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Case report
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Transient electrocardiographic recording of acute myocardial ischemia with ST-segment elevation of the diaphragmal location accompanied by a total atrioventricular block as an initial manifestation of Prinzmetal's angina – A case report

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Abstract: Prinzmetal's angina, also known as Prinzmetal's variant or Prinzmetal's vasospastic angina is characterized by angina attacks caused by spasm of the great epicardial coronary arteries. Coronary artery endothelial dysfunction plays a crucial role in the development of this vasospastic angina. The attacks of vasospastic angina can be prevented with calcium antagonists and nitrates, whereas in refractory variant angina, coronary angioplasty with stenting may help prevent further coronary spasm. In this case report, we present a 52-year-old male patient with a transient electrocardiographic recording of acute myocardial ischemia with ST-segment elevation of the diaphragmal location accompanied by a total atrioventricular block immediately after exercise testing and as a first manifestation of Prinzmetal's angina. After regression of the symptoms and electrocardiographic changes, significant pathomorphologic changes of coronary arteries were excluded by coronary angiography. Following discharge, the patient was treated with calcium antagonists and did not show symptoms during a 4-year follow-up period.

Keywords: Coronary disease, vasospasm, Prinzmetal's angina, exercise

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1 Introduction

Prinzmetal's angina, also known as Prinzmetal's variant or Prinzmetal's vasospastic angina, is a clinical entity characterized by chest pain that occurs predominantly at rest, rarely after exercise, and as a result of a spasm of the great coronary arteries. It is typically manifested by ST-segment elevation on the electrocardiogram (ECG) in leads that reflect the perfusion area of the coronary artery affected by the spasm. Coronary angiograms show mostly nonsignificant stenotic changes of the coronary arteries, although in a vast minority of cases, the epicardial coronary arteries are angiographically normal. Between the attacks of angina, which may show a circadian pattern with prevalence in the early morning or nocturnal hours, ECG recordings are generally normal [1].

The etiology and incidence of Prinzmetal's angina in the general population is not known. It most frequently appears in adult males (50 to 60 years), although some authors have reported cases of vasospastic angina in adolescents [2]. Epidemiological studies show that 0.5% to 1% of patients hospitalized for angina pectoris suffer from Prinzmetal's angina [3]. It is thought that pathophysiological changes in blood vessels due to atherosclerosis cause a dysfunction of the endothelium, thus lowering the secretion of nitric oxide, which plays a basic role in the development of vasospastic angina [1].

Patients with Prinzmetal's angina that lack pathomorphologic changes of coronary arteries have an excellent prognosis (>90% survival over a 5-year period). Patients with associated stenotic changes of coronary arteries have a higher risk. In these patients, the incidence of acute myocardial infarction (MI) increases 15% in the 3-month period following diagnosis of variant angina [1]. The most common complications of vasospastic are MI, malignant arrhythmia, and sudden cardiac death [1].

Attacks of vasospastic angina can be effectively prevented by moderate or high doses of calcium antagonists in addition with long term nitrates, whereas the use of beta blockers is contraindicated. Sublingual use of short term nitrates usually leads to regression of chest pain as well as ECG changes.

In the current case report, we present a 52-year-old male patient with a transient ECG recording of acute MI with ST-segment elevation at the diaphragmal location accompanied by a total atrioventricular (AV) block immediately after exercise testing as a first manifestation of Prinzmetal's angina.

