

CORONARY SPASM, Does It Exist On Healthy Coronary Artery?

S.Es-sebbani, Pr F.Boccaro and Pr A.Cohen

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ABSTRACT

The incidence of coronary spasm appears to be decreasing, probably due to the widespread use of bradycardia calcium channel blockers, statin, and reducing smoking also. Many studies have shown the pathological association of atheroma plaque and coronary spasm. The new imaging techniques: Optical coherence tomography (OCT) and Intravascular ultrasound (IVUS), allows accurate analysis of arterial tunnels. In this article, we report a case of a patient who presented with refractory spastic angina, which was complicated by acute coronary syndrome many months later.

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Introduction

Vasospastic angina (VSA), called Prinzmetal angina, has been reported for the first time more than 50 years ago, but remains poorly understood. VSA is a clinical entity associated with hyper-reactivity of large coronary arteries to vasoconstrictor stimuli.¹ The importance of diagnosing VSA relates to the major adverse events including: sudden cardiac death,² acute myocardial infarction³ and syncope.⁴ So, avoiding potential coronary artery spasm by the use of established effective therapies, allows to prevent these adverse events. Case report : A 50 years old woman, arresting smoking for 2 months, under nicotine substitutes. No history of cardiovascular disease nor other medical problems. No medical therapy, menopausal and no illicit drug use. She complains of acute thoracic chest pains, evocating an angina pectoris at rest and was admitted to the intensive coronary unit for acute coronary syndrome without ST-elevation. The Per-critical electrocardiogram shows negative T waves in the antero-septo-apical territory, Troponin was positive at 0.49 ng/ml, the trans-thoracic echocardiography showed an apical akinesia, with a normal left ventricle ejection fraction at 50 %. She underwent a coronary angiogram which did not show any coronary artery disease, nor dissection . Two days later, she made a recurrent angina, sensitive to glyceryl trinitrate, without re-ascending troponin . The per-critical ECG showed an offset of the ST to V1-V2 She underwent a 2nd coronary angiogram which suspected a plaque image of the ostial LAD responsible for a non-significant stenosis Confirmed by the OCT which showed a fresh flat thrombus with a ruptured plaque of the ostial of LAD Decision of a medical treatment by double platelet aggregation and high dose of statin with repeating the coronary angiogram after 7 days. Evolution marked by the occurrence of multiple angina, always in the morning, with ST- elevation, sensitive to the glyceryl trinitrate, without enzymatic movement, motivating the introduction of anti-spastic treatment. The patient was placed under Risordan® and Tildiem® IV, then relayed orally by Corvasal®, Isoptin® and Discotriner®. She had a 3rd coronary angiogram which showed the disappearance of the

plaque of the ostial LAD, and the OCT wasn't realized in the absence of residual lesion The evolution was marked by the occurrence of anterior myocardial infarction during peritonitis surgery. A coronary angiogram showed a LAD occlusion at the plaque seen by the OCT, an angioplasty with the placement of an active stent was performed.

Service de cardiologie-Hôpital Saint Antoine – Paris

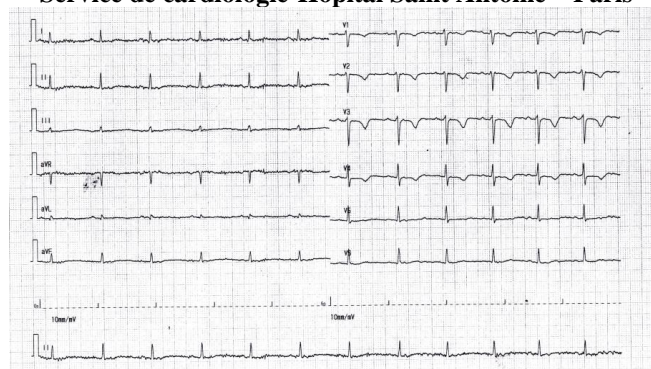


Figure 1. negative T-waves in V1-V2-V3-V4 without ST-elevation.



Figure 2. Normal Coronary angiogram.

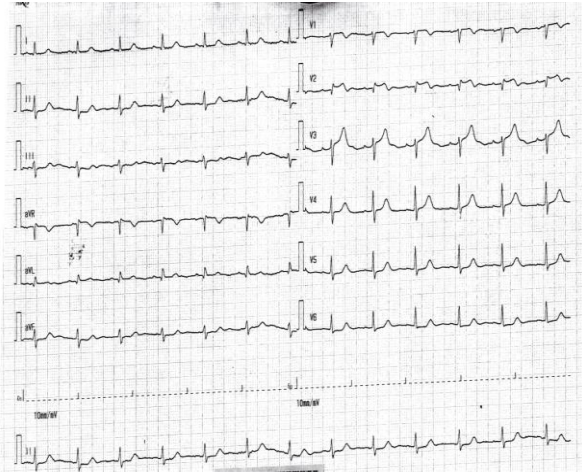


Figure 3. positif ST on V1-V2.



