

12. HYPERTROPHIC CARDIOMYOPATHY WITH INVOLVING OF THE RIGHT VENTRICLE

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Introduction. Hypertrophic cardiomyopathy (HCM) is defined by the presence of increased left ventricular (LV) wall thickness that isn't solely explained by abnormal loading conditions. Most previous studies include the LV for definition of HCM, neglecting the right ventricle (RV), but recently the RV involvement in HCM was reported. Cardiac magnetic resonance (CMR) is useful in characterizing the phenotypes of RV and LV in HCM. RV involvement is associated with increased risk of arrhythmias, dyspnea, pulmonary thromboembolism, heart failure and sudden death.

Case presentation. The 32y.o. man was admitted at Institute of Cardiology in December, 2021 with complaints: intermittent chest pain on physical endeavor, low tolerance to exertion. Family history revealed the father's death in middle age of unknown cause. In childhood was suspected of rheumatic fever followed by tonsillectomy at the age 18y.o. without improvement of symptoms due to which wasn't admitted to compulsory military service. In March 2020 suffered an episode of fainting. ECG performed at that time showed sinus rhythm with repolarization abnormalities, manifested by T-wave inversion in precordial leads. Patient was referred to the emergency department with suspicion of acute coronary syndrome. Subsequently diagnosis was not confirmed either clinically or laboratory. Physical examination at admission revealed regular rhythm without extracardiac sounds, HR-75b/min, BP-110/75mmHg,SpO2-98%. On ECG deep negative T wave in V1-V4. NT-proBNP-253ng/ml, ESR-14mm/h. EcoCG: normalsized heart chambers, normal systolic function (EF 62%), IVS medio-apical thickness-20mm, apical segment of RV-17mm, obstructed outflow tract of RV. Holter ECG: pronounce respiratory arrhythmia, negative T wave in V1-V5. CMR: Accentuated LV trabeculae type `non-compact spectrum`. Fibrotic intramural lesion in hypertrophic LV, late gadolinium enhancement. Hypertrophic RV myocardium. Two septal interventricular defects. Non-obstructive HCM, asymmetric form. Regardless of the patient's complaints and instrumental findings the diagnosis of Non-obstructive hypertrophic cardiomyopathy was confirmed. The treatment with b-blockers was initiated.

Discussion. This case represents clinical interest because of detection of RV myocardial hypertrophy, less described in the literature. It's certain that the patient suffers from a genetically determined cardiomyopathy, however a more accurate diagnosis requires a comprehensive genetic evaluation, including storage disease which have similar features. Unfortunately, HCM is delayed diagnosis as in the patient's case. Thus making a differential diagnosis for chest pain and syncope in the young population is an important heart assessment by CMR. This patient is supervised by a cardiologist and will continue treatment with b-blockers. The thromboembolic risk assessment and the decision of anticoagulants are required.

Conclusion. The case emphasized that assessment of young patients with chest pain and fainting is multidimensional and can hide life-threatening conditions.