2 Case report

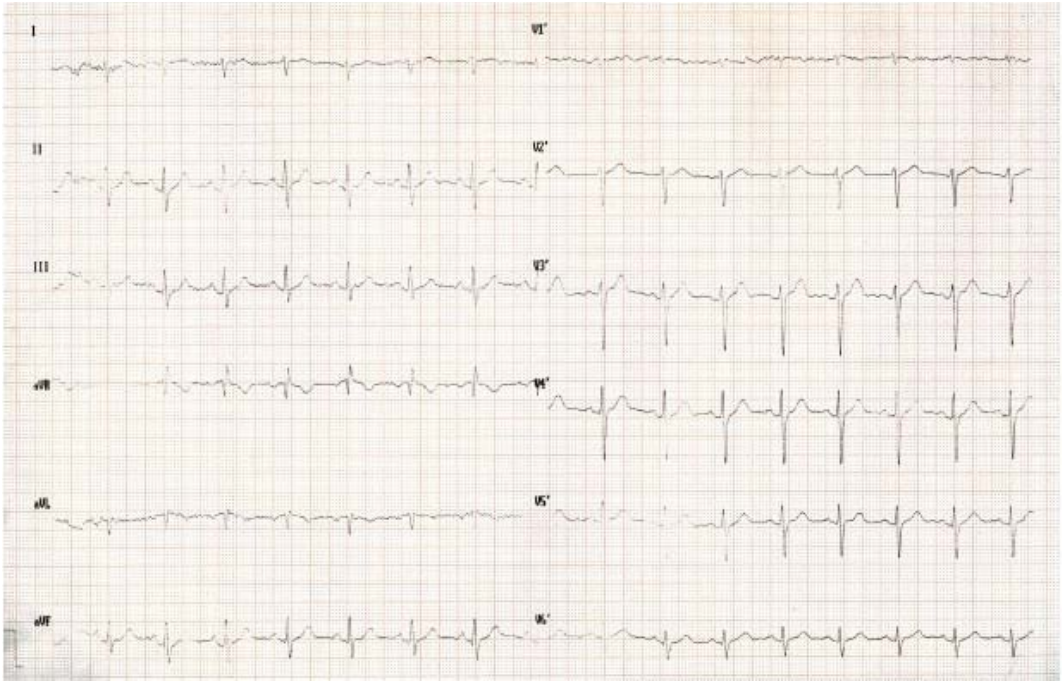
A 52-year-old male smoker was referred by his general practitioner for an exercise test in April 2001. The patient was healthy until a month before his visit to the general practitioner. At that time, after a mild physical effort (a slow 20-min walk on a flat surface), he felt discomfort and non-spreading precordial oppressions that lasted for about 5 min and spontaneously ended. Until his arrival in our hospital for the exercise test, he had not experienced additional similar discomfort.

