

Asymptomatic Coronary Artery Spasm and Severe Ventricular Arrhythmias

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SUMMARY

Coronary artery spasm generally evolves with episodes of chest pain and ST elevation. However, there are cases of coronary artery spasm without chest pain with documented ventricular tachyarrhythmias. Its incidence is unknown and it should be included in the differential diagnosis of idiopathic ventricular tachycardia or fibrillation. The case of a patient with history of two syncopal episodes with no apparent structural heart disease is described in this work. Continuous ECG monitoring revealed episodes of ST elevation that led to polymorphic ventricular tachycardia. With the diagnosis of asymptomatic coronary artery spasm, a treatment with calcium channel blockers was begun and an automatic implantable cardioverter defibrillator was implanted.

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Key words

> Coronary artery spasm - Ventricular tachycardia - Implantable cardioverter defibrillator

Abbreviations

> ICD	Implantable cardioverter defibrillator	VF	Ventricular fibrillation
ECG	Electrocardiogram	VT	Ventricular tachycardia

CLINICAL CASE

57-year-old male patient with history of hypertension and ex-cigarette smoker who is admitted to our hospital due to two syncopal episodes with no triggered factor, with general discomfort, lower limb weakness and blurred vision, with no chest pain. After his admission, the physical examination was normal and the basal electrocardiogram (ECG) showed sinus bradycardia with normal conduction and repolarization intervals (Figure 1). A cardiology study through echocardiogram and ergometry was begun. This study was interrupted 14 minutes after Bruce protocol due to physical tiredness, without findings in none of these tests. A tendency to sinus bradycardia and asymptomatic episodes of ST-segment elevation (Figure 2a and 2b) were revealed in the 24-Holter; continuous electrocardiographic monitoring was performed while awaiting the realization of a coronarography. In subsequent days, telemetry showed morning episodes of progressive elevation of ST-segment accompanied by presyncopal episodes, without chest pain, with record of fast polymorphic ventricular tachycardias (VT) (Figure 3). The electrophysiology study ruled out other arrhythmia mechanisms and flecainide test was negative. The coronarography showed a non-significant lesion in the mid segment of the right coronary artery. The coronary artery spasm provocation tests were not performed.

With a diagnosis of coronary artery spasm without chest pain and polymorphic ventricular arrhythmias, a treatment with nifedipine and isosorbide mononitrate was begun; this last one was withdrawn due to intense cephalgia and the implantation of an automatic implantable cardioverter defibrillator (ICD), which was programmed with VF zone over 200 beats per minute, was carried out. The patient was discharged and after 15 days, he was readmitted to the hospital due to two electric shocks from the device while he was going for a walk, asymptomatic. Both shocks, with episodes of polymorphic VT in VF zone, were appropriate (Figure 4). This was checked after the evaluation of the ICD. The treatment with nifedipine was intensified, verapamil was combined with it. Six months later, the patient remained asymptomatic and without new episodes of VT.

DISCUSSION

Coronary artery spasm has an important role in the pathogenesis not only of variant angina but also of ischemic heart disease in general, including cardiac sudden death. (1) Generally, it takes place during rest between midnight and early morning, although it may be induced with slight exercise at early hours in the morning, and rarely with intense exercise in the afternoon. The causes of this circadian variation are not so clear yet.

The main manifestations are chest pain and

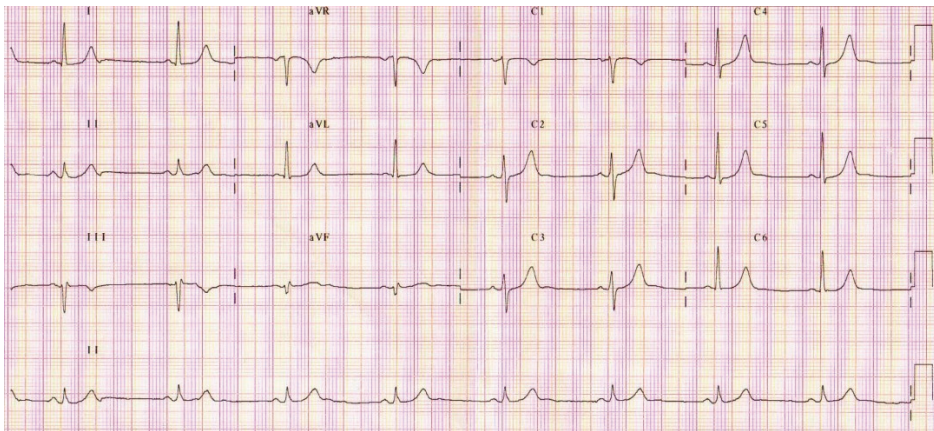


Fig. 1. Patient's basal ECG of 12 derivations and rhythm strip, in sinus rhythm, with conduction and repolarization intervals without alterations.



