

Ventricular septal rupture after blunt chest trauma

Pęknięcie przegrody międzykomorowej po tępych urazie klatki piersiowej

Anna M. Piekarska¹, Bartłomiej Perek², Arkadiusz Niklas¹, Andrzej Tykarski¹

¹Department of Hypertension, Angiology and Internal Medicine, Poznan University of Medical Sciences, Poznan, Poland

²Department of Cardiac Surgery, Poznan University of Medical Sciences, Poznan, Poland

Ventricular septal rupture (VSR) is a rare complication of blunt chest trauma. The commonest effect of this non-penetrating injury of the thorax is pulmonary and myocardial contusion. We present the case of a 30-year-old man with post-traumatic ventricular septal rupture. Two days earlier he fell from a roof (about 4 m high) and was admitted to the emergency department of the regional hospital. On admission, he was fully conscious and by virtue of his left forearm being broken he was referred to the surgical department, where his left radius bone was attended to. Several hours later, the patient's general clinical condition started gradually worsening. Symptoms of progressive pulmonary failure were observed, and eventually he was intubated and mechanically ventilated. Moreover, due to cardiac status deterioration, an echocardiographic examination was performed urgently and muscular VSR with left-to-right flow was visualised (Fig. 1). After this diagnosis was established, the patient was referred emergently to our Cardiosurgical Department.

On admission he was mechanically ventilated and deeply unconscious with no reaction to pain. At that time no sedation was employed. Moreover, he required marked doses of positive inotropic agents (dobutamine and dopamine). By virtue of serious neurological status, he was disqualified from salvage cardiac surgical procedure. In transthoracic echocardiographic examination, a rupture in the middle, muscular part of the ventricular septum, about 10–12 mm in diameter, 40 mm from the mitral annulus and 30 mm from the apex, with accompanying left-right flow velocity of approximately 2.0 m/s was visualised. In the meantime, computed tomography (CT) angiography was performed, which revealed normal thoracic aorta. Abdomen ultrasonography did not reveal serious abnormalities. Hypotonia (RR 70/40 mm Hg) was observed from the beginning and it was accompanied by high fever (up to 40°C). Several laboratory cardiac necrosis marker tests, such as cardiac troponin I, and creatinine kinase MB isoenzyme confirmed cardiac injury. Soon after admission, an intra-aortic balloon pump (IABP) was inserted through the puncture of the right femoral artery and continuous veno-venous haemofiltration (CVVH) was initiated due to renal failure with oliguria. In spite of the aggressive treatment with IABP and CVVH, the patient developed multi-organ failure including liver (alanine aminotransferase activity increased up to 8,328 IU/L and asparagine aminotransferase to 2,406 IU/L). A head CT (Fig. 2) revealed diffuse axonal injury, extensive post-traumatic changes in the form of significant swelling of the brain, overall tightening of the subarachnoid space in the third and fourth ventricles and also in the lateral ventricles. Disseminated air zones were found in the subcortical white matter. Small haemorrhagic changes were revealed in the right cerebellar hemisphere and single small (4 mm) changes were present in the frontal lobes. On the basis of the patient's general status, and the results of imaging studies, the patient was disqualified from invasive treatment of VSR. On the seventh day of hospitalisation in the Department, the patient's general condition rapidly worsened, hypotension non-responsive to increased doses of catecholamines appeared, and the patient developed coagulopathy. One day later, the patient developed progressive bradycardia and hypotension. Eventually, in spite of pharmacological resuscitation, he did not respond and he died.

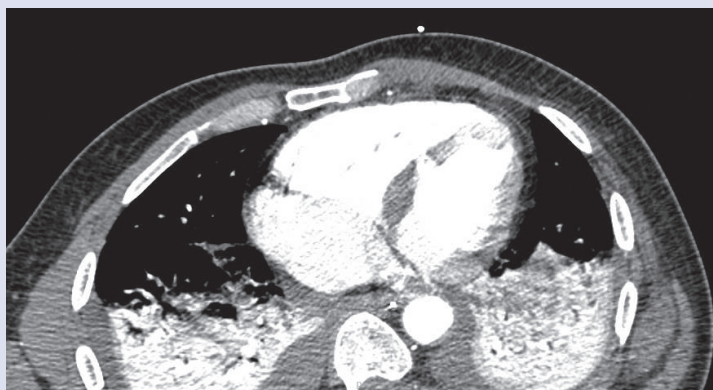


Figure 1. Computed tomography of heart, rupture in the middle part of ventricular septum

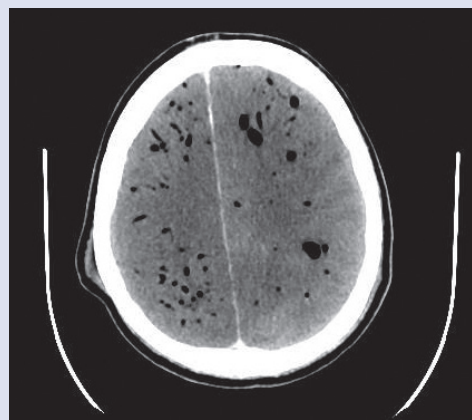


Figure 2. Disseminated air zones in the brain

Address for correspondence:

Anna Marta Piekarska, MD, Department of Hypertension, Angiology and Internal Medicine, Poznań University of Medical Sciences, ul. Długa 1/2, 61-848 Poznań, Poland, e-mail: anna.m.piekarska@gmail.com

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