

# Vasospastic angina

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A 51-year-old man was admitted to our department in October 2013. His risk factors for coronary artery disease included a history of heavy cigarette smoking (up to 40 cigarettes per day for 40 years; although he quit in 2006) and family history of ischaemic heart disease. He was a hepatitis C virus carrier (known since 2001).

In July 2012, he was referred for an episode of angina pain at rest followed by syncope. Clinical investigations (including electrocardiogram and myocardial biomarkers) at the time were negative and the treating clinician excluded the presence of coronary artery disease at that time.

In early January 2013, however, he complained of recurrent episodes of angina, lasting 10-15 min, occurring at rest. For such symptoms, he was admitted to a hospital, where the resting electrocardiogram (ECG), myocardial biomarkers, and stress ECG results were negative. He was then discharged home. The next day, due to recurrence of symptoms, he was readmitted to the cardiology department of the same hospital, where he underwent coronary angiography and a drug-eluting stent was implanted in the right coronary artery. One month later, he was readmitted for recurrence of symptoms and a second drug-eluting stent was implanted in the left anterior descendent artery.

In March 2013, again, he reported a recurrence of similar symptoms, this time followed by syncope. A third coronary

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angiography was performed, showing diffuse atherosclerosis and patent stents. In April 2013, he was readmitted to another hospital. Repeat coronary angiography was proposed, but the patient refused the procedure and was discharged. A few days later, he was again readmitted. The resting ECG, myocardial biomarkers, and stress ECG results were negative and, once again, he was discharged home.

In May 2013, the patient was admitted again to another hospital, where an esophagogastroduodenoscopy was performed, showing erosive gastritis. Specific therapy was then initiated. However, 1 month later, he had another episode of chest pain followed by syncope; the coronary angiography confirmed patency of the previously implanted drug-eluting stents. A few days later, he ended up in another hospital because of chest pain and syncope. A myocardial single-photon emission computerized tomography showed a limited area of inducible ischaemia of the inferior wall. Repeat coronary angiography confirmed the patency of the stents. This patient's story is not yet concluded!

A few weeks later, due to the persistence of angina at rest, a sixth coronary angiography was performed. An intravascular ultrasound study showed a 'newly developed' stenosis of the left anterior descendent artery, proximal to the previous stent, which was 'successfully' treated with an overlapping stent. A few days later, at rest, the patient presented with another identical episode of angina, which was partially responsive to sublingual nitrates. In the emergency department of our hospital, his resting ECG and serial myocardial biomarkers were negative. He was then transferred to our unit for further evaluation.

The physical and all blood exams (including cardiac biomarkers) were normal during the entire hospitalization period. The ECG showed that he was in sinus rhythm with a heart rate of 51 b.p.m. (under beta-blocker therapy). The echocardiography showed a normal regional and global left ventricular systolic function, an ejection fraction of 66%, normal diastolic function, and normal right ventricular function. The patient underwent a stress ECG, which was negative for inducible ischaemia, and Holter ECG monitoring, which excluded arrhythmias and significant STsegment deviations. The patient was discharged on dual antiplatelet therapy, a gastroprotective agent, statin

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therapy, trimetazidine 20 mg three times daily, and a nondihydropiridinic calcium antagonist. The patient has been asymptomatic since then.

#### Discussion

This is a case of angina at rest that is associated with syncope but in the absence of significant coronary atherosclerotic obstructions. The association of angina and syncope is highly suggestive of right coronary artery spasm.<sup>1-3</sup> In the present case, this hypothesis is consistent with the presence of a small ischaemic area in the territory of the right coronary artery and with a clear improvement in symptoms after substitution of previously prescribed beta-blockers with calcium channel antagonists to prevent spasm and trimetazidine to reduce the ischaemic burden.

Different mechanisms should always be taken into account (e.g. microvascular dysfunction, coronary vasospasm, and intracellular metabolic impairment) in patients with chronic ischaemic heart disease and medical therapy should be selected accordingly. This appears of great clinical relevance given the known potentiating effect of beta-adrenergic blockade on coronary vasoconstriction.<sup>4,5</sup>

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