Abellás-Sequeiros et al. Int J Clin Cardiol 2015, 2:5
ISSN: 2378-2951

International Journal of
Clinical Cardiology

Case Report: Open Access

Arrhythmic Manifestation of Prinzmetal´ Sangina Induced by Therapeutic Hypothermia

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Introduction

Variant angina was first described by Prinzmetal et al. [1] like an episode of chest pain with transient ST-segment elevation. However, about 80% of patients course in an asymptomatic way [2]. In other cases, syncopeor sudden cardiac death are the mainly manifestation.

Case Report

We report the case of a 37-year-old Caucasian man, with prior history of smoking and DM. He had complained about multiple episodes of syncope during last year. However, the day of his hospitalization, he described a sudden and acute episode

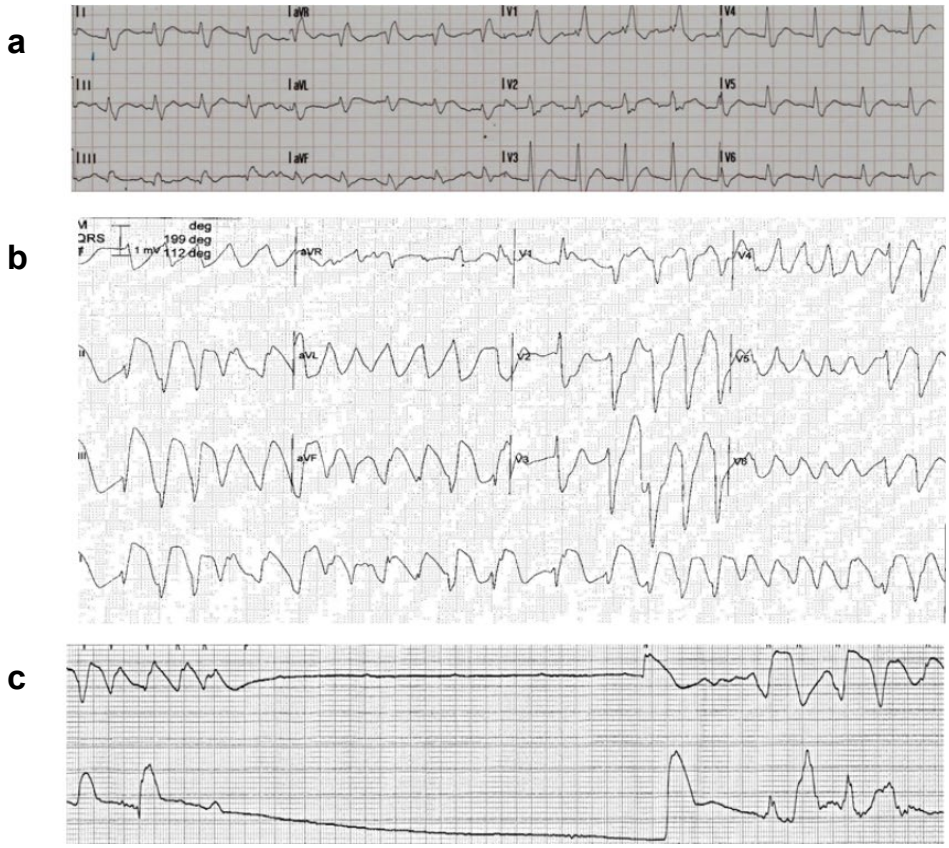

Figure 1a: Control ECG, **Figure 1b:** ECG recordings with ventricular tachycardia during rewarming phase of hypothermia, **Figure 1c:** ECG recordings showing complete AV block

