

Prinzmetal's angina of difficult control

Academic authors: **Jéssica Silva Nicolau, Henrique Ryu Yamanaka Nakano**

Academic advisor: **Fabio Grunspun, Eduardo Gomes Lima, Carlos Vicente Serrano Jr**

Introduction: Coronary vasospastic angina (CVsA) is an uncommon and severe form of non-atherosclerotic angina caused by coronary artery spasm, resulting in reduction of blood flow, transient ST-segment elevation and myocardial injury. The vasospastic angina can occur simultaneously to the atherosclerotic angina, or in an isolated form, and can be associated or not to physical activities. Endothelial dysfunction is an important mechanism involved with the disease, mediating hypercontractility of smooth muscle of vessels, associated with an imbalance of the vagal and sympathetic tonus. In this Case Report we will exhibit an instance of a 45 years old woman who presented three episodes of cardiorespiratory arrest (CRA) and ventricular fibrillation (VF) associated with CVsA and obstructive sleep apnea (OSA).

Objectives: Show the importance of coronary vasospastic angina in daily clinical practice and its association with obstructive sleep apnea (OSA).

Case Report (c.4.): L.M.M, female, 45 years old, presented in August 2011 precordial pain and three episodes of CRA-VF. There were no changes in the electrocardiogram. The patient underwent a catheterization in the period, which showed a lesion of 40% in the right middle coronary artery (CD) and a hypertrophy of left ventricle (LV). Though, the cause of CRA wasn't from atherosclerosis disease, but from severe vasospasms, reversed by monocordil.

The patient's past medical history showed systemic arterial hypertension, diabetes mellitus, dyslipidemia, obesity, bronchospasms with the use of beta-blockers and obstructive sleep apnea syndrome (OSAS). She also had a 10 pack-year smoking history. Faced with this dramatic case, it's possible to say the patient has brought us an atypical case of CVsA, once it's rare to see CRA as a complication of an intermittent spasm, and the patient presented three episodes. There is no mechanism that can fully explain the pathophysiology of CvsA, but it's known that vascular smooth muscle hyperreactivity plays an important role of it. This is explained from the presence of leukotrienes and vasoconstricting substances and from deficiency in endothelial nitric oxide activity. All of these substances also play important role in the others diseases, as in bronchial asthma, due to the hyperreactivity of the airways. The patient also has an obstructive sleep apnea syndrome, another condition characterized by vasoconstriction, but in the pulmonary circulation. The suggested pathways that would justify both pathogens are autonomic dysregulation, decreased nitric oxide bioavailability, decreased endothelial repair, oxidative stress, and inflammation. Thus, L.M.M may present a systemic manifestation of vascular smooth muscle hyperreactivity and endothelial dysfunction, which can explain the association between CVsA and OSAS.

Keywords: Coronary vasospastic angina; Obstructive sleep apnea.