Except for mild arterial hypertension (150/90 mm Hg), there were no aberrations

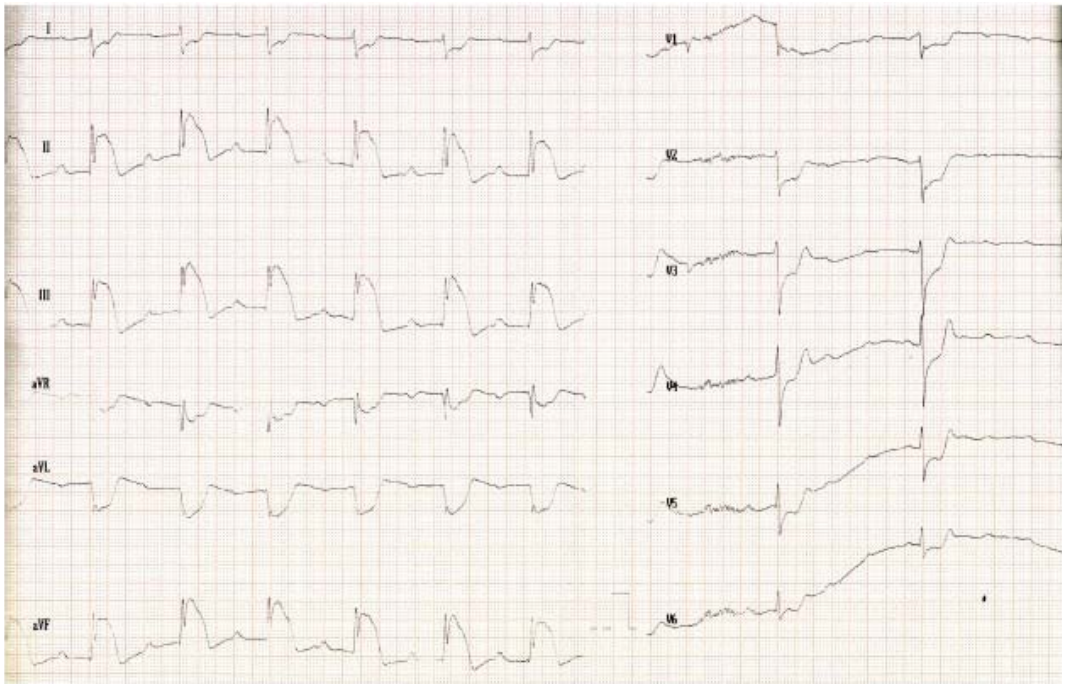
before the beginning of the exercise test. A baseline ECG recording showed a sinus rhythm of 91 beats per min, without ventricular ectopic activity or ischemic ST-T changes. A symptom-limited exercise test was performed on a bicycle ergometer (Cardioline ECT WS 2000) with an initial workload of 50W and progressive augmentation for 25 W every 2 min. During 8 min of testing, the patient achieved a workload of 125 W (86% of theoretical maximum for age, 7.7 metabolic equivalents (METs). The maximum heart rate was 170/min (101% of theoretical maximum), and the maximum values of arterial blood pressure were 205/105 mm Hg. During the exercise test, no sinus rhythm aberrations or significant pathological ST-T segment oscillations were recorded. Five minutes after testing, the patient began to complain about a severe chest pain spreading in his left shoulder and upper arm, accompanied by a hipotensive reaction (80/60 mm Hg), still with no pathological ECG changes. The patient was immediately placed in Trendellenburg's position. After 30 s, the ECG recording showed a PR prolongation of up to 360 ms, followed by a ST-segment elevation in the D2, D3, and aVF leads up to 0.7 mV, with ascendant ST-segment denivelation in D1, aVL, and precordial leads V1-V5 up to 0.7 mV. Seven min after testing, the heart rate rapidly decreased from 125/min to 62/min, followed by an ECG recording of a total AV block with a ventricular frequency of 38/min (Figure 1).

The clinical findings and ECG changes suggested that the patient suffered from an acute MI with ST-segment elevation accompanied by a total AV block as a result of the exercise test. The patient was promptly transferred to our coronary care unit. After administration of sublingual and parental nitrates (bolus dosage of 0.5 mg intravenous.) and infusion of saline over a period of 5 min, there was a rapid and complete regression of ECG changes and chest pain. The laboratory findings showed no significant increase in cardioselective biomarkers (peak CK-MB = 15 [reference range <16] and troponin I = 0.02 [reference range 0.04–0.4]). Hypercholesterolemia was found (total cholesterol = 6.0 [reference range <5.0], low density lipoproteins (LDL)l = 4.2 [reference range <3.0], high density lipoproteins (HDL) = 1.2 [reference range >1.1], and triglycerides = 0.8 [refrence range <1.7]). Immediately after the patient was stabilized, a coronary angiography was performed, and intact coronary arteries were found. During intracoronary catheter manipulation, a significant right coronary artery (RCA) spasm was found on the middle and distal third of the RCA that promptly disappeared after intracoronary application of nitrates (Figure 2). During the catheterization procedure, the patient did not report chest pain. Also, ST segment denivelation or AV block was not observed.

When significant stenotic changes of coronary arteries were excluded, the patient was treated with calcium channel antagonist lacidipine 4 mg, acetylsalicylic acid 100 mg, and simvastatin 20 mg. During further treatment, the patient was stable, with normal arterial blood pressure and heart rate, without further angina attacks. On the fourth day of treatment, the patient was discharged. A second symptom-limited exercise test was performed a month later, during which the patient achieved a workload of 125 W with no ECG recorded ischemic ST-T changes or aberrations in the heart rate. In the follow-up period of almost 4 years after the first episode of angina, during which the patient stopped

