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CLINICAL IMAGE

Ventricular septal rupture in a patient with non-ST-segment elevation myocardial infarction caused by myocardial bridge

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A 68-year-old woman with a non-ST-segment elevation myocardial infarction (NSTEMI) was admitted to our department for emergency coronary angiography. She had a history of 2 NSTEMIs without coronary artery stenosis on coronary angiography. On admission, she was hypotensive but hemodynamically stable. Auscultation did not reveal any pathological heart murmurs. An electrocardiogram showed negative T waves in leads II, III, aVF, and V_5–V_6. On coronary angiography, the only abnormality was a myocardial bridge (systolic stenosis, 60%–70%) of the left anterior descending coronary artery (FIGURE 1AB). Further conservative treatment was scheduled. Since the recorded values of creatine kinase (CK) / CK-MB (245/51 IU/l) and troponin I (1.79 µg/l; cut-off value, 0.01) were the highest on admission, we estimated that the MI occurred 2 days earlier.

On the next day, a new holosystolic murmur appeared. Echocardiography[1] showed a 10-mm dropout in the apical part of the septum, with turbulent left-to-right transseptal flow with a peak gradient of 53 mmHg (FIGURE 1C–H). The size of the left ventricle (LV) was normal, with regional septoapical akinesis, but with an ejection fraction of 80% due to hyperkinesia of all segments. The right ventricle (RV) was significantly enlarged (5.8 cm in an apical 4-chamber view) with a 7-mm gap in tricuspid valve leaflet coaptation and severe tricuspid regurgitation (vena contracta, 8 mm). The maximum RV–right atrium (RA) gradient was 50 mmHg. Another finding was an intraventricular gradient (maximal flow velocity, 2.5 m/s) in the LV at the level of mid-cavity segments, typical for a systolic narrowing of the LV; interestingly, the systolic flow was directed towards the LV apex. An intraaortic balloon pump was immediately inserted.[2,3] A cardiac surgeon decided to postpone the closure of the ventricular septal rupture (VSR) because of a high risk of recurrent septal defect. Percutaneous closure with an Amplatzer septal occluder was also postponed for the same reason. On the fourth day of hospitalization, a sudden hemodynamic deterioration occurred, with a decrease in blood pressure to 70/50 mmHg, dyspnea, dizziness, and oliguria, considered to be signs of cardiogenic shock. Echocardiography[1] showed enlargement of apical septal dropout to 13 mm and a decrease of transseptal pressure gradient to 47 mmHg with deterioration of tricuspid regurgitation (gap in leaflet coaptation, 10 mm; decrease of the maximum RV–RA gradient to 26 mmHg). Emergency surgical closure of VSR and cut of the myocardial bridge were performed. The patient's condition continued to deteriorate despite surgery and intensive pharmacological treatment including high doses of inotropes and prolonged mechanical ventilation. On day 7 after the surgery, the patient died of refractory multiorgan failure.

In our patient, NSTEMI was probably caused by a narrowing of the left anterior descending coronary artery due to the myocardial bridge. Cases of VSRs with "normal" coronary arteries have been reported, where a normal coronary angiogram was attributed to clot lysis, or a coronary artery spasm was suspected.[4] So far, only 1 case report of VSR associated with a myocardial bridge has been published.[5] Thus, acute MI and its mechanical complications should be considered even in patients without coronary artery thrombosis.

