A case of lactic acidosis and stress hyperglycemia in a non-diabetic patient with vasospastic angina

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Introduction: Coronary artery spasm can cause transient ischemia that can lead to acute myocardial infarction, arrhythmias and sudden cardiac death[1]. Due to ischemia and stressful stimuli in patients with myocardial infarction, lactate and glucose levels can be elevated and possibly dependent on each other[2].

Case report: 63-year-old woman presented to the Emergency Department with dull chest pain at rest, palpitations and vomiting, preceded by a headache. Her past medical history was significant for myocardial infarction with non-obstructive coronary arteries (MINOCA) accompanied by transient hyperlactatemia and stress hyperglycemia after a stressful event 3 years prior. At that time, coronary angiogram showed normal coronary arteries without significant stenosis and echocardiography showed no significant pathology. Stress cardiac magnetic resonance imaging was also performed and the results were negative for inducible ischemia. During the current admission, the patient was in a fair general state, pale and tachypnoic upon examination, with the same symptoms as in the previous hospitalization. 12-lead electrocardiography (ECG) showed inferolateral ST-segment depression and premature ventricular contractions. Troponin T level measured 210 ng/L. Acid-base status revealed hyperglycemia, lactic acidosis, hypokalemia and hypocalcemia. Standard treatment led to correction in acid-base status. Echocardiography again showed preserved ejection fraction with no wall motion abnormalities and no significant valvular disease. The patient was referred to nephrologist and other causes of lactic acidosis were ruled out. Invasive assessment of coronary microcirculation showed normal findings and a provocative test with acetylcholine was then performed. It revealed diffuse coronary artery vasospasm accompanied by chest pain, inferior and anteroseptal ST depression on ECG with resolution after nitrate application. A diagnosis of vasospastic angina (VSA) was made and diltiazem 60 mg two times a day was recommended.

Conclusion: VSA is a known cause of MINOCA[3]. This case discusses the possibility of VSA being triggered by systematic metabolic disorders, like in our case, lactic acidosis, in opposition to the possibility of VSA being the underlying cause of lactic acidosis and stress hyperglycemia.

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LITERATURE

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