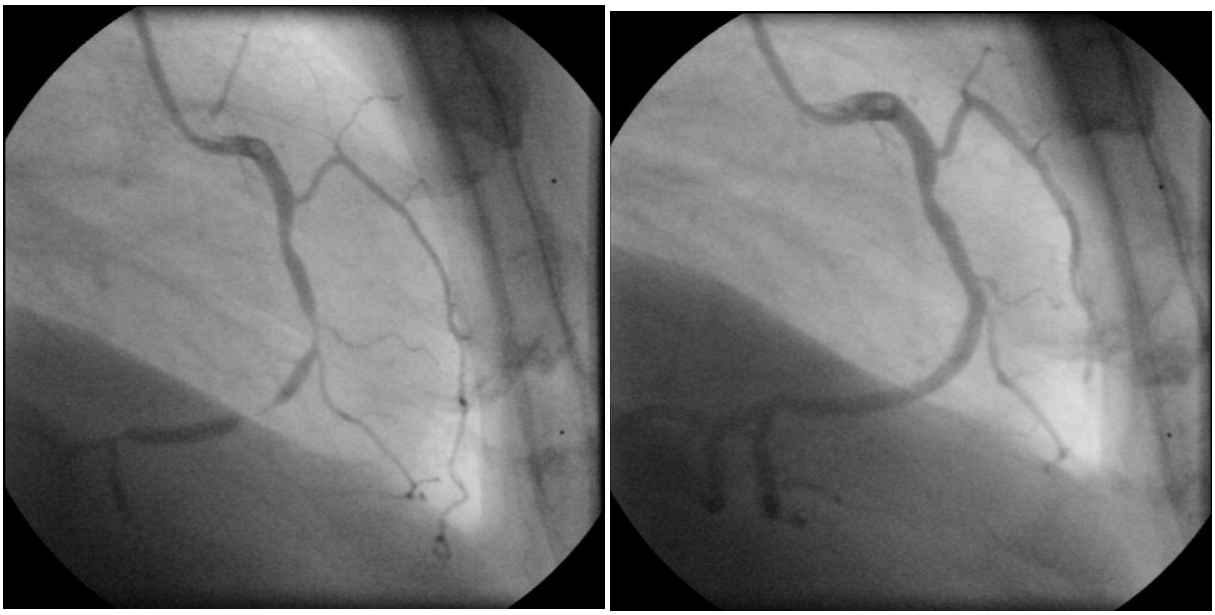


a)

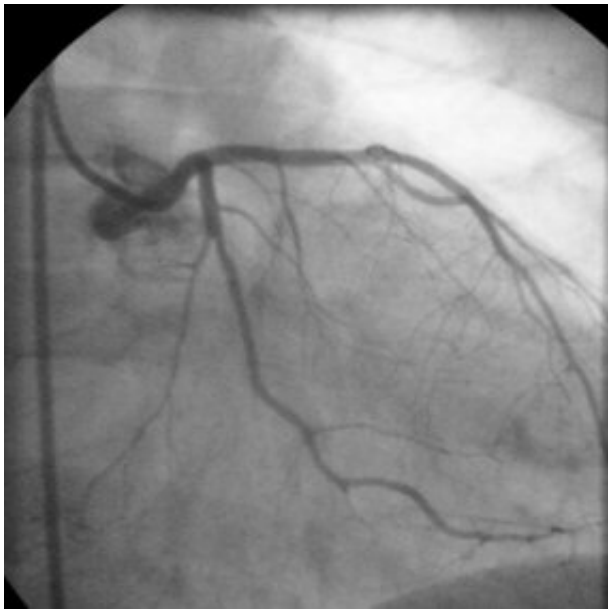


b)

Fig. 1 Dynamics of ECG changes during exercise testing.
a) ECG before exercise testing;
b) ECG in the seventh min after exercise testing. Severe ST elevation in II, III, and aVF leads, accompanied by a total AV block (see precordial leads)



a) b)



c)

Fig. 2 Coronary angiogram of the patient.
a) Right coronary artery showing spasm;
b) Right coronary artery after intracoronary application of nitrate;
c) Left coronary artery.

smoking and continued the drug therapy, he did not report symptoms. In July 2005, a control coronary angiography was performed, during which significant pathomorphologic changes of coronary blood vessels were excluded. A third symptom-limited exercise test was performed at this time, during which there were no ECG ischemic ST-T changes or aberrations in the heart rate.