Figure 4. Coronary angiogram shown an ostial LAD plaque.



Figure 5. Fresh plane thrombus (posterior attenuation related to red blood cells).

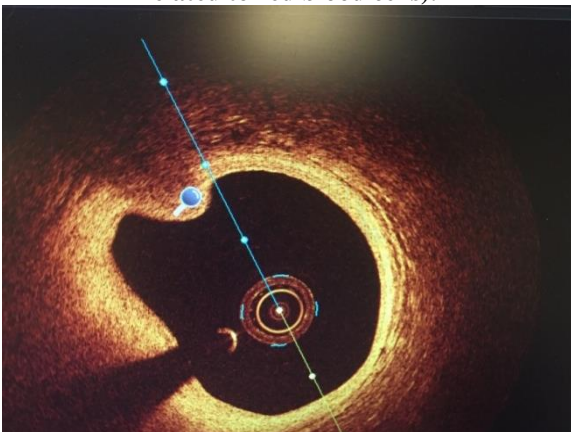


Figure 6. A ruptured plaque of the ostial LAD responsible for a non-significant eccentric stenosis.

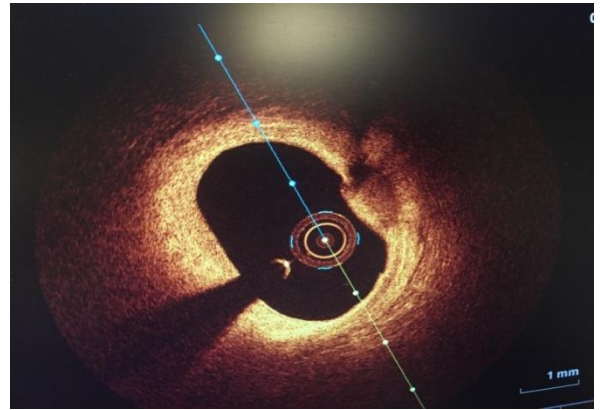


Figure 7. Calcified plaque (black zone) with image of rupture of the ostial LAD.



Figure 8. Normal Coronary angiogram.

Discussion:

Diagnosis and management of VSA is very challenging. However, an accurate diagnosis remains of paramount importance, many studies have suggested the potential value of OCT to diagnose coronary artery disease. Our case report describes a recurrent VSA related to plaque rupture diagnosed with the use of OCT. It's a new intravascular imaging modality that allows cross-sectional imaging of tissue with a resolution of 10 μ m.

Among the first publications concerning OCT, an article appeared in "Circulation" in 2003 concerning the use of OCT to quantify macrophage content in atherosclerotic plaques⁵. Indeed, the macrophage degradation of fibrous cap matrix is an important contributor to atherosclerotic plaque instability. OCT images of 26 lipid-rich atherosclerotic arterial segments obtained at autopsy were correlated with histology. Cap macrophage density was quantified morphometrically by immunoperoxidase staining with CD68 and smooth muscle actin and compared with the standard deviation of the OCT signal intensity at corresponding locations. There was a high degree of positive correlation between OCT and histological measurements of fibrous cap macrophage density ($r=0.84$, $P<0.0001$), which suggests that this technology may be well suited for identifying vulnerable plaques in patients⁵. In a recent study of patients with coronary spasm, the aim was to define the morphological features of coronary artery spasm sites using optical coherence tomography in patients with vasospastic angina (VSA). 69 patients diagnosed with VSA (confirmed with provocative spasm testing) were included, patients who had fixed stenosis (>50% of the lumen diameter) in the spasm site; comorbidities such as congestive heart failure, cardiomyopathy, or chronic kidney disease; or past history of fatal arrhythmias, prior myocardial infarction, or cardiogenic shock was excluded. OCT was performed at

the spasm site after administration of 200 mg of intracoronary nitroglycerin⁶.

Almost all VSA patients had coronary plaques at OCT (79/80 spasm sites). Fibrous cap disruption was detected at 3 sites (4%). OCT-defined erosion was observed at 21 spasm sites (26%). Thrombus with lumen irregularity was observed in 20 sites (25%), whereas 1 site had thrombus without lumen irregularity. Lumen irregularity without thrombus was observed at 49 spasm sites (61%).⁶ Therefore, OCT could have incremental value for the diagnosis of VSA showing the presence of coronary plaques, followed by lumen irregularity without thrombus erosion and thrombus were present in less than a quarter.

The aim of this study was to investigate the use of OCT for identifying macrophages in fibrous caps.

Conclusion:

OCT can confirm the absence of significant atherosclerosis or indicate the degree of subclinical atherosclerotic lesion formation. This may have relevance to modulate the aggressiveness of medical therapeutic strategies for primary prevention

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