Case report

Coronary vasospasm and aborted sudden death treated with an implantable defibrillator and stenting

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In selected patients suffering from variant angina, an implantable cardioverter-defibrillator (ICD) and coronary stenting can be helpful to prevent sudden death and treat coronary artery spasm.

We report a case of a 47-year-old woman suffering from variant angina, who experienced an episode of ventricular fibrillation promptly cardioverted. After coronary angiography documentation of a mild atherosclerosis, an ICD was implanted and oral nitrates and calcium antagonists were prescribed. The recurrence of chest pain and palpitations prompted us to perform a second coronary angiography that documented a focal coronary artery spasm successfully treated with stent implantation. No other episodes of angina or ventricular arrhythmia were documented during the following 6 months of follow-up.

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Introduction

Coronary vasospasm is a diagnostic and therapeutic challenge. Rest angina and transient ST-segment elevation characterize the clinical presentation of coronary vasospasm. The spasm is usually localized in an epicardial coronary segment, and in about two thirds of patients it occurs at the site of a mild to moderate atherosclerotic lesion[1]. Commonly, treatment for coronary vasospasm includes medical therapy with nitrates and calcium channel blocking agents. However, despite the fact that this therapy may improve the prognosis, 5 to 30% of patients continue to complain of recurrent chest pain, often associated with serious complications such as myocardial infarction, ventricular tachyarrhythmias and sudden cardiac death[2-5].

In selected cases, an implantable cardioverter-defibrillator (ICD) and coronary stenting may be helpful, relieving angina attacks and preventing sudden death. Few data are available about the combination of these two therapeutic options in the setting of vasospastic angina.

Case report

A 47-year-old female underwent cardiological evaluation for recurrent atypical chest pain and palpitations. Episodes of chest pain were mostly related to emotional stress and partially relieved by benzodiazepines. The patient's cardiac risk factors included a family history of coronary artery disease, hypertension treated with an ACE-inhibitor, and dyslipidemia. The stress test and echocardiography were normal. One month later, she presented with typical chest pain occurring at rest, radiating to the neck and left arm and followed by cardiac arrest. Basic life support was promptly initiated and documented ventricular fibrillation which was successfully treated with a DC shock.

At the time of admission, the patient's blood pressure was 145/80 mmHg and the heart rate 77 b/min; the ECG showed sinus rhythm, without evidence of acute ischemia; mild, aspecific, widespread abnormalities of ventricular repolarization were present (Fig. 1A). Cardiopulmonary evaluation and the X-ray film were normal. The computed tomographic head scan did not reveal any neurological lesions. Chemical tests were normal except for a high cholesterol level. The transthoracic echocardiogram, promptly performed, did not show any abnormalities of the regional wall kinetics.

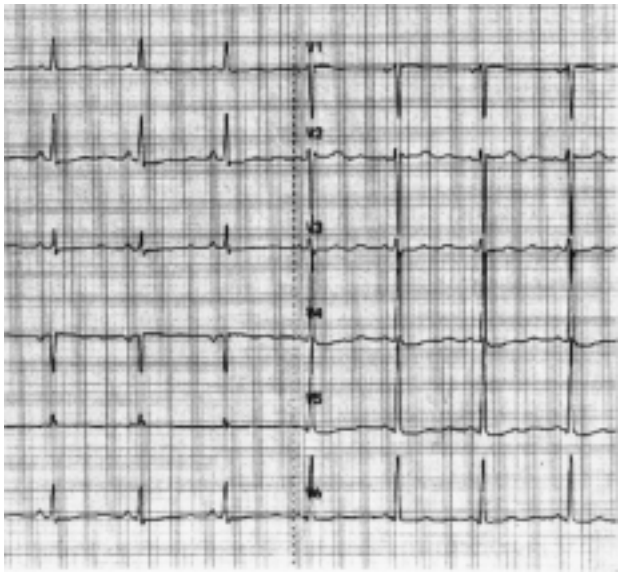
Two days later, she was submitted to coronary angiography which revealed a mild stenosis (39% on quantitative coro-

nary angiography analysis) of the proximal left anterior descending coronary artery (LAD) (Fig. 2A). The diagnosis of variant angina was considered. Pharmacological tests to demonstrate coronary spasm were not performed because of the high risk due to the recent resuscitation. Thereafter, according to the international guidelines[6], an ICD (GEM III VR Medtronic, Inc., Minneapolis, MN, USA) was implanted and the patient was submitted to oral therapy with verapamil (240 mg/die) and transdermal nitroglycerin (10 mg/die), plus acetylsalicylic acid (160 mg/die) and pravastatin (40 mg/die).