3 Disscusion

We describe here a case of Prinzmetal's angina that is interesting for several reasons. First, after just one attack with relatively non-specific symptoms, during the seventh minute of an exercise test, the patient showed ECG signs of acute occlusion of the RCA accompanied by a total AV block and haemodynamic instability. Exercise testing is a safe diagnostic method and is rarely accompanied by serious complications when performed according to current standards and guidelines. Recent reports show that the incidence of sudden cardiac death during exercise test is 0.03%, whereas the risk of development of MI or malignant arrhythmias is 0.09% [4]. Also, some authors report that Prinzmetal's angina itself is rarely accompanied by significant arrhythmias, which we also found in this case [5].

Second, shortly after admission to the coronary care unit, during the course of planning an urgent coronary angiography and Percutaneous coronary intervention (PCI), the patient received intravenous and sublingual nitrates, resulting in an immediate regression of chest pain and ECG changes, along with complete haemodynamic stability. These findings suggested that this was an episode of Prinzmetal's angina or, less likely, a case of promptly recanalyzed acute coronary artery thromboembolism. A final diagnosis of Prinzmetal's angina was made after coronary angiography, during which catheter manipulation verified significant spasm of the RCA without effects on other coronary arteries. Because the spasm of RCA was verified "ad hoc", neither acetylcholine nor ergonovine tests were performed, which would otherwise be the next standard step in the diagnosis of Prinzmetal's angina [3]. Instead, an immediate application of intracoronary nitrates led to full and rapid regression of the spasm. Interestingly, despite a coronary artery spasm lasting approximately 10 min, the laboratory findings showed no significant myocardial lesion, and repercussions on regional myocardial contractility were found.

According to previous reports and our experience, Prinzmetal's angina usually presents at rest and rarely during the exercise, as in the current case. It is also interesting that, after diagnosis of Prinzmetal's variant angina and administration of calcium antagonists, the patient showed no significant pathomorphologic changes of coronary arteries in the coronary arteries during an approximately 4-year follow-up period.

The etiology and pathophysiology of vasospastic angina has been the subject of many studies. It is currently thought that dysfunction of the coronary artery endothelium plays a crucial role in the pathogenesis of this disease. Using intravascular ultrasound, Miyao et al. [6] showed that intimal thickening in patients with vasospastic angina is limited to the coronary arteries. Some recent reports have shown that ongoing inflammation of the coronary artery endothelium also increases the risk of developing vasospasm [7, 8]. Many other studies have examined the efficiency of different ways of preventing coronary spasm, including administration of magnesium sulfate [9] or vitamin E [10], although these are not supported for routine use by recent guidelines [3]. Some cases of vasospastic angina are refractory to optimal drug therapy. Major cardiac complications such as acute MI or sudden cardiac death can be prevented in these patients by implantation of

an intracoronary prosthesis in areas showing moderate or significant stenotic coronary artery changes [3, 11]. Some studies also report percutaneous coronary intervention by implantation of a stent, even in angiographically normal segments of coronary arteries, for the treatment of recurrent spasm that is refractory to drug therapy. In such cases, the use of intravascular ultrasonography proved to be helpful in identifying subclinical high-risk plaques in positively remodeled and angiographically normal vessels [12, 13].

This case report demonstrated the importance of an early-stage diagnosis of Prinzmetal's angina. The complications occurred in controlled hospital environment, allowing us to act promptly. Should they have occurred outside of the hospital, the patient's outcome and the results of treatment would surely have been poorer.

Because Prinzmetal's angina with ECG changes of such magnitude is relatively rare and potentially fatal, the aim of this case report was to highlight the various clinical presentations of vasospastic angina and to emphasize the contribution of invasive cardiology in its diagnosis as well as the efficiency of calcium antagonists in its treatment.

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