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Vasospastic angina: A forgotten acute coronary syndrome and the usefulness of twelve-lead electrocardiogram monitoring in diagnosis

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More than fifty-five years ago, Prinzmetal et al. described vasospastic angina (VSA) as a clinical disorder with electrocardiographic manifestations due to coronary artery spasm [1,2]. VSA is typically defined by angina, which is responsive to short-acting nitrates, with spontaneous episodes at rest, frequently between mid-night and early in the morning. In order to fulfill diagnostic criteria, either transient ischemic changes in the electrocardiogram or one spontaneous or provoked coronary artery vasospasm of more than 90% on angiographic images during an angina episode are necessary [2,3]. Transient ST elevation that quickly resolves after administration of nitrates was first described by Prinzmetal et al. [1] Subsequently different studies have also demonstrated transient ST depression and U wave changes during spontaneous VSA episodes [2,3]. Holter monitoring is included in the recommendations of the European Society of Cardiology (ESC) when VSA is suspected (IIb) [4]. R-test and twelve-lead ECG monitoring could also be helpful for VSA diagnosis. Most of the time, monitoring needs to be repeated several times until an angina episode with ischemic changes on the ECG can be objectified [3].

The following case description will illustrate the relevant diagnosis of vasospastic angina. A 36 year-old man, known for high blood pressure, active smoking of one pack per day and alcohol consumption of four units per day, had previously presented with ventricular fibrillation and cardiorespiratory arrest following a slight effort four years ago. He was successfully resuscitated after electric cardioversion and standard CPR. Transthoracic echocardiography showed a normal left ventricular ejection fraction and no valvulopathy. The coronary angiography showed no lesions and cardiac MRI was normal. He was implanted with an ICD for secondary prevention and was discharged with beta-blocker medication. Diagnosis was not determined, however idiopathic ventricular fibrillation was suspected.

During the following two years, he suffered several episodes of chest pain at rest, especially during the night, on and off and lasting 10 to 15 min. These symptoms became recurrent and he decided to consult his cardiologist. He reported drinking alcohol, smoking electronic cigarettes and doing regular physical activity. Defibrillator interrogation demonstrated four ventricular fibrillation episodes with successful internal electric cardioversion. His cardiologist performed two Holter tests without any findings. Confronted with a patient who had recurrent episodes of chest pain a 24-h ambulatory twelve-lead ECG monitoring was recorded (Fig. 1) demonstrating an ST elevation in leads V1 to V3 during chest pain episodes, which occurred mostly during the night, and lasted 10 to 15 min. Consequently his cardiologist realized a transthoracic echocardiography, which showed a slight decrease in the left ventricular ejection fraction with hypokinesia in the antero-septal midsection of the left ventricle. A second coronary angiography showed a mild spasm in the midsection of the left anterior descending artery, which resolved after intracoronary nitroglycerin.

VSA was retained as the new diagnosis. The patient was treated with verapamil 120 mg once a day and was advised to stop electronic cigarettes and alcohol, thought to be possible triggers The patient was already treated with an angiotensin converting-enzyme inhibitor and a statin. One year later, the patient reported no further chest pain episodes with a treatment of verapamil 240 mg once a day and the left ventricular ejection fraction was back to normal.

The importance of diagnosing VSA relates to the major adverse events associated with this disorder including sudden cardiac death — as in our patient, acute myocardial infarction, and syncope, which may occur before the diagnosis of VSA is considered; the potential to prevent these adverse events by avoiding potential coronary artery spasm precipitants (e.g. vasoconstrictors) and the use of established effective therapies (calcium channel blockers and nitrates) [2,3].

