Cardiology & Cardiovascular Research

*Case Report*

# Coronary Thrombosis Due To Atypical Mechanism during Radiofrequency Ablation: A Case Report

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## Abstract

Radiofrequency ablation is currently used for the treatment of several arrhythmias, including premature ventricular complexes (PVCs) in cases of persistent symptoms despite optimized medical treatment and arrhythmia-induced-cardiomyopathy. The most common complications are those related to the vascular access, followed by cardiac tamponade. Atrioventricular blocks, coronary lesions and neurological alterations may also occur. We report a case of a fifty-year-old female referred for catheter ablation of PVCs, who evolved with cardiac tamponade and cardiorespiratory arrest during procedure. After pericardiocentesis and return of spontaneous circulation, a persistent anterior wall ST-segment elevation was noted. Immediate coronary angiography showed occlusion of the left anterior descending artery, probably secondary to extrinsic compression by the pigtail catheter in the pericardial sac during chest compressions, a mechanism not yet described of myocardial infarction during ablation. Primary percutaneous coronary intervention was successfully performed. The patient had favorable evolution after the complications, with no ventricular dysfunction, and was discharged from hospital after seven days.

## Keywords

Premature ventricular complexes, Ablation, Cardiac tamponade, Acute myocardial infarction, Cardiac arrest.

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## Introduction

Premature ventricular complexes (PVCs) are common arrhythmias in clinical practice that range from asymptomatic to heart failure due to PVC-induced cardiomyopathy. When, despite medication, the patient remains symptomatic or evolve with arrhythmia-induced cardiomyopathy, suppression of PVCs with catheter ablation must be considered [1]. This is an increasingly widely used therapy and receives class I recommendation in these scenarios, except in patients with normal left ventricle function with PVCs originating elsewhere other than the right ventricle outflow tract. In this case, a class IIa recommendation is given [2]. Noteworthy, catheter ablation of PVCs is usually a safe procedure, with rare major complications [3].

The most common complications of catheter ablation are those related to the vascular access. However, the risk of cardiac tamponade cannot be underestimated [3]. We report a case of PVC catheter ablation that evolved with cardiac arrest due to

cardiac tamponade, then presenting acute ST-elevation myocardial infarction (STEMI) after return of spontaneous circulation due to an atypical mechanism.

## Case Report

Fifty-year-old female outpatient with no previous comorbidities, presenting symptomatic PVCs. The resting electrocardiogram (ECG) showed frequent PVCs probably originated in the right ventricle outflow tract (figure 1). She reported frequent palpitations compromising quality of life. The 24-hour Holter monitorization showed 13,008 PVCs in 24 hours (14.1% of all heartbeats). During the investigation, a coronary angiography was performed, which did not show obstructive lesions. Magnetic resonance imaging of the heart showed late epicardial enhancement of non-ischemic etiology in the anterolateral (basal) and inferolateral (basal and middle) segments of the left ventricle, with normal cavity diameters and without ventricular dysfunction.

Treatment with amiodarone 200 mg/day was started, but the patient did not tolerate dose titration and symptoms of palpitations remained. Catheter ablation was then indicated. The procedure was performed under general anesthesia, through the right femoral vein, with atrial transseptal puncture. Electrophysiological mapping was performed in the right and left ventricles, guided by mapping score with the EnSite Precision® electroanatomical system (Abbott Cardiovascular, Chicago, USA). The mapping demonstrated that the origin of the arrhythmia was in the right ventricular outflow tract (best score of 91%). Radiofrequency applications were carried out in the area of interest. At the end of the procedure, the rhythm was sinus with episodes of junctional escape. The patient suddenly developed hemodynamic instability. Alteration of the cardiac silhouette was observed in fluoroscopy, suggesting pericardial effusion. The patient presented cardiac arrest in asystole, with return to spontaneous circulation after six minutes of cardiopulmonary resuscitation and pericardial drainage of 350 ml through pericardiocentesis. A 6 French pigtail catheter was used for pericardial drainage and was placed in the pericardial sac. After 15 minutes, the patient evolved with ventricular tachycardia, and electrical cardioversion was immediately performed. Then, the electrocardiogram showed sinus rhythm and ST-segment elevation in the anterior wall. Bedside echocardiogram confirmed resolution of the pericardial effusion and showed segmental systolic dysfunction affecting the anterior wall of the left ventricle. Urgent coronary angiography was performed, which showed occlusion of the left anterior descending artery (LAD) by a thrombus (figure 2A). Primary percutaneous coronary intervention (PPCI) was successfully performed with mechanical recanalization and implantation of a sirolimus-eluting stent Supraflex Crux 3,0x18 mm (SMT, Surat India), with a TIMI III flow at the end (figure 2B). The patient presented persistent shock with the need for norepinephrine and maintenance of mechanical ventilatory assistance, and was transferred to the intensive care unit (ICU).