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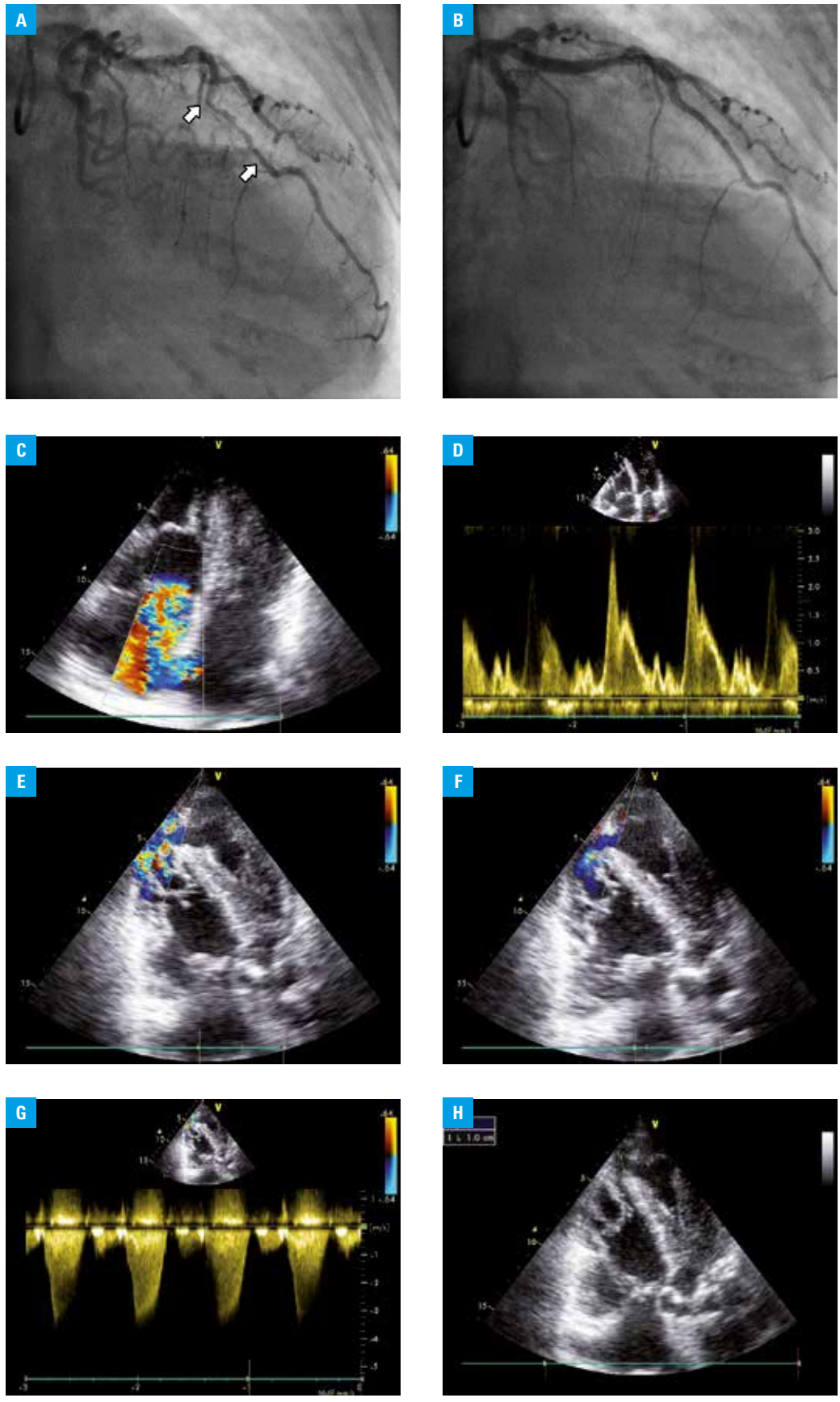

FIGURE 1 **AB** – coronary angiography in the right anterior oblique cranial view showing systolic compression of the mid-left anterior descending coronary artery; **A** – systole, arrows indicate the start and end of the myocardial bridge; **B** – diastole; **CH** – transthoracic echocardiography; **C** – color Doppler imaging of severe tricuspid regurgitation; **D** – pulsed-wave Doppler imaging of intraventricular obstruction on the level of mid-cavity segments of the left ventricle; **EF** – transseptal flow on color Doppler imaging (systole and diastole, respectively); **G** – continuous-wave Doppler imaging of the transseptal flow; **H** – 2-dimensional image of the rupture

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