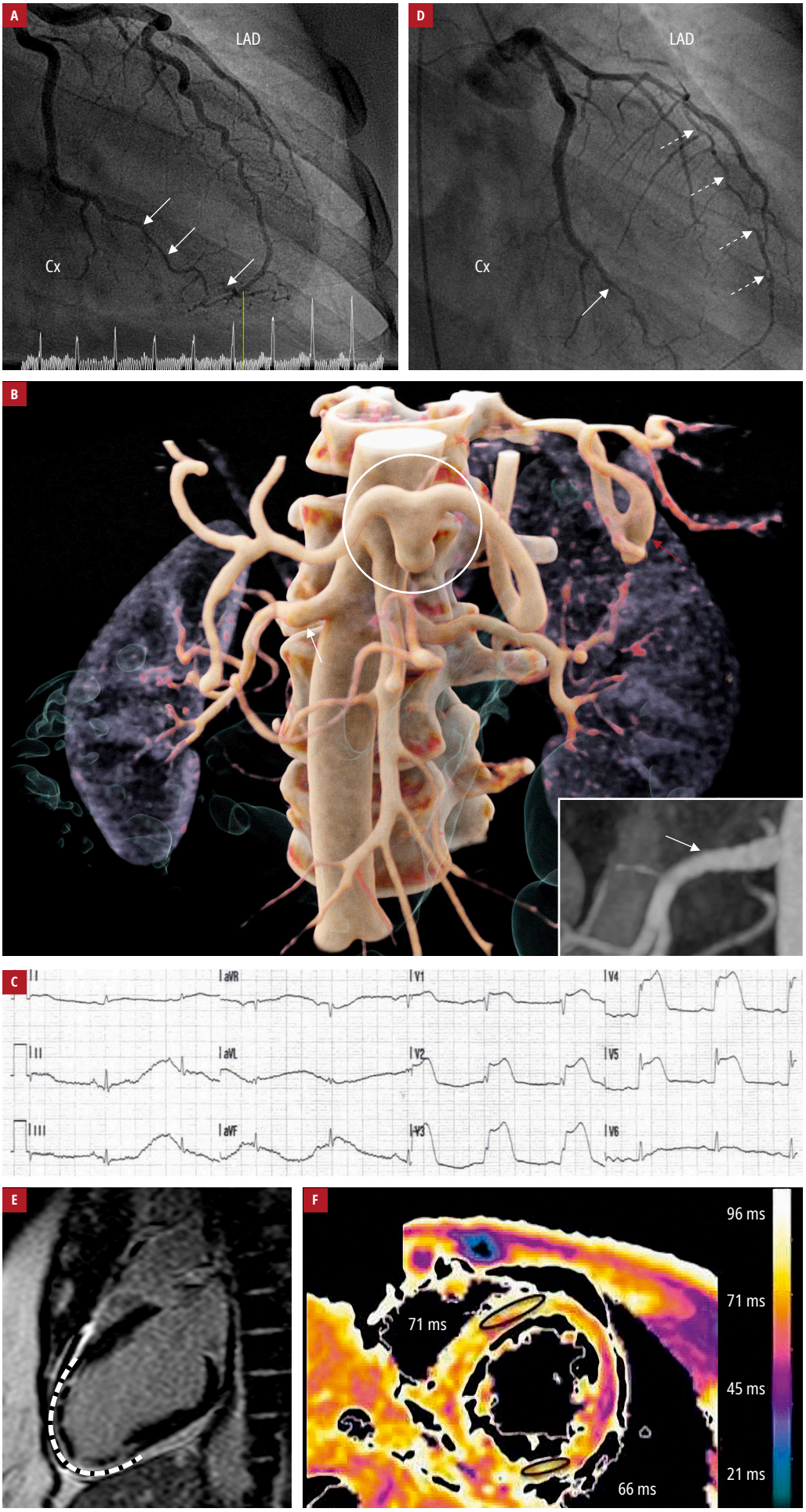

FIGURE 1 **A** – coronary angiography on admission: spontaneous dissection of the first marginal branch of the circumflex artery (arrows); **B** – extracoronary abnormalities on computed tomography angiography of the abdomen and pelvis: an ectatic celiac trunk (circle), an aneurysm of the distal splenic artery (red arrow), ectasia of the left iliac internal artery (not shown) and a "string of beads" appearance of the right renal artery (arrow and magnified picture); **C –** 12-lead electrocardiogram showing ST-segment elevation in all anterior wall leads (V_1 through V_5); **D** – emergency coronary angiography: a new spontaneous dissection of the left anterior descending artery (dotted arrows) and a residual dissection in the distal circumflex artery (arrow); **E**, **F** –cardiac magnetic resonance imaging showing transmural late gadolinium enhancement in the anterior and inferior apical segments of the left ventricle with microvascular obstruction (**E**: 2-chamber view, dashed line) and edema in all midventricular segments except the anterolateral ones (**F**: T2 mapping, short-axis view)
Abbreviations: Cx, circumflex artery; LAD, left anterior descending artery

rate is observed.[1,2] Conservative management is suggested in patients with preserved flow in the coronary arteries, followed by prolonged inpatient monitoring.[1-4] Pharmacotherapy and its duration in SCAD remain controversial, and the benefits from antiplatelet, anticoagulant, and statin therapies are still a matter of debate.[1,4] Evaluation for underlying causes is recommended, including screening for extracoronary arteriopathy such as FMD; however, in some patients, no definitive diagnosis of the underlying cause can be made at the time of SCAD occurrence.[1,2,5]

SUPPLEMENTARY MATERIAL

Supplementary material is available at www.mp.pl/kardiologiapolska.

ARTICLE INFORMATION

ACKNOWLEDGMENTS SJM is a beneficiary of the Polish National Agency for Academic Exchange (NAWA) in 'The Iwanowska Programme' at the Department of Cardiology, MRI Cardiac Unit, University Hospital Zurich, Switzerland.

CONFLICT OF INTEREST None declared.

HOW TO CITE Sokolska JM, Meier LM, Holy EW, et al. A young woman with recurrent spontaneous coronary artery dissection. Kardiol Pol. 2020; 78: 1059-1061. doi:10.33963/KP.15526

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