The patient evolved favorably, and the pericardial catheter was removed after 48 hours, after she was extubated. She was discharged from the ICU within four days, and discharged from the hospital seven days after the procedure. At the outpatient return appointment after five months, the patient was asymptomatic and a transthoracic echocardiogram showed no left ventricular dysfunction.

## Discussion

PVCs are very common within the spectrum of ventricular arrhythmias, with an incidence that increases with age [4]. The estimated prevalence of PVCs is 1 to 4% in the general population on standard electrocardiography and 40 to 75% of subjects on Holter monitoring [4-7]. Some studies have shown that, in the general population, frequent PVCs (at least one PVC on a 12-lead ECG or >30 PVCs per hour) are associated with increased cardiovascular risk and increased mortality. Because of this association, the detection of PVCs is generally considered as a risk factor for cardiac events [4]. PVCs in the structurally normal heart are usually benign [7]. However, investigation of underlying conditions such as ischemic heart disease and ventricular dysfunction are necessary, since these cases carry greater risk of sudden death [4,5].

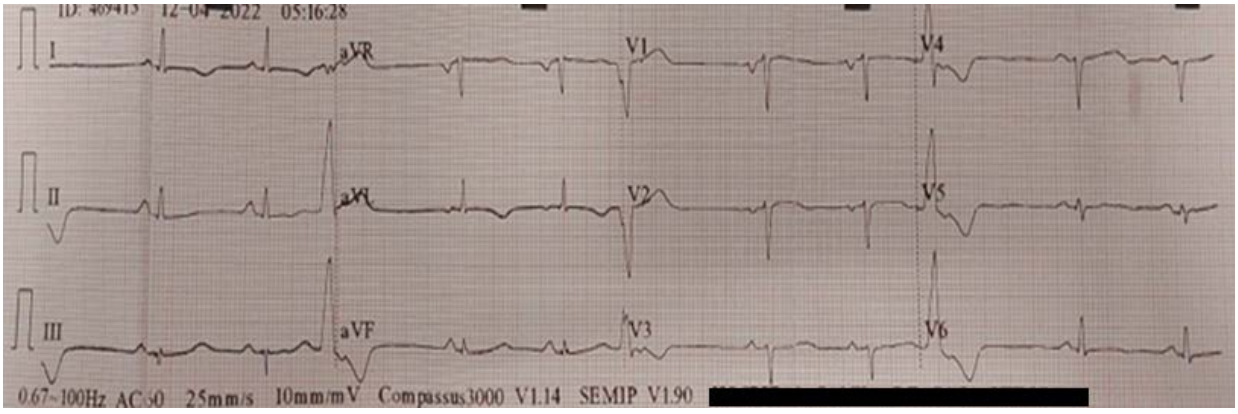


**Figure 1:** Baseline resting electrocardiogram showing sinus rhythm, normal axis of QRS complex, negative T waves in the lateral wall, slow progression of R wave in the anterior wall, and premature ventricular complexes with a left bundle branch block pattern.