Nonetheless she continued to complain of episodes of chest pain that were relieved with sublingual nitrates. She even experienced one episode of palpitations requiring the delivery of an ICD shock. Symptoms did not relieve although pharmacological therapy was increased up to the maximal tolerated dose (320 mg/die of verapamil, and 15 mg/die of transdermal nitroglycerin). The ICD was interrogated and showed two episodes of ventricular tachycardia. One was successfully treated with appropriate therapy, the second did not last long enough to necessitate ICD intervention.

The patient was readmitted to our hospital. The ECG was continuously monitored and showed ST-segment elevation in the anterior leads and concomitant ventricular arrhythmias despite continuous nitroglycerin and verapamil infusion (Fig. 1B). At the same time the echocardiogram showed antero-septal hypokinesis with a mildly decreased left ventricular ejection fraction (45%).

Therefore the patient was again submitted to coronary angiography that documented spontaneous focal coronary spasm leading to temporary subocclusion of the proximal LAD (at the site of the previously de-



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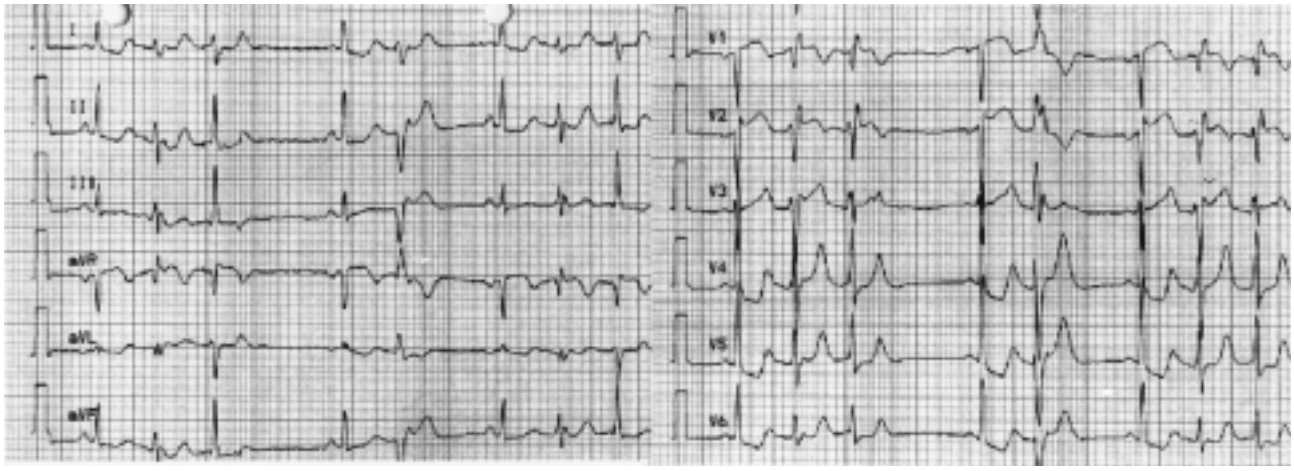


B

Figure 1. A: *electrocardiogram at the time of admission, after resuscitation from ventricular fibrillation.* B: *electrocardiogram during chest pain.*

scribed mild stenosis) during an episode of typical chest pain and the ECG changes (Fig. 2B). The spasm was promptly relieved by the intracoronary administration of 200 μg of nitroglycerin. Moreover, a left ventricular angiogram revealed an area of hypokinesis located on the antero-lateral wall. Therefore it was decided to perform direct stenting of the LAD (Multilink Tetra 4.0 × 18 mm; Guidant Advanced Cardiovascular Systems, Inc., Temecula, CA, USA) (Fig. 2C). The procedure was successful. The echocardiogram performed prior to discharge was suggestive of a normal systolic left ventricular function (ejection fraction 68%) and of mild hypokinesis on the anterior septum.

During the following 6-month follow-up symptoms did not recur. The ICD was interrogated several times and did not show ventricular arrhythmias. Dynamic ECGs were performed without any documentation of ventricular tachycardia or of transient silent ischemia. The patient refused to undergo control coronary angiography. Nitroglycerin was discontinued and verapamil gradually reduced to 120 mg/die.

