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Case Report

Recurrent acute coronary syndrome due to coronary artery spasm in different coronary arteries

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ABSTRACT

Coronary artery spasm (CAS) plays an important role in the pathogenesis of a wide variety of ischemic heart diseases. Several case reports have shown that CAS due to hypocalcemia can mimic myocardial infarction. However, in all these cases there were coexisting electrolyte abnormalities or other medical conditions that are risk factors for coronary spasm. We describe recurrent acute coronary syndrome in an otherwise healthy young woman without cardiac risk factors caused by coronary spasms, alternatively occurring in all three major coronary arteries.

<**Learning objective:** Isolated hypocalcemia may cause acute coronary artery spasm, leading to recurrent acute coronary syndrome. This unusual cause of recurrent acute coronary syndrome occurred in a young, otherwise healthy female with no risk factors for coronary artery diseases. Medical treatment would resolve the spasm in the large majority of the patients. However, in patients having medically resistant vasospasm, mechanical revascularization may be an option.>

Introduction

About 2% of patients admitted with acute myocardial infarction have no obstructive epicardial coronary artery disease (CAD). In these patients, thromboembolism, perimyocarditis, or coronary artery spasm may play a role in the etiology of myocardial damage. Coronary artery spasm (CAS) plays an important role in the pathogenesis of a wide variety of ischemic heart diseases, not only in variant angina but also in unstable angina, myocardial infarction, ventricular arrhythmias, and sudden death [1–4]. We describe a 42-year-old woman with recurrent acute coronary syndrome due to CAS in different coronary arteries.

Case report

A 42-year-old woman was referred to our hospital because of non-ST elevation myocardial infarction (non-STEMI). She had a continuous chest pain since the day before presentation. She had a family history of heart disease, she did not smoke and had no other risk factors for CAD. On admission, the blood pressure was 110/60 mmHg, heart rate 78 beats per minute, and the respiration rate was 12 min^{-1}. The oxygen saturation was 97%. Further physical examination revealed no abnormalities. The initial electrocardiogram (ECG) (Fig. 1A) showed a sinus rhythm 78 bpm and negative T wave in the leads II, III, aVF, and V3 to V6. In addition, the QTc interval on admission as calculated using Bazett's correction (in which the raw interval from beginning of QRS complex to the apex of the T wave is divided by the square root of the R–R interval) was 490 ms (normal range for female <470 ms).

Cardiac catheterization was performed and revealed a normal right (RCA) and circumflex coronary artery (CX). However, narrowing of a long part of the mid and distal left descending artery (LAD) was visible with thrombolysis in myocardial infarction (TIMI) 3 flow (Fig. 2A). Because of adequate flow and relief of chest pain, medical therapy was initiated with intravenous tirofiban during 48 h, subcutaneous heparin, acetylsalicylic acid, clopidogrel, metoprolol, and a statin followed by repeat coronary angiography (CAG) a week later. Cardiac enzymes were only slightly elevated [creatinine kinase (CK) 232 U/L, CK-MB 32 U/L]. CAG one week later showed a normal aspect of the coronary arteries, including the LAD.

Ten months later the patient was re-admitted to our hospital because of unstable angina pectoris. CAG was performed and showed a normal LAD (including the mid and distal part which was narrowed on the previous study) and RCA, but now coronary spasm in the mid CX was visible (Fig. 2B). A calcium antagonist (diltiazem XR 200 mg once daily) was added to her medication and the patient was transferred back to her own hospital. However, two days later she was again referred because of acute chest pain and electrocardiographic signs of inferior myocardial infarction (Fig. 1B). During transportation in the ambulance two episodes of

