Abstract: P581

Hypertrophic cardiomyopathy, myocardial bridging and tako-tsubo syndrome: on the trail of the culprit.

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Topic(s):
Cardiac Magnetic Resonance: Myocardium

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Introduction
In patients with hypertrophic cardiomyopathy (HCM) the compression of sub-endocardial vessels could cause angina. In the last years it was described an association between left ventricle outflow tract (LVOT) obstruction and Tako – Tsubo syndrome (TTS). Moreover, in patients with HCM can be common the presence of a myocardial bridging contributing to myocardial ischemia. We described a case in which coexist TTS, HCM/LVOT obstruction and myocardial bridge to understand their interaction in the development of a myocardial infarction.

Report
A 41-year-old man presented at the EMS for a growing chest pain, risen after an intense physical exercise. He explained to medical staff to be affected by HCM with LVOT obstruction. ECG showed QS complexes in leads V1 – V3 with ST-segment elevation and the blood biochemical tests an increase in biomarkers of cardiac necrosis. It was urgently performed a coronary angiography that showed uninjured coronary arteries and an intramiocardic course of the proximal tract of left anterior descending coronary artery with a compression of the vessel during the systole. The ventriculography revealed a typical "apical ballooning". After the procedure the echocardiography documented a hypertrophic left ventricle (LV) with a significant intra-ventricular gradient across the outflow tract (64 mmHg), akinesia of apical tract of LV with reduced ejection fraction (30%) and a systolic anterior movement (SAM) of the mitral valve anterior leaflet causing a moderate mitral regurgitation. The medical therapy was optimized with an increase of beta – blocker doses to reduce the coronary spasm. After 3 days an echocardiographic control described an improvement of ejection fraction (45%), ipokinesia of left ventricular apex and a reduction of LVOT obstruction (peak gradient 36 mmHg) with a mild mitral regurgitation. The patient was discharged with diagnosis of Myocardial Infarction With Nonobstructive Coronary Arteries (MINOCA) and indication to undergo cardiac magnetic resonance imaging (MRI) after one month. It portrayed: left ventricular hypertrophy with normal systolic function; diffused ipokinesia and enhancement of the signal after gadolinium injection of the apical segment compatible with an edematous reaction but in absence of myocardial fibrosis or apical ballooning (Figure) and a flussimetric acceleration in subvalvular aortic seat to relate likely to SAM of the anterior leaflet. The patient did not complain of any symptoms and reported to have resumed his daily activity.

Conclusions
Association between TTS, HCM/LVOT myocardial bridge and its clinical implication remains uncertain. This case suggests their compresence increases the risk to develop MINOCA. We can speculate that in this patient physical exercise likely caused release of catecholamines and coronary spasm of myocardial bridge and both conditions, in presence of HCM and LVOT obstruction, have contribute to develop apical ballooning and myocardial damage.
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