Rupture of ventricular septum leading to acute heart failure due to myocardial infarction of the inferior heart wall

Pęknięcie przegrody międzykomorowej — powikłanie zawalu serca ściany dolnej znamienowane jako ostra niewydolność serca

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Ventricular septal rupture (VSR) is one of the most severe complications of myocardial infarction (MI). It occurs rarely, and the advancement in reperfusion treatment has decreased in incidence. Early surgery is the method of choice for the treatment of VSR, and patients who do not undergo surgery have 90% mortality. We present a case of a 44-year-old male patient who was diagnosed with VSR one month after invasive treatment of MI. The patient had MI of the inferior heart wall and was hospitalised in a Scandinavian centre of cardiology, where percutaneous balloon angioplasty of the right coronary artery was performed; the remaining coronary vessels did not have significant stenoses. The peri-procedural period was uneventful, and the patient was discharged six days after the procedure. One month later, the patient was admitted to a general hospital in a critical state, with cardiogenic shock and symptoms of insufficiency of the right heart ventricle that had aggravated over the previous days. On electrocardiogram, there was a persistent elevation of the ST segment and a negative T wave in the AVF lead, and a Q wave in leads II and III (Fig. 1). In laboratory studies, elevated concentrations of myocardial necrosis markers were observed, along with an increased concentration of N-terminal pro B-type natriuretic peptide (3869 pg/mL; the upper limit of normal of 125 pg/mL). Echocardiography revealed rupture of the intraventricular septum (IVS), approximately 23 mm in size. In the middle part of the IVS, there was right-to-left shunt (Fig. 2). On chest X-ray, there was significant bilateral pleural effusion (Fig. 3). We instituted pharmacological treatment (catecholamines and diuretics) and performed thoracentesis, which resulted in an improvement of the clinical state. The patient was referred to the Department of Cardiosurgery, where the IVS defect was closed with two patches (5 × 4 mm) and tissue glue. The procedure was performed with extracorporeal circulation (Deville’s method, Fig. 4). On follow-up echocardiography, there was an impairment in the contractile function of the left ventricle, ejection fraction of 38%, akinesis of the inferoposterior heart wall, residual left-to-right shunt in the apex area (1.0–1.3 cm), and a residual shunt in the basal area. There were no significant arrhythmias and atrioventricular conduction disorders on 24-h electrocardiography. The peri-procedural period was uneventful. Six months later, the patient remains stable, without symptoms of heart failure.

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