Fig. 2. Fragments from a 24-hour Holter. a) basal sinus rhythm with tendency to bradycardia, with isoelectric ST-segment, and b) episodes of temporary ST elevation, none of them turned into ventricular arrhythmia.

changes in ST-segment in the ECG. Chest pain is similar to that one of effort angina, although it should be more intense and long and with more vegetative courtship. However, coronary artery spasm sometimes evolves without chest pain. In fact, the incidence of silent myocardial ischemia caused by coronary artery spasm is even three times more than symptomatic ischemia. Some studies have shown that angina pain is a bad sign of the frequency in which a significant cardiac ischemia happens. (2) As regards ST elevation, its magnitude is correlated with the degree of ischemia. The affected derivations used to be the same in each attack, which indicates that the coronary artery spasm always affects the same vessel. Nevertheless, some patients present simultaneously ST elevation in derivations with different location; they are patients with multi-vessel coronary artery spasm and they present: a) more prevalence of healthy coronary arteries, b) more resistance to medical treatment and c) more predisposition to the development of severe arrhythmias. The most frequent ventricular arrhythmia associated with coronary artery spasm is polymorphic VT which may degenerate into ventricular fibrillation (VF). This VT has been documented in patients with asymptomatic coronary

artery spasm (3) and it appears more frequently when ST elevation takes place in previous derivations. There exists a good correlation between the degree of ST elevation and the incidence of arrhythmias, mainly polymorphic VT.

The realization of diagnostic tests in view of the suspicion of silent coronary artery spasm in presence of syncope is important. (4) Coronary artery spasm and the changes in the ECG do not happen during exercise, that is why ergometry has a limited value in this context. Acetylcholine and ergonovine are substances capable of inducing coronary artery spasm and they are used as provocative tests through their intracoronary injection. Ergonovine may not show coronary artery spasm in both coronary arteries. If positive, the use of nitroglycerin to revert the effect is necessary and therefore coronary artery spasm may not be caused in other coronary artery. Acetylcholine, due to its short half-life, is useful in the diagnosis of multi-vessel coronary artery spasm. However, these tests are not necessary and they are not recommended when coronary artery spasm has been spontaneously documented, (5) as it is shown in our case. It would be interesting to arise them in cases of idiopathic VF, where there are no electrocardiographic alterations

Fig. 3. Continuous electrocardiographic monitoring where sinus rhythm with isoelectric ST, with progressive elevation with morphology of subepicardial lesion and onset of autolimited polymorphic ventricular tachycardia, with output in sinus rhythm and ST-segment normalization are initially objectified.

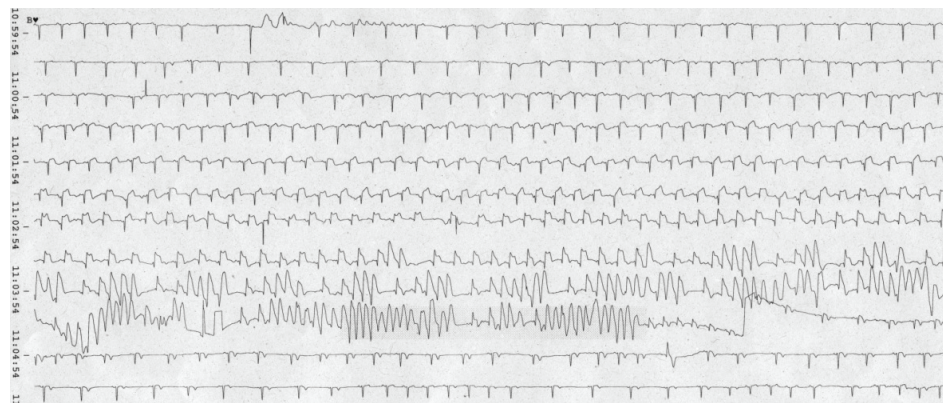
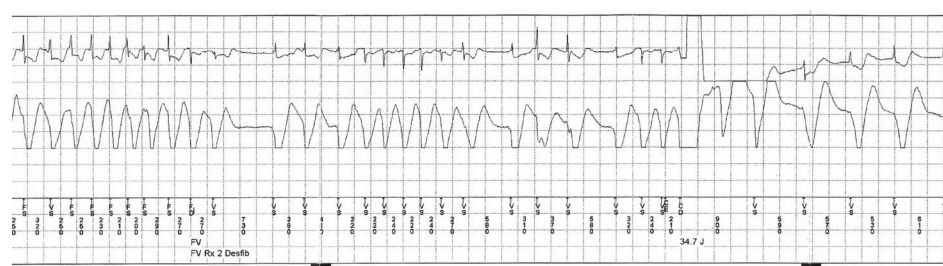


