34. DRESSLER'S SYNDROME AFTER MYOCARDIAL INFARCTION

Grib Andrei, Abraş Marcel, Morkunaite Kristina, Lapinskas Tomas

Academic adviser: Revenco Valeriu, Ph.D., Professor, Department of Cardiology, SMPhU "Nicolae Testemiţanu", Chişinău, Republic of Moldova

Introduction: Dressler's syndrome is a secondary form of pericarditis following myocardial infarction (MI) or postsurgical injury of the pericardium. It presents with fever, pleuritic pain and pericardial effusion between 2 weeks to several months after MI, and is affecting 1-5% of patients.

Case Report: A 37-year-old male presented to the emergency room (ER) with acute chest pain, accompanied by general weakness after 18 hours from onset of symptoms. Cardiac risk factors included hypertension, smoking, dyslipidemia and a family history of coronary artery disease. The ECG was consistent with an acute inferior ST-segment elevation MI. Despite the late presentation, he was taken to the catheterization lab because of his ongoing pain and persistent ST elevation. Coronary angiography revealed proximal circumflex artery occlusion. Target lesion angioplasty was performed and a bare metal stent (INTEGRITY 3.5×15 mm) was implanted, obtaining a successful opening of the artery with a TIMI 3 flow. Following loading doses of aspirin and Clopidogrel in the ER, the treatment continued with Metoprolol, Zofenopril, Atorvastatin and Heparin. Level of troponin I was elevated to 166.23 µg/l. Transthoracic echocardiogram (EcoCG) showed akinesis of the inferior, posterior and lateral walls of the left ventricle (LV) from the base to the apex with a reduced systolic function (EF 35%) and no pericardial effusion. His impatient stay was complicated by pyrexia, raised inflammatory markers (CRP 239.72 mg/l) and a negative chest X-ray dynamic with worsening venous stasis to grade 2, focal infiltration in the right 7th and 8th segments and minimal amount of pleural fluid. A right lung lower and middle lobes pneumonia was diagnosed and antibacterial therapy (Ampicillin/Sulbactam 1.5 g × 4 times/day) was added. Further laboratory findings revealed mild anemia, leukocytosis, raised inflammatory and liver markers. Sputum culture was negative. Two weeks after admission, despite antibiotic therapy, the patient continued to have fever, dyspnea, dry cough and pleuritic pain. EcoCG examination showed progressive pericardial effusion with no visible mechanical defects. Subsequent cardiac magnetic resonance revealed pericardial effusion of heterogeneous fluid: LV lateral wall 28 mm, inferior wall 22 mm, anterior wall 27 mm, right ventricle lateral wall 20 mm, left atrium 30 mm, right atrium 24 mm, with its systolic collapse. Pericardiocentesis for decompression was not performed because of no clinical signs of hemodynamic compromise. In the absence of sufficient data for a "hidden" cardiac rupture and the presence of rich pericardial fluid accumulation, a diagnosis of Dressler's syndrome was considered, and a glucocorticoid therapy with Prednisolone 1 mg/kg/day was initiated. Because of persistent inflammatory indicators, antibacterial treatment was changed to Piperacillin/Tazobactam 4.5 g \times 4 times/day. During the next few days on treatment patients' fever subsided, cough and dyspnea were reduced. Heart rate normalized to 74 beats/min and blood pressure was 120/70 mmHg. Auscultation revealed vesicular breathing in the lungs, without rales. A prednisolone dose reduction to 5 mg/week was recommended to be continued after discharge. One month later the patient presented to the hospital for reevaluation and he had no recurrence of pericardial symptoms and a repeated EcoCG showed no pericardial effusion.

Keywords: Dressler syndrome, myocardial infarction

35. SCORAD INDEX EVOLUTION IN CHILDREN WITH ATOPIC DERMATITIS Buraga Natalia

Academic adviser. Stasii Ecaterina, M.D., Ph.D., Associate Professor, Pediatric Department, State Medical and Pharmaceutical University "Nicolae Testemițanu", Chișinau, Republic of Moldova

Introduction: Atopic dermatitis (AD) is a chronic inflammatory disease of the skin conditioned mostly by IgE allergic reactions genetic associated with atopy. It was proven that AD clinic evolution has some particularities related to the age, the severity of process and disease's duration. The severity of the disease is appreciated by SCORAD index assessment (scoring atopic dermatitis).