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Case Report
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Vasospastic Coronary Artery Disease or Microvascular Angina or Coexisting Syndromes – Extensive Perfusion Defect Resolution after Treatment Modification

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Abstract

Less frequent manifestation of Coronary Artery Disease (CAD) that has to be taken into consideration in such cases is vasospastic angina. Coronary spasm has been observed on angiograms, but exceptionally in left main artery. Vasospastic pathomechanism was repeatedly proven by intracoronary spasm inducement with ergonovine or acetylcholine injection. Although focal (single or multifocal) coronary artery occlusion occur mostly at rest and is related with ST elevation (variant or Prinzmetal angina), there are scarce reports of exertional related vasospastic angina leading to ST depression.

Case Presentation

The 43-year old Caucasian male was admitted to the Emergency Department due to one hour lasting chest pain. The routine treadmill stress test performed prior to the admission to hospital revealed significant ST segment depression in leads II, III, aVF, V3-V6 after 3, 5 minutes of exercise, without clinical symptoms characteristic for ischemia. The patient had a history of several episodes of chest pain on exertion in the last several months before the stress test; he was not diagnosed and treated for any chronic disease. Smoking was the only known cardiovascular risk factor. The exercise tolerance was sustainably optimal. The same day the ECG positive stress test was performed; patient was hospitalized in the cardiology department. The early coronary angiography revealed two proximal, borderline stenoses in the Left Main Coronary Artery (LMCA) and in Right Coronary Artery (RCA). After the Heart-Team consultation repeated evaluation of coronary arteries was advocated, with additional Intravascular Ultrasound (IVUS). The latter angiography showed no narrowing's in the epicardial vessels. Subsequent IVUS of LMCA, proximal Left Anterior Descending (LAD) and Circumflex (LCX) arteries showed absence of stenoses, with no evidence of atherosclerosis (Figure 1, 2). Three weeks after coronary angiography the nuclear stress test was performed according to Bruce protocol. Exercise test was terminated due to increasing chest pain in the sixth minute on exertion (achieved heart rate of 130 per min – 70% of maximal expected heart rate, workload of 4.6 METs). There was significant ST segment depression in leads V5 and V6. Myocardial Perfusion Imaging (MPI) performed one hour later showed extensive and severe perfusion deterioration within the apex, anterior and inferior walls of the left ventricle. The rest MPI revealed substantial perfusion improvement of radiotracer uptake in the regions with decreased accumulation. Furthermore, estimated region of improved perfusion comprised approximately 40% of the left ventricle's myocardium. Considering all of these results, the vasospastic mechanism has been suggested, as the reason of described coronary arterial narrowing's observed in first angiography and not confirmed in further diagnostics. Therefore the treatment has been modified (beta blocker was withdrawn; calcium channel blocker treatment was initiated). Another MPI was suggested by interventional cardiologists a month after treatment modification. Two sublingual doses of 0.4 mg Nitroglycerine (NTG) each were administered within one hour before the next treadmill stress test. Two day stress/rest MPI with ^{99m}Tc-sestamibi was performed, showing only slight reversible region of perfusion abnormalities in the basal segments of the lateral wall. The enormous discrepancy was observed between two subsequent images of the MPI (Figure 3). No coronary intervention was performed in this subject between these two MPI examinations. Following the treatment modification patient has been released from exertion related symptoms.

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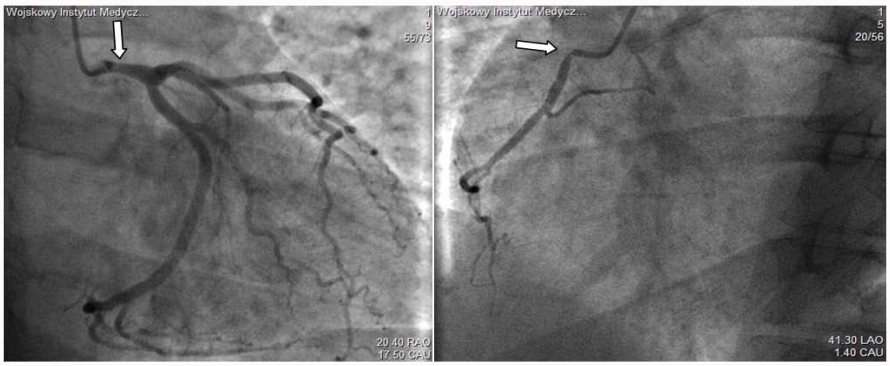

Figure 1: Angiograms of the LCA (left image) and RCA (right image). Arrows indicate LMCA and RCA borderline stenoses, possibly caused by catheter irritation.

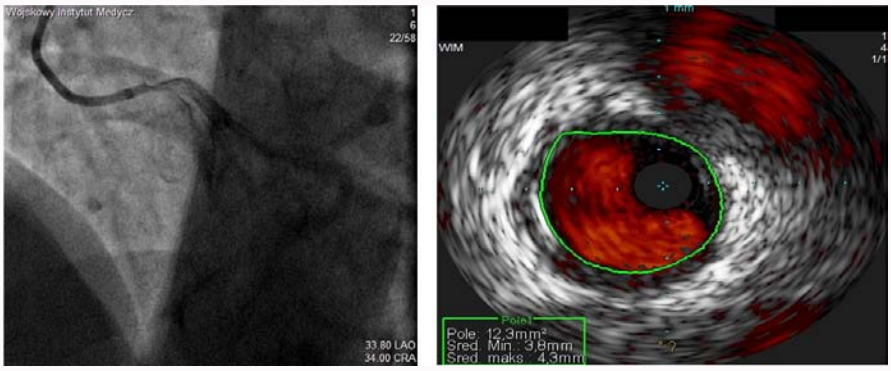

Figure 2: The following day LMCA angiography and IVUS revealed absence of stenosis.