Fig. 3. Bipolar electrogram (up) and morphology electrogram (down) registered by implantable cardioverter defibrillator. Repeated sequences of polymorphic VT were objectified. Some of them were autolimited but with immediate restarting, generating a detection in VF zone and effective therapy of 35 J.



and in cases in which complementary tests, echocardiogram and coronarography, are normal. Moreover, in asymptomatic coronary artery spasm, other causes of non-clarified sudden death should be considered. Considering the diagnostic presumption of coronary artery spasm with severe arrhythmias, we completed the study with flecainide test to exclude Brugada syndrome and with an electrophysiology study to exclude inducible ventricular arrhythmias.

The acute attack of coronary artery spasm is sensitive to the use of fast-acting nitrates. The effect of these drugs is short and drugs with long effect should be used for the prevention of new attacks. Calcium channel blockers have shown a great effectiveness. Taking into account that attacks used to happen between midnight and early morning, the treatment should be arranged to midnight schedule, with gradual increase of the dose. In the multi-vessel coronary artery spasm, the treatment should not be suspended, even in absence of symptoms, since silent myocardial ischemia happens very frequently and the risk of developing lethal arrhythmias is always present. (1, 6) The cases of coronary artery spasm, which are refractory to the treatment, may require the combination of calcium channel blockers with long-acting nitrates or the association with another calcium channel blocker. In coronary artery spasm which is not related to heart disease, betablockers may deteriorate the manifestations due to isolated alpha effect, mediator of coronary artery spasm.

There are no studies that have researched the effectiveness of ICD in coronary artery spasm with ventricular arrhythmias and its indication is controversial. (7) ICD may be adequate in patients: a) revived from a VF, b) with multi-vessel spasm and ventricular arrhythmias or c) those who have presented ventricular arrhythmias and remain symptomatic despite the treatment, since the effectiveness of the medication is limited. Calcium channel blockers and nitrates reduce the incidence of episodes, but they do not guarantee the absence of recurrences of coronary

artery spasm which trigger severe arrhythmias. Recurrence cases were described some years after the first event. ICD is effective in these patients, but not in all of them, since cases in which VF was not reverted by ICD due to a persistent ischemic mechanism were documented. (7) This fact emphasizes the importance of an aggressive medical therapy, regardless of the decision to implant an implantable cardioverter defibrillator or not. Despite the treatment with calcium channel blockers, our patient continued presenting episodes of asymptomatic coronary artery spasm with development of polymorphic VT, that is why we defend the implantation of ICD despite an ideal medical treatment. (8, 9)

Asymptomatic coronary artery spasm should be considered as one of the causes of arrhythmic sudden death. Its incidence is unknown, but it should be included in the differential diagnosis of idiopathic VF. Our case reveals the phenomenon of ST-segment elevation due to coronary artery spasm and the beginning of malignant ventricular arrhythmias. The treatment with calcium channel blockers, alone or associated with nitrates, have a good prognosis; however, there are groups of patients that present elevated risk, as those with multi-vessel or silent coronary artery spasm, and an implantation of ICD should be considered. (10)

RESUMEN

Implante de cardiodesfibrilador por vía transauricular. Descripción de un caso

El vasoespasma coronario generalmente evoluciona con episodios de dolor torácico y elevación del ST. No obstante, existen casos de vasoespasma sin dolor torácico con taquiarritmias ventriculares documentadas. Su incidencia se desconoce y debe incluirse en el diagnóstico diferencial de taquicardia o fibrilación ventricular idiopática. En esta presentación se describe el caso de un paciente con historia

de dos cuadros sincopales sin cardiopatía estructural aparente. La monitorización electrocardiográfica continua objetivó episodios de elevación del ST que conducían a taquicardia ventricular polimorfa. Con el diagnóstico de vasoespasma coronario asintomático se inició tratamiento con calcioantagonistas y se implantó un cardio-desfibrilador automático.

Palabras clave > Vasoespasma coronario - Taquicardia ventricular - Cardiodesfibrilador implantable

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Conflict of interest statement

Authors declare no conflict of interest.