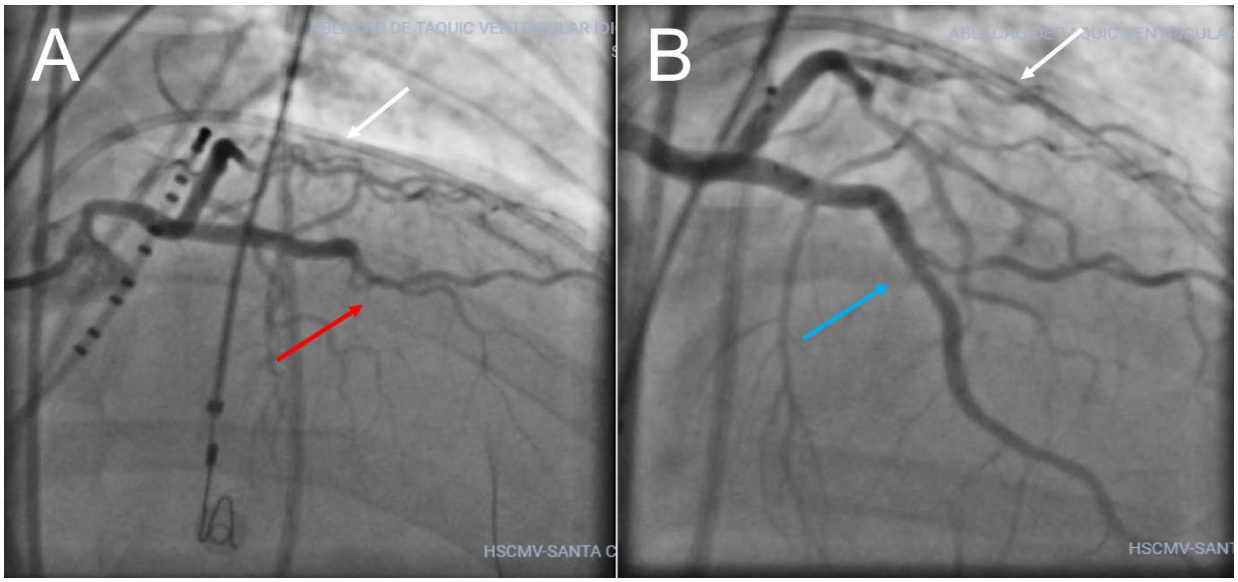


**Figure 2:** Urgent coronary angiography and primary percutaneous coronary intervention. A- Anteroposterior cranial view showing occlusion of the left anterior descending artery (red arrow); pigtail catheter placed in the pericardial sac (white arrow). B- Left anterior descending artery with normal anterograde flow after primary percutaneous coronary intervention with implantation of a sirolimus-eluting stent (blue arrow); pigtail catheter placed in the pericardial sac (white arrow).

Many patients with PVCs are asymptomatic, whereas some may present symptoms of palpitations, dizziness, near-syncope, dyspnea, chest pain, or fatigue [4,8]. Nevertheless, even in asymptomatic patients, an incidence of PVCs >10,000 to 20,000 per day can lead to impairment of left ventricular systolic function, an entity recognized as PVC-induced cardiomyopathy, an often reversible cardiomyopathy [4-6]. Given the silent development of this endpoint, the ABC-VT score measures the patient's risk of adverse events by analyzing 14-day ECG patch monitoring data, such as PVC morphology, axis, burden, coupling interval and presence of ventricular tachycardia. It classifies patients with PVCs according to their score into low (0-4), intermediate (5-8) and high risk (9-12) [9]. In cases of PVC-induced cardiomyopathy, reducing the density of PVCs with catheter ablation or pharmacological suppression has the potential benefit of reversing the condition [4,5]. Drug treatment with beta-blockers, non-dihydropyridine calcium channel blockers, and some antiarrhythmic medications can be used to relieve symptoms [2,5]. If antiarrhythmic medications are ineffective, not tolerated, or not desired by the patient, catheter ablation plays an important role as a treatment option, with more effectiveness for reducing the number of PVCs per day and for increasing left ventricle ejection fraction than medical therapy alone [4,5,10].

As most ventricular arrhythmias originate close to the subendocardium, the interventional approach can be through a transvenous (for the right ventricle) or transaortic/transeptal (for the left ventricle) catheterization [4]. An integration of visual information from fluoroscopy and/or intracardiac echocardiography as well as localizing the site of earliest activation is important [5]. Although there are several diagnostic algorithms that use the data collected on the ECGs during PVCs, given the failure to diagnose the exact origin of the PVC in some cases, an activation map is recommended, using either a standard ablation catheter or a mapping system capable of multielectrode mapping [5,11]. The catheter ablation procedure usually involves attempts to induce ventricular tachycardia by programmed electrical stimulation to confirm the diagnosis and guide radiofrequency ablation [3,4]. Problems limiting success include inability to induce an arrhythmia from mapping (common with idiopathic ventricular arrhythmias), or origin of the arrhythmia from an inaccessible location in the myocardium, which is more common in some cardiomyopathies [4].

Complications from catheter ablation of PVCs are relatively low – 2.2 to 2.4% of major complications in two recent multicenter studies. Among these, one-half were related to vascular access, and pericardial tamponade was hardly seen (0.22 to 0.8%). In both studies, no patients experienced perioperative stroke, and no patients died [3,6]. Other risks include atrioventricular block, damage to the coronary arteries, aortic dissection and neurologic complications [1,8].

In our case, the patient evolved with cardiorespiratory arrest after cardiac tamponade, which is itself a rare and life-threatening complication. After pericardial drainage from pericardiocentesis during a successful cardiopulmonary resuscitation, a 12-lead electrocardiogram showed ST-segment elevation in the anterior wall. As the patient had undergone a coronary angiogram prior to the procedure, which did not show coronary obstructions, we considered that the probable mechanism of the myocardial infarction was coronary thrombosis. This unexpected additional complication was most likely caused by extrinsic compression from the pigtail catheter placed in the pericardium sac, combined with

chest compressions for cardiopulmonary resuscitation. Despite the serious complications, the patient was promptly treated and evolved well.

## Conclusion

Over the years, advances have been made in catheter ablation techniques, making it even faster and safer. However, although the rate of complications shown in recent studies is low, indication of the procedure must be done carefully and risks must be weighted against benefits. In our case, an acute myocardial infarction occurred during procedure due to coronary thrombosis apparently caused by extrinsic compression from a pigtail catheter placed in the pericardium sac, combined with chest compressions for cardiopulmonary resuscitation after a cardiac tamponade. This sequence of serious complications and this mechanism of myocardial infarction during ablation had not yet been described.

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