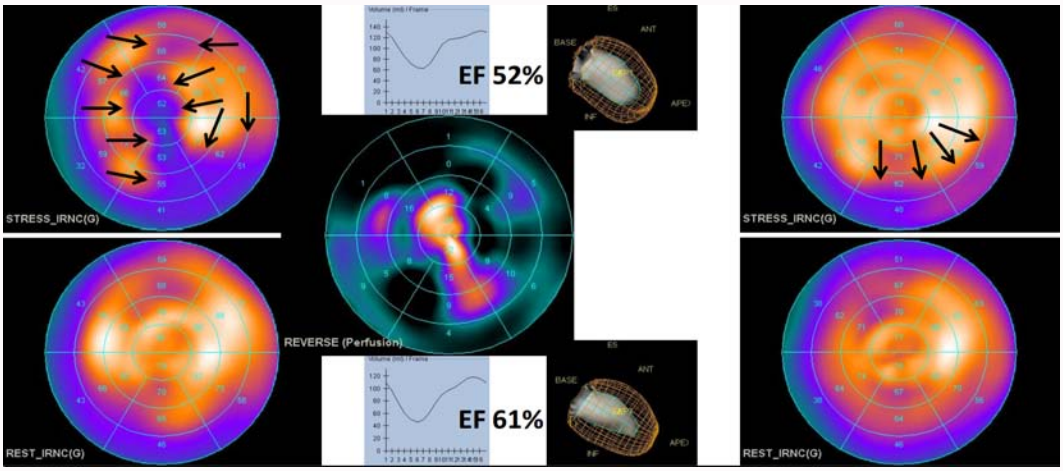

Figure 3: Polar maps of the MPI before (left polar maps) and after (right polar maps) modification of the treatment (beta blocker substituted by calcium channel blocker, nitroglycerine before the latter stress MPI). In the center reversibility polar map of first MPI and left ventricular Ejection Fraction (EF) of the stress (upper) and rest (lower) imaging.

Discussion

Occasional chest pain, whether typical anginal or accidental precordial, substernal pain/discomfort, bring about some difficulties in diagnostic process, especially in patients with low and intermediate probability of coronary artery disease. Less frequent manifestation of Coronary Artery Disease (CAD) that has to be taken into consideration in such cases is vasospastic angina. Coronary spasm has been observed on angiograms, but exceptionally in left main artery. Vasospastic pathomechanism was repeatedly proven by intracoronary spasm inducement with ergonovine or acetylcholine injection. Although focal (single or multifocal) coronary artery occlusion occur mostly at rest and is related with ST elevation (variant or Prinzmetal angina), there are scarce reports of exertional related vasospastic angina leading to ST depression [1,2]. This phenomenon could be induced by diffused spasm in distal coronaries. According to the 2019 ESC guidelines for the diagnosis and management of chronic coronary syndromes the presence of anginal symptoms and abnormal non-invasive tests in patients with non-obstructed epicardial vessels should lead to the suspicion of a non-obstructive cause of ischemia but because diagnostic pathways to investigate microcirculatory or

vasospastic coronary disorders are often not implemented, a final diagnosis supported by objective evidence is seldom reached. The possibility of a microcirculatory dysfunction should be considered in patients with angina, abnormal non-invasive functional tests, and coronary vessels that are either normal or have mild stenosis considered as functionally non-significant on invasive or non-invasive coronary angiography. Additionally development of ECG changes and angina in response to acetylcholine testing but without severe epicardial vasoconstriction is suggestive of microvascular spasm [3]. AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR Guideline for the evaluation and diagnosis of chest pain emphasizes, that in the presence of non-obstructive coronary artery disease reduced myocardial blood flow reserve may indicate coronary microvascular dysfunction, especially among women [4-7]. In this particular case the vasospasm appeared in arteries at the catheter tip, which can indicate the mechanical irritation coexisting with hypersensitivity of vessels as potential explanation. Nonetheless it does not expound the extended and severe myocardial perfusion abnormalities in stress MPI with pronounced improvement on the rest images. The ST depression in stress MPI with reversible perfusion defects would rather suggest unoccluded, spastic variant. The repeated stress/rest MPI with mild perfusion abnormalities after treatment was changed (continuous calcium channel blocker treatment, sublingual NTG before stress test) seems to confirm the vasospastic mechanism but do not exclude microvascular dysfunction. Studies with Acetylcholine (ACH) testing revealed, that in stable CAD with normal or near normal arteries in coronary angiography, intracoronary ACH injection provoked vasospasm in 62% (the ACOVA study). In 55% of them the ACH test results (ECG changes, symptoms) were interpreted as microvascular spasm. The abnormal ACH tests are directly related with endothelial dysfunction. In response to ACH uptake by endothelial receptor, nitric oxide and other compounds are released, stimulating vasodilation. Instead, when endothelial cells remain dysfunctional, ACH can induce coronary vasospasm *via* receptors within smooth muscles. Vasospastic variant of angina pectoris, if not demonstrated by ST segment elevation, as well as microvascular angina still cause clinical difficulties in diagnostics and management of our patients. The studies show, that in many cases of CAD there is more than one underlying pathomechanism responsible for anginal manifestation. Yet advanced diagnostic instruments may lead us to appropriate conclusion so we can introduce accurate treatment and relieve patients from inconvenient symptoms.

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