Unfortunately VSA diagnosis is complex. Most of the time, physicians are faced with patients who have recurring attacks of chest pain consistent with intermittent myocardial ischemia, however, too often the ECG during a stress test is negative or equivocal, and coronary angiogram fails to reveal any critical coronary lesions. Repeated attempts have to be made to obtain objective observations (usually by ECG) during the height of a typical attack. Repeat multiple Holter-type ECG

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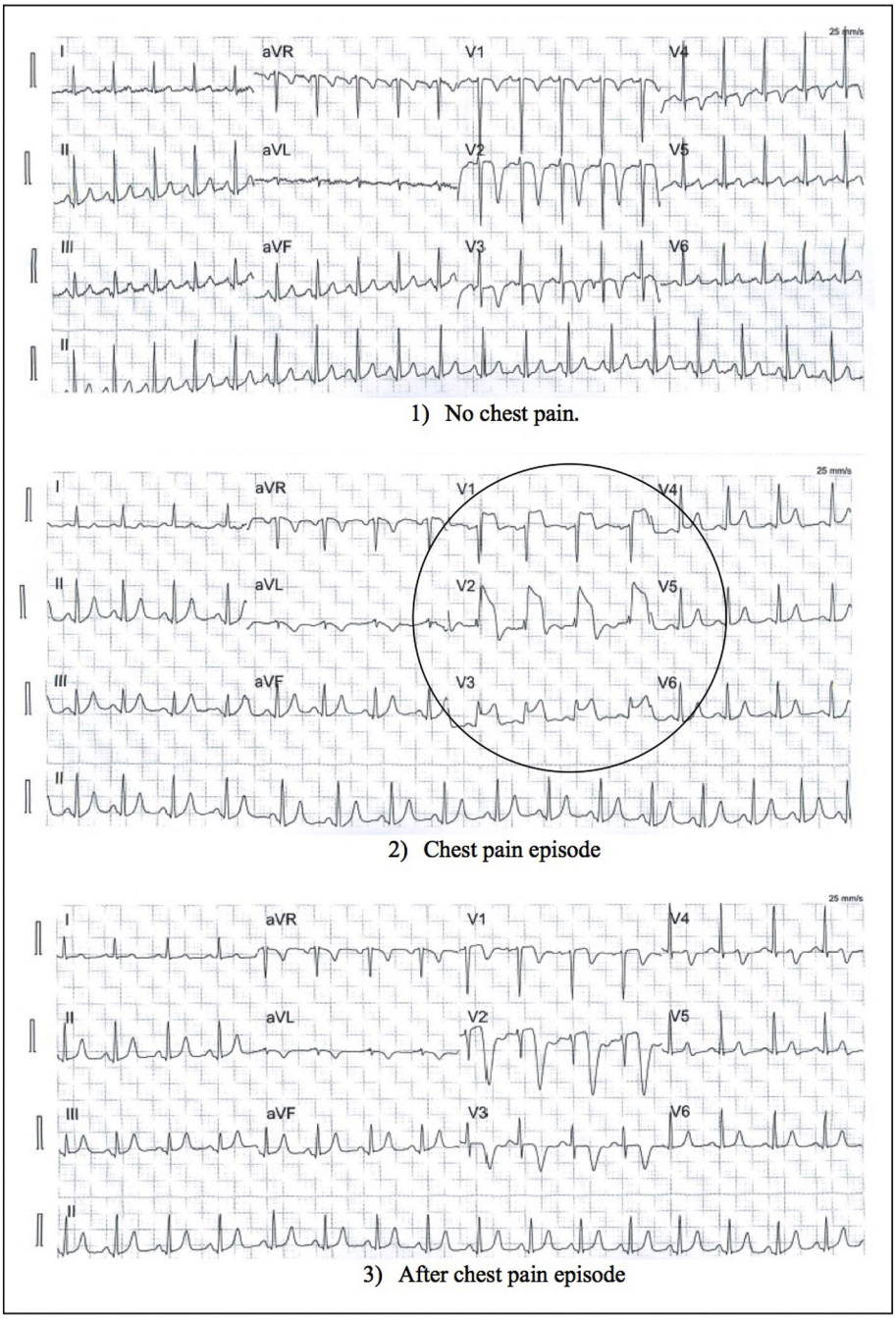

Fig. 1. 24-h ambulatory twelve-lead ECG monitoring.

recordings (twelve-lead ECG monitoring for instance), a stress test first thing in the morning and hyperventilation, can be tried [4].

Our case shows that VSA is sometimes a forgotten acute coronary syndrome and that twelve-lead electrocardiogram monitoring could be a very helpful and affordable tool in the diagnosis of VSA.

Conflict of interest

The authors report no relationships that could be construed as a conflict of interest.

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