

236. MYOCARDIAL INFARCTION WITH NONOBSTRUCTIVE CORONARY ARTERIES: A PUZZLED STORY

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Introduction. Myocardial infarction with non-obstructive coronary arteries (MINOCA) in contemporary practice involves a large amount of causes and the same number of therapies. MINOCA should be considered as a working diagnosis in order to determine physicians find the specific causes of its appearance, clarify the underlying individual mechanisms and achieve patient-specific treatments, although the mechanism of the myocardial damage in these patients remains unclear.

Aim of the study. This review aims to better understanding the clinical diagnosis of MINOCA

Materials and methods. The article is based on international publication data and on-line materials.

Results. Myocardial infarction without obstructive coronary artery disease (MINOCA) is a syndrome defined by the presence of the universal acute myocardial infarction (AMI) criteria among with normal or near normal coronary arteries and no clinically overt specific cause for the acute presentation. As different clinical studies have reported a prevalence with a range between 4 - 25% of AMI cases, physicians have been regularly confronting with many questions on its management. The demographic and clinical characteristics of MINOCA are different from patients with AMI, being more common in younger and in women, having a lower prevalence of traditional cardiovascular disease risk factors. Studies made pointed a different profile with previous history of depression, emotional stress, inflammatory conditions and malignancy. The diagnosis of MINOCA should exclude first other causes for elevated troponin, overlooked obstructive coronary disease, nonischemic causes for myocardial injury, including Takotsubo syndrome. There are disparate aetiologies causing MINOCA, including: coronary disorders (coronary plaque disruption, coronary dissection, coronary spasm, coronary thrombus/embolus, microvascular dysfunction); myocardial disorders; non-cardiac disorders (e.g. pulmonary embolism). Failure to identify the underlying cause may result in inappropriate therapy in these patients. As the plaque disruption, spontaneous coronary artery dissection are common in MINOCA, it is recommended to use optical coherence tomography or intravascular ultrasound imaging to confirm it. Coronary vasospasm and microvascular dysfunction are other frequent findings in MINOCA patients undergoing provocative testing with acetylcholine – the gold standard technique. Multiple diagnostic pathways have been proposed to evaluate patients with MINOCA, considering as priority cardiac magnetic resonance imaging. Rational treatment follows from etiologic diagnosis, since same therapy will not be appropriate for all MINOCA patients. The outcome of MINOCA depends on the underlying cause, but its overall prognosis is serious with a 1 year mortality about 3,5%.

Conclusions. MINOCA is a distinct clinical diagnosis with different pathophysiological causes. It is essential that healthcare professionals become familiar with it, use proper

diagnostic criteria, additional investigation techniques and determine target therapies for each patient, in order to improve their clinical outcome.

Key words: MINOCA, coronary disorders, cardiovascular disease

237. THYROTOXIC CARDIOMYOPATHY: A CASE REPORT

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Background. Heart failure (HF) is the final common pathway of many cardiovascular diseases. It imposes significant socio-economic and health care burden to both patients and healthcare systems. Although the most common cause of HF is ischemic heart diseases, other less common causes such as hyperthyroidism (thyrotoxicosis), severe anemia, arrhythmia should also be considered during diagnosis to improve overall clinical management of HF.

Case report. The 42-year-old man was admitted to cardiology department with mixed (inspiratory and expiratory) dyspnea at moderate effort, palpitations, fatigue, the loss in weight of about 15 kg during 9-10 months. Anamnesis: general condition worsened the last 2 months when appeared generalized edema and mixed dyspnea. During this time did not address to doctor, any treatment has not received. Physical examination revealed swelling in the legs, ankles, ascites, an irregular pulse, at a rate of 130 beats/min, BP- 110/70mmHg. On ECG - atrial fibrillation with rate - 120-57 b/min, electric axis of heart is normal. Signs of left ventricular hypertrophy. The chest X-ray -pulmonary congestion, bilateral pleural effusion. The abdominal X-ray – fluid levels with air on the left. On TTE- thickening of the walls of the aorta and valve apparatus. Dilatation of all heart chambers, significant dilatation of the right atrium and right ventricle, and moderate dilatation of the left atrium and the left ventricle. Contractile function of the left ventricular myocardium is moderately reduced. Ejection fraction = 42%. The second degree mitral regurgitation and third-fourth -degree tricuspid regurgitation. Moderate pulmonary arterial hypertension (PASP= 52mmHg). Sheets of the pericardium are thickened. Fluid in the pleural cavity up to 11 millimeters in the region of the right atrium. Bilateral pleurisy - inhomogeneous fluid with floating elements on the left - about 1,000 milliliters, to the right - about 800 milliliters. Cytological analysis of fluid from pleural cavity pointed to the inflammatory etiology of the effusion. On the ultrasound examination of the thyroid gland – fourth –degree hyperplasia, multiple diffuse changes. On the ultrasound examination of abdominal cavity - ascites, bilateral pleuritic, diffuse changes in the parenchyma of the liver. The glycemic profile -7-00: 4.7 mmol/l, 13-00: 6.3 mmol/l, 17-00: 10.6 mmol/l, glycated hemoglobin - 5,6%. Analysis of thyroid hormones- free Triiodothyronine – 17,22 Pmol/l, free Thyroxine – 79,52 Pmol/l. TSH – < 0, 05 uIU/ml; anti TPO- 144 IU/ml. Tumor marker CA 19-9 - <3.0 U/ml. During hospitalization was consulted by endocrinologist, surgeon. After 11 days of complex treatment with diuretics, anticoagulants, beta-adrenoblockers, antithyroid drugs, cardiac glycosides, corticosteroids, histamine-2-receptor blockers - the general condition improved: dyspnea and general swelling disappeared, general weakness was reduced.