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Case report

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Prinzmetal angina

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Learning Point for Clinicians

Prinzmetal angina is characterized by recurrent angina symptoms at rest and transient ST-segment changes. Prinzmetal angina can be a life-threatening condition with complications reaching from malignant arrhythmia, high degree AV-block to death. Therefore, prompt recognition of Prinzmetal angina is essential to avoid complications and provide appropriate management to improve outcome.

Case report

A 67-year-old female patient and former smoker presented to the emergency room (ER) with severe onset of chest pain. Admission ECG demonstrated ST-segment elevation in the inferior leads (Figure 1a). The patient reported to have chest pain the last couple of days associated with dyspnea. Her past medical history revealed familial adenomatous polyposis since 4 years and she has had a whipple surgery. Furthermore she had polyarthritis and esophageal spasm. For the latter she had been taken nitroglycerin if required because she did not respond to calcium-channel blockers. This time chest pain was completely different in character compared to the esophageal spasm that she had experienced in the past and she had never felt dyspnea and dizziness before for which reason she presented in the ER.

In the ER symptoms spontaneously resolved after some minutes and ST-segment elevation slightly disappeared in a repeated ECG. The patient was immediately brought to cath lab because of suspicion of myocardial infarction. Interestingly, emergency coronary angiography imposed narrowing of all epicardial vessels (Figure 1b) while the patient experienced chest pain and transient ST-segment elevation. After application of nitroglycerin intravenously impressive vasodilation of all vessels was observed (Figure 1c). Cardiac enzymes, which were available after cath, showed troponin, CK and CK-MB within the normal range. NTpro-BNP was slightly elevated (317 ng/l). Notably, the patient had consulted her general practitioner 2 weeks before her presentation in the emergency department, because of dizziness, pre-syncope and chest pain symptoms. A 24-h Holter-ECG monitoring had demonstrated intermittent AV-block-III. She was already scheduled for an appointment for a pacemaker (PM) implantation, which was then performed, during her current hospitalization.

This clinical picture is consistent with Prinzmetal angina complicated by AV-block. The patient was treated with a calcium-channel blocker and nitroglycerin was continued. Furthermore fluvastatin was added due to potential beneficial effects on endothelial function and reduction of coronary spasm. Repeated counseling and 1-year follow-up revealed no more angina symptoms.

