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Multivessel spontaneous coronary artery dissection with angiographic documentation before and after the acute event

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A 49-year-old woman with a history of rheumatic heart disease treated with mitral valve replacement and tricuspid annuloplasty 11 years ago was admitted for chest pain. ECG showed no significant ST-T changes compared with previous ECGs (Fig. 1). Troponin I was 2.08 ng/ml (upper normal limits: 0.30 ng/ml) 8 h after the onset of chest pain, consistent with a non-ST segment elevation myocardial infarction. She became chest pain-free after intravenous morphine (4 mg). Coronary angiography revealed no significant stenoses (Fig. 1, Supplemental digital content 1, <http://links.lww.com/MCA/A31>). Because recent transesophageal echocardiography revealed severe prosthetic mitral valve stenosis, moderate tricuspid regurgitation, and pulmonary hypertension, she was scheduled for repeat valve surgery. Eight days later, she again experienced chest pain, but with anterolateral ECG changes that were not present on admission (Fig. 1). Coronary angiography showed new diffuse stenoses in the left anterior descending artery, left circumflex artery, ramus, and obtuse marginal arteries along with perivascular contrast staining in the ramus consistent with a spontaneous coronary artery dissection (SCAD) (Fig. 1, Supplemental digital content 2, <http://links.lww.com/MCA/A32>). Intravascular ultrasound (IVUS) of the left anterior descending artery and ramus demonstrated a long medial dissection with intramural hematoma, but little atherosclerotic plaque (Fig. 2, Supplemental digital contents 3, <http://links.lww.com/MCA/A33> and 4, <http://links.lww.com/MCA/A34>). Because there was no flow-limiting obstruction after intracoronary nitroglycerin and her symptoms subsided, she was treated medically. After echocardiographic re-evaluation, prosthetic mitral valve stenosis was not deemed to be critical; and surgery was canceled. One year later, she is asymptomatic; and follow-up coronary angiography has not been done.

SCAD may have happened at the time of initial admission. However, coronary angiography was not remarkable, the ECG did not show ischemic changes, and IVUS and optical coherence tomography were not performed at that time. Vanzetto *et al.* [1] have reported that

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Conflicts of interest

There are no conflicts of interest.

in female patients below 50 years old with an acute coronary syndrome, the prevalence of SCAD increased up to 8.7%.

At the time of recurrent chest pain in this patient, coronary angiography demonstrated progression of multivessel stenosis, confirmed as SCAD by IVUS. Tweet *et al.* [2] reported that 10% of the conservative-treated SCAD patients experienced recurrent chest pain, ischemia, and angiographic findings of SCAD progression within 2–7 days of initial presentation.

We report multivessel SCAD with ECGs and angiography both before and after clear evidence of SCAD that was confirmed by IVUS.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Fig. 1.

Coronary angiography and ECG. The coronary angiogram at the time of admission revealed normal coronary artery anatomy with no ischemic ECG changes (left panel). However, repeat coronary angiography (right panel) 8 days later after prolonged chest pain with ECG changes (asterisks) showed extensive and diffuse reduction in lumen calibers in the left anterior descending artery, left circumflex artery, ramus, and obtuse marginal arteries (white arrows), with linear perivascular contrast staining in the ramus (white triangle).

Fig. 2.

Coronary angiography with corresponding IVUS images 8 days later. IVUS demonstrated medial dissection that was circumferential in the LAD (h–j) with intramural hematomas in the ramus (white arrows, b–e) and in the LAD (white arrows, g–j). There was little atherosclerotic plaque (f) in the ramus and (k) in the LAD was almost normal in appearance as was the proximal LMCA (a). IVUS, intravascular ultrasound; LAD, left anterior descending artery; LMCA, left main coronary artery.