

Extensive myocardial infarction in a 20-year-old associated with diabetic ketoacidosis

Rozległy zawał serca u 20-letniej chorej powiązany z cukrzycową kwasicią ketonową

Martin Janík¹, Jozef Krajčovič¹, Ľubomír Straka¹, Petr Hejna², František Novomeský¹

¹Department of Forensic Medicine and Medicolegal Expertise, Jessenius Faculty of Medicine, Comenius University, Martin, Slovak Republic

²Department of Forensic Medicine, Faculty of Medicine, Charles University and University Hospital, Hradec Kralove, Czech Republic

Autopsy serves an important role in diagnosis and as a teaching tool for clinicians. We report a case of a 20-year-old female with long-lasting type 1 diabetes mellitus (DM1), coeliac disease, and episodes of diabetic ketoacidosis (DKA), who presented to the emergency room with severe DKA (glycaemia 53.8 mmol/L, base excess 27.4 mmol/L, arterial pH 6.93), abdominal pain, and frank coma. She had no history of chest pain, previous ischaemic events, or use oral contraceptives. Admission electrocardiogram (ECG) showed absent P waves, and the early repolarisation pattern in all leads possibly related to severe acidosis. She was treated with fluid and electrolyte replacement, insulin administration, and correction of acidosis. Several hours later, she was found to have elevated levels of cardiac troponins. Her ECG changes were suggestive of acute anterior ST-segment elevation myocardial infarction (MI). Echocardiogram showed extensive akinesis involving all apical segments and the anterior wall, with severely impaired left ventricular function. Emergent coronary angiography confirmed total thrombotic occlusion of the left anterior descending artery (LAD) territory (Fig. 1A, B). Thrombosuction and dilatation failed to restore normal anterograde coronary perfusion (Fig. 1C). Further clinical course was complicated by progressive circulatory failure, culminating with the patient's death on the fourteenth day. The autopsy confirmed semi-circumferential transmural infarct of the left ventricle (Fig. 1D). Cross-sections of the LAD revealed focal fibrolipid thickening with occlusive antemortem thrombus (Fig. 1E). Histology demonstrated microcalcifications of the LAD (Fig. 1F), and subacute MI (at approximately two weeks) with neovascularisation (Fig. 1G), myocytolysis, and early collagenous replacement (Fig. 1H). Additional findings included pulmonary artery thrombosis with foci of pulmonary infarctions, left atrial auricle thrombosis, and MI-associated fibrinous pericarditis. DM1-related MI at a young age is exceedingly rare. Nonetheless, DM1 is likely to result in higher incidence of MI, because of hypercoagulability and increased plaque burden that are dictated by a chronic hyperglycaemic state. The former relates to reduced anticoagulant factors, increased platelet activation, and impaired fibrinolysis; the latter is driven by oxidative stress, endothelial dysfunction, alterations in mineral metabolism, and increased cytokine production. This report is clinically significant on two counts. First, acute metabolic disturbances with severe hyperglycaemia in young diabetics, as in DKA, may further worsen the hypercoagulable state, placing them at appreciable risk for fatal coronary thrombosis. Second, as the clinical picture and initial electrocardiographic changes of myocardial ischaemia may have been concealed during

severe DKA, high index of suspicion for this association, coupled with urgent recognition are key features to avoid delay in life-saving therapy.

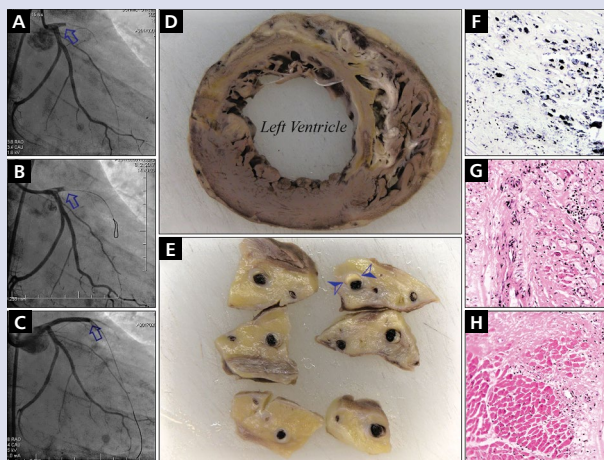


Figure 1. A, B. Angiograms of left anterior descending artery (LAD) showing total occlusion in the proximal part (blue arrows); C. Postprocedural angiogram showing insufficiently reperfused LAD (blue arrow); D. Cross-section of the heart demonstrating transmural infarction of the left ventricle; E. Cross-sections of the proximal LAD showing plaque with lipid core (blue arrowheads), the lumen is occluded by thrombus; F. Calcium deposition in the LAD (haematoxylin-eosin ×20); G. Myocardial granulation tissue and neovascularisation (haematoxylin-eosin ×20); H. Myocytolysis, and early collagenous replacement (haematoxylin-eosin ×20)

Address for correspondence:

Martin Janík, MD, PhD, Department of Forensic Medicine and Medicolegal Expertise, Jessenius Faculty of Medicine, Comenius University, Kollárova 2, 036 59 Martin, Slovak Republic, e-mail: janik.mato@gmail.com

Conflict of interest: none declared

Kardiologia Polska Copyright © Polskie Towarzystwo Kardiologiczne 2017