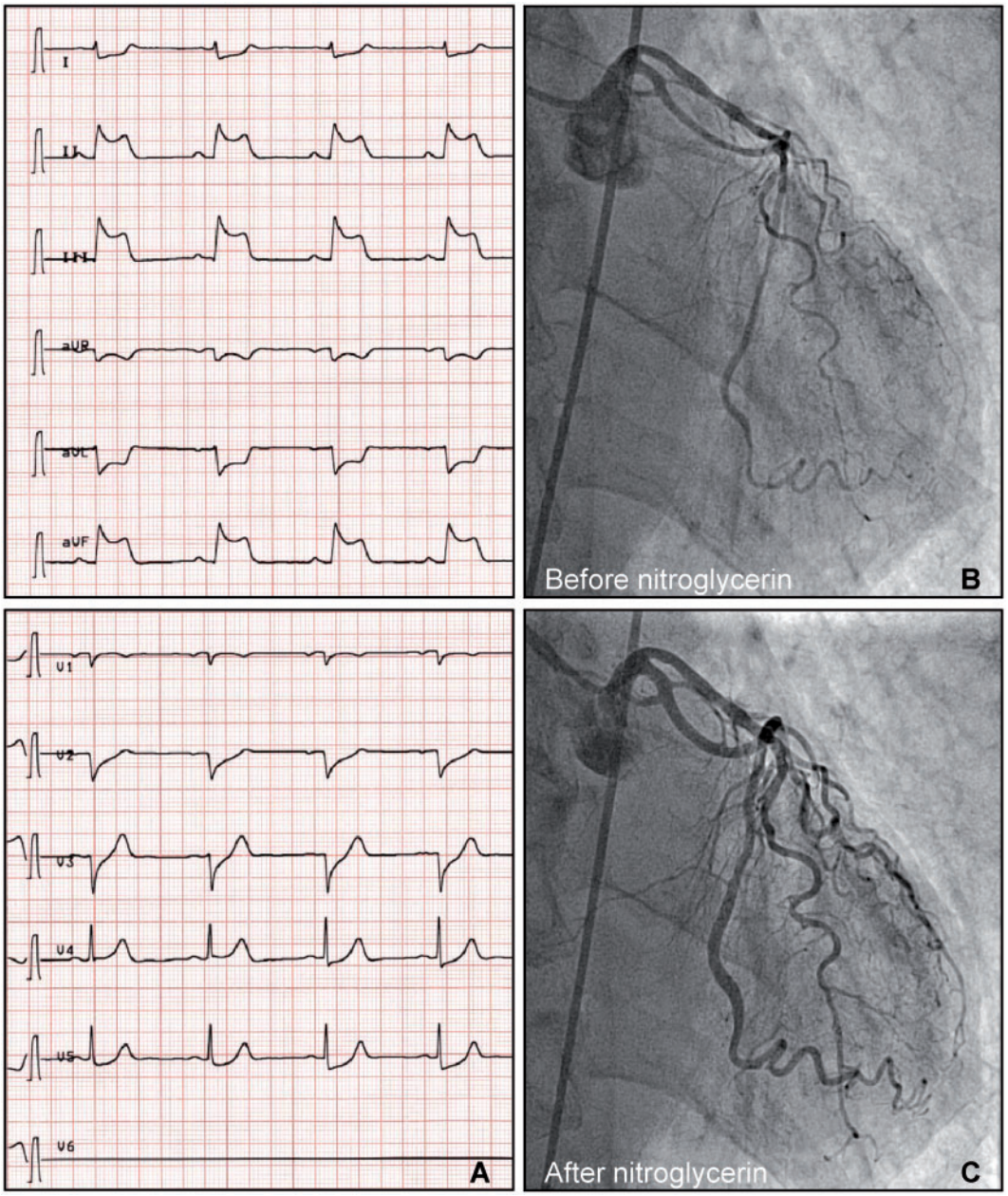

Figure 1. ECG demonstrated ST-segment elevation in the inferior leads (**A**), Emergency coronary angiography imposed narrowing of all epicardial vessels (**B**) while the patient experienced chest pain and transient ST-segment elevation. After application of nitroglycerin intravenously vasodilation of all vessels was observed (**C**).

Discussion

Here we describe a case of Prinzmetal angina complicated by advanced degree AV-block. The first case of Prinzmetal angina was described in 1959 by Prinzmetal.[1] Since then, several trigger factors have been reported to be associated with Prinzmetal angina these include, illict drugs such as cocaine, amphetamine or marijuana, but also bitter-orange, alcohol, butane, chemotherapy drugs, over-the-counter medication and different antibiotics. However, vasospastic angina can also occur without any triggering factor.[2] The classical symptom is recurrent angina at rest with spontaneous remission. A circadian pattern has been noted and Prinzmetal angina preferentially occurs in the morning hours. Complications comprise myocardial infarction, malignant arrhythmia and even sudden cardiac arrest or death. A complete AV-block can also result in Stokes–Adam-attacks and patients should be then treated with PM. Conflicting results exists on the pathophysiology, proposed mechanisms responsible for this disease entity are hyperactivity of the sympathetic nervous system and vagal withdrawal or reduced

nitric oxide synthase and endothelial dysfunction. While Egashira *et al.*[3] demonstrated enhanced Phopholipase C enzyme activity resulting in vasospastic angina without impairment of nitric oxide synthase. A genetic predisposition has also been discussed.[4] Therapeutic management consists of calcium-channel blockers and long-term nitrates with their vasodilatory effects. Furthermore Fluvastatin demonstrated a positive effect on endothelial function and can be therefore recommended.[5] In patients with life-threatening arrhythmia an ICD should be considered.[6]

Conflict of interest: None declared.

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