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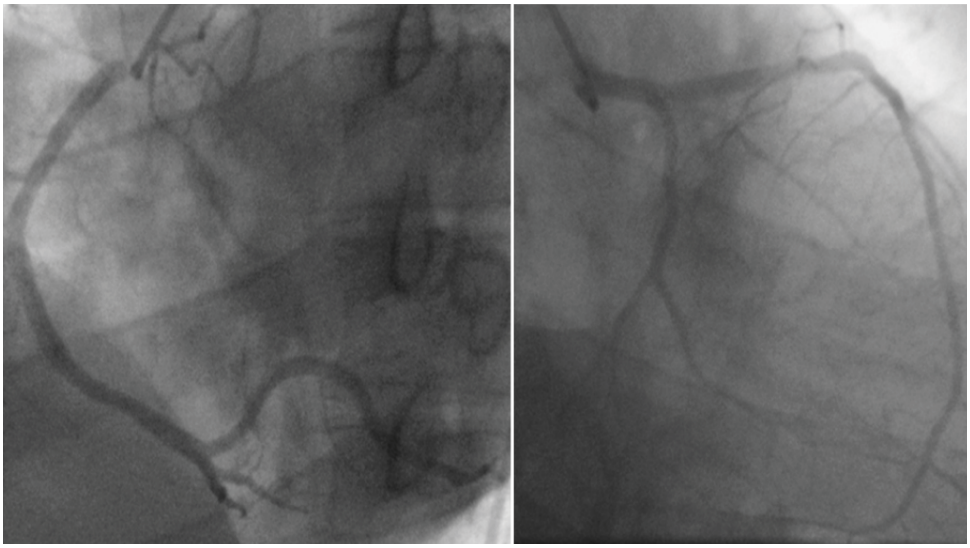
Citation: Abellás-Sequeiros RA, Ocaranza-Sanchez R, García-Acuña JM, González-Juanatey JR (2015) Arrhythmic Manifestation of Prinzmetal´ Sangina Induced by Therapeutic Hypothermia. Int J Clin Cardiol 2:048

Received: August 11, 2015: **Accepted:** August 31, 2015: **Published:** September 03, 2015



a b

Figure 2a: Right coronary artery with no angiographically significant lesions, **Figure 2b:** Normal left coronary angiogram

of chest pain which stopped him from sleeping. He arrived to the emergency department after 45 minutes of successful resuscitation, with VF as first cardiac rhythm detected. Admission ECG revealed sinusal rhythm with RBBB (Figure 1a) and two-dimensional echocardiogram showed a generalized hypokinetic myocardium with severe depression of LVSF. Haemogram and ionography blood tests were normal. Coronariography showed no significant lesions on epicardial coronary arteries (Figure 2a and Figure 2b). He fulfilled the entire criteria for therapeutic hypothermia. A rapid induction phase was followed by 24 hours at 32°C. During rewarming phase ECG showed transient ST-elevation followed by polymorphic ventricular tachycardia and VF (Figure 1b) and an episode of complete AV block (Figure 1c). The image of the bedside echocardiogram was not compatible with Takotsubo cardiomyopathy. This echo represent one of the main differential diagnosis in this case [3,4]. In fact, we could see a global myocardium hypokinesis with no segmentary defects in left ventricle contraction. These arrhythmic events and ST-T changes immediately disappeared after initiating parenteral nitroglycerine. Further treatment consisted in nitrates and calcium channel blockers, and an ICD was implanted to him. He was discharged after an uneventful hospital stay and, at that moment, the echocardiogram demonstrated the improvement of LVSF which was in normal range. Today, he keeps asymptomatic without electrical therapies of ICD.

Discussion

Pathophysiology of variant angina remains unclear. 70% of patients have normal angiograms [5]. Artery spasm could be the result of an inadequate tone in smooth muscle fibers of the vessels in addition to a dysfunctional endothelium [6]. Tabaquism is a major precipitating factor of this entity [7], as free radicals generated by smoking can damage the endothelium and decrease NO levels. Ischemia is not always synonym of angina. In fact, 80% of patients with vasospastic angina are asymptomatic. Khisida et al. [2] showed 12% of patients course with history of syncope, which could be the clinical manifestation of malignant arrhythmic events, inadequate atrio-ventricular conduction, sinusal disfunction or asystolia. Provocation tests could be considered to prove coronary artery spasm (level of indication IIa C) [8]. We decided not to perform them according to European guidelines [8] because of the high degree of accuracy of the diagnosis based on typical clinical presentation in a patient with acute ECG changes and no obstructive lesions on epicardial coronary arteries. Therapeutic hypothermia is worldwide used to avoid neurological damage after cardiac arrest. During rewarming phase, Firmin et al. [9] demonstrated an increased in catecholamine levels, which favored arterial spasm due to the activation of alfa receptors of the vessels. During induction phase of hypothermia [10], decreasing serum levels of magnesium are warranted with high risk of arterial spasm due to higher intracellular levels of calcium in smooth muscle fibers. Arterial spasm could be controlled by intravenous nitrates so we support hypothermia use as neurological benefits are higher than risks. According to EHRA expert consensus on ventricular arrhythmias [11] we inserted the patient an ICD. Matsue et al. [12] also suggested its use in patients with history of VT or VF and variant angina, who are in high risk of sudden cardiac death. Controversial discussion focused on patients without documented arrhythmic events, as there is a lack of evidence about ICD use in primary prevention. Nevertheless, we want to highlight medical treatment with drugs which induce vasodilatation as the cornerstone in management of patients with vasospastic angina. Medical treatment must be optimized and be continued after an ICD implantation.

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