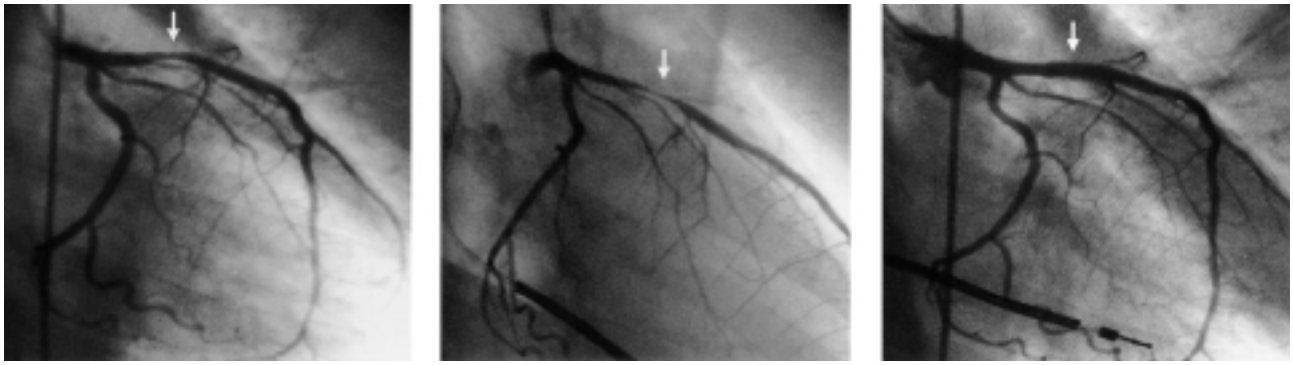
Discussion

The syndrome of coronary spasm with angiographically fairly normal coronary arteries may be disabling because of the persistence of symptoms and the occurrence of myocardial infarction, malignant tachyarrhythmias and sudden death. In most patients with variant angina, the commonly used pharmacological therapeutic strategy consists of a combination of nitrates and calcium channel blockers. This is effective in about 80% of patients. Symptoms are relieved and the prognosis is good. However, such a therapeutic regimen does not eliminate the risk of ventricular arrhythmias and of sudden death[2-5,7].

Even though the non-invasive diagnosis of variant angina is often demanding and in spite of the recent reports which suggest that accelerated exercise following mild hyperventilation and cold pressor stress echocardiography after hyperventilation can be helpful[8,9], currently a coronary artery angiography remains a "milestone". In fact, it permits the direct visualization of the site and entity of spontaneous or pharmacologically induced spasm of the epicardial coronary artery segments. Moreover, in the stenting era, after the angiographic diagnosis of a focal spasm, a coronary angioplasty may constitute a concrete therapeutic option.

In the second half of the '90s, several anecdotal reports suggested that variant angina can be approached with stent implantation whenever a segmental coronary artery spasm, localized at the level of a moderate atherosclerotic lesion, is documented[10-14]. More recently, Gaspardone et al.[15] reported a series of 9 patients with variant angina refractory to medical treatment, which resolved following stent implantation. In all patients, the intracoronary administration of methylergometrine maleate resulted in segmental coronary spasm at the level of a mild-moderate atherosclerotic lesion. At a mean follow-up of 10 months, 6 patients were asymptomatic, even though one developed asymptomatic in-stent restenosis. In 2 cases a methylergometrine-related coronary spasm occurred proximal to the previously implanted stent, and in one in the non-stented coronary branches.

With regard to the implantation of an ICD as a prophylactic option in the subgroup of patients with documented ventricular arrhythmias, Lacroix et al.[16] described 2 patients with documented vasospastic angina without any significant coronary lesions, who were resuscitated from cardiac arrest, and received an ICD. The device discharged during follow-up, in both cases. Again, Fuertes et al.[17] reported recurrent syncope and an episode of documented ventricular fibrillation in one patient. This patient had a mild atherosclerotic plaque in the mid LAD and extensive coronary spasm during acetylcholine infusion. The ICD documented several episodes of non-sustained ventricular tachycardia.



A B C

Figure 2. A: *left coronary angiogram showing a mild stenosis of the proximal left anterior descending coronary artery* (arrow). B: *left coronary angiogram (2 months later) revealing spontaneous spasm of the proximal left anterior descending coronary artery* (arrow) *during chest pain and ST-segment elevation.* C: *left coronary angiogram after stent implantation* (arrow).

This short report shows that in selected patients suffering from variant angina and concomitant episodes of sustained ventricular arrhythmias, whenever focal coronary artery spasm has been documented and when appropriate medical therapy fails, ICD implantation and stent can be considered as useful therapeutic options.

The few data available show that a stent definitively relieves symptoms in about two thirds of cases, with the possibility of developing spasm at other coronary segments in one third of patients.

Thus, due to this unstable substrate, the implantation of an ICD is mandatory in patients with life-threatening ventricular arrhythmias, even though the underlying pathology may have been successfully treated with stent implantation.

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