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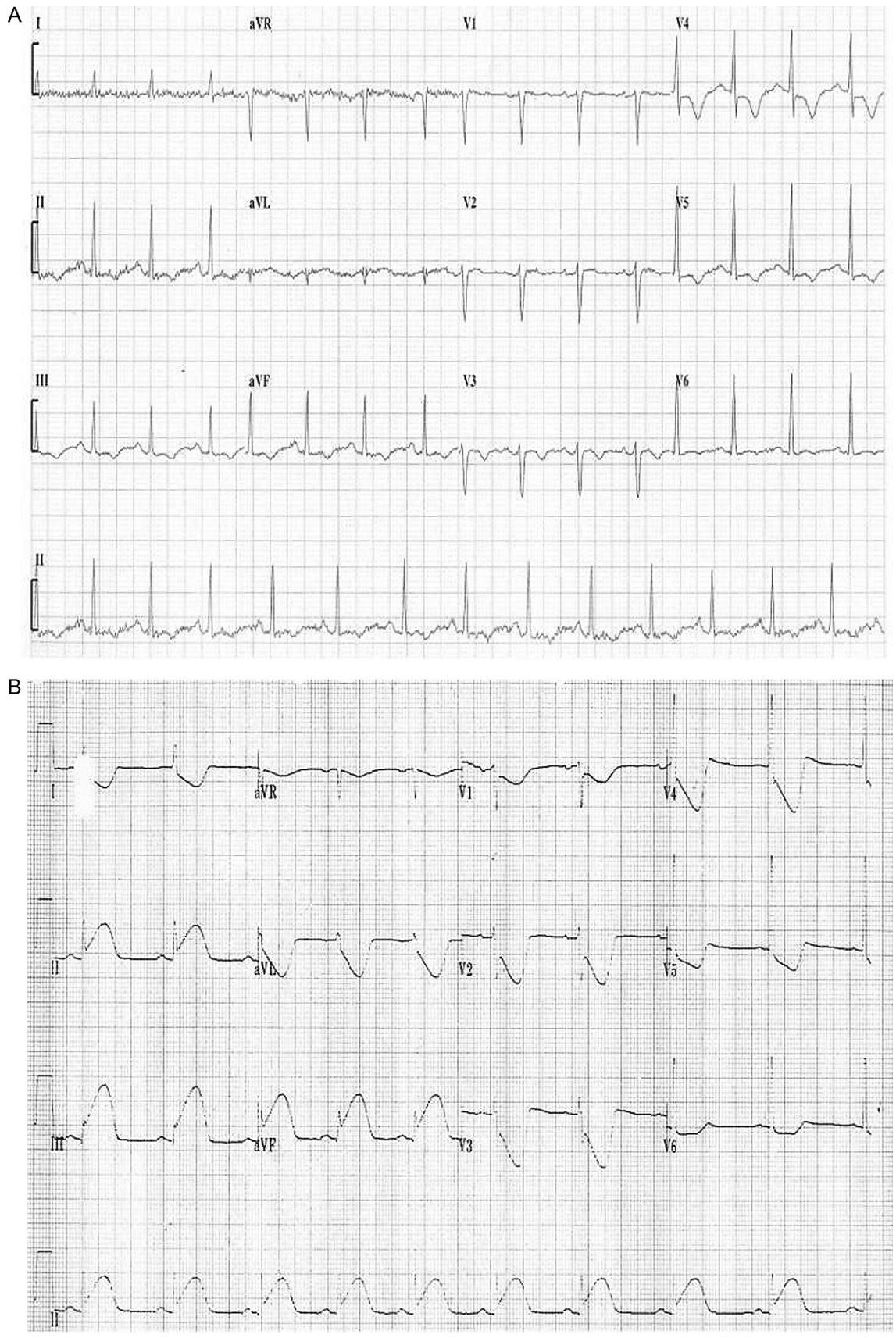

Fig. 1. (A) Sinus rhythm 78 bpm, normal axis, normal PR and QRS duration, QTc 490 ms, negative T-waves in leads II, III, aVF, and V3 to V6. (B) Sinus rhythm 62 bpm, normal axis, normal PR and QRS duration, marked ST elevation in leads II, III, aVF, ST segment depression in leads I, aVL, and V1 to V6.