Uncommon cause of complicated myocardial infarction with normal coronary arteries in a Saudi patient

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A case of a young Saudi patient with a previous diagnosis of bronchial asthma, nasal polyps, and chronic smoker, presented with atypical chest pain, elevated serum troponin and borderline ischemic electrocardiogram (ECG) changes, with no significant regional wall motion abnormalities at bedside echocardiography is reported. The patient was admitted to the coronary care unit for continuous monitoring as possible acute coronary syndrome, non-ST elevation myocardial infarction (STEMI). One hour after admission, the patient had ventricular fibrillation (VF) cardiac arrest that required three DC shocks and amiodarone bolus before returning of spontaneous circulation, which followed the fourth shock. The resuscitation took 15 minutes of cardiopulmonary resuscitation (CPR). An immediate 12-leads ECG showed significant ST elevation in precordial leads that mandate an urgent coronary angiogram that revealed patent coronary arteries, therefore spasm of normal coronary arteries was postulated as the operative factor. The cardiac magnetic resonance image (MRI) showed a picture of transmural anterior myocardial infarction, which correlates with the follow up echocardiogram reporting hypokinetic anterior wall. A complete history was taken and no use of illicit drugs or alcohol was found. The unusual presentation in such a patient with evidence of extensive anterior STEMI and normal coronary arteries raise the thought of considering uncommon causes. In view of previous medical history and laboratory evidence of eosinophilia, Kounis syndrome was considered dominant in the differential diagnosis.

Keywords: Coronary, Myocardial, infarction, Kounis

1. Introduction

Myocardial infarction with normal coronary arteries angiography (MINCA) is a life-threatening event with many open questions for physicians and patients [1]. MINCA is common with a prevalence of 1–12% of all myocardial infarctions. The pathogenic mechanisms of MINCA are still unknown, but endothelial dysfunction has been suggested as a possible cause [2].

A history of inflammatory disease was more common in patients with MINCA than in healthy controls. Hypercoagulability due to inflammation

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has been also reported as a possible reason for MINCA. There are some case reports with patients presenting with MINCA during exacerbation of systemic inflammatory disease without evidence of coronary vasculitis, whereas others have identified a secondary myocarditis. The condition of inflammation and hypercoagulability needs to be elaborated in future studies of MINCA [2].

Our case of a 32-year-old Saudi male soldier presented with a history of 10 days atypical chest pain with increased intensity over the past 7 hours. The patient's medical history included nasal polyps, bronchial asthma for 10 years on bronchodilators and cigarette smoker (smoking 10 cigarettes/d for 14 years). There was no other significant medical history with irrelevant family history and no history of sudden cardiac death. The patient was hemodynamically stable with normal first and second heart sounds on clinical examination. An electrocardiogram (ECG) on presentation showed borderline ST segment elevation of <1 mm in the inferior leads (Fig. 1). Initial transthoracic echocardiography in the emergency room showed no significant regional wall motion abnormalities (RWMA) with normal left ventricular size and ejection fraction of >55%. The first set of serum troponin was elevated (746 pg/mL, normal troponin <34.2 pg/mL). Normal lipid profile, toxicology screen was sent and chest radiograph showed no abnormality (Fig. 2).

The patient was admitted to the coronary care unit for clinical management as possible non-ST elevation myocardial infarction, started on guidelines directed anti-ischemic measures.

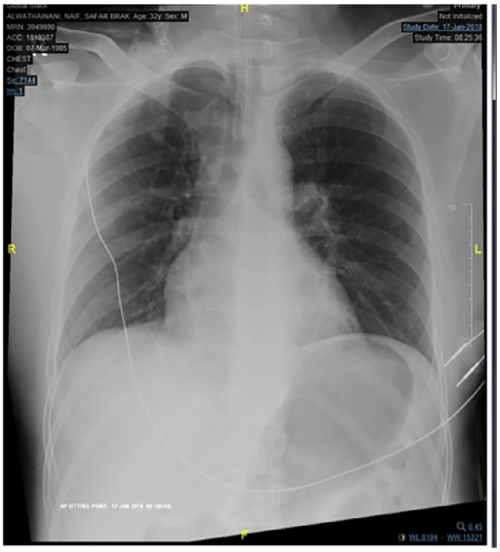

Figure 2. Chest X ray: reported normal.

One hour later the patient suddenly had ventricular fibrillation cardiac arrest (Fig. 3) with immediate cardiopulmonary resuscitation (CPR) (according to American resuscitation guidelines) was commenced, three DC shocks and epinephrine, which did not give any response. Following the fourth shock and amiodarone bolus (150 mg IV), he returned to spontaneous circulation within a total of 15 minutes CPR. The patient was intubated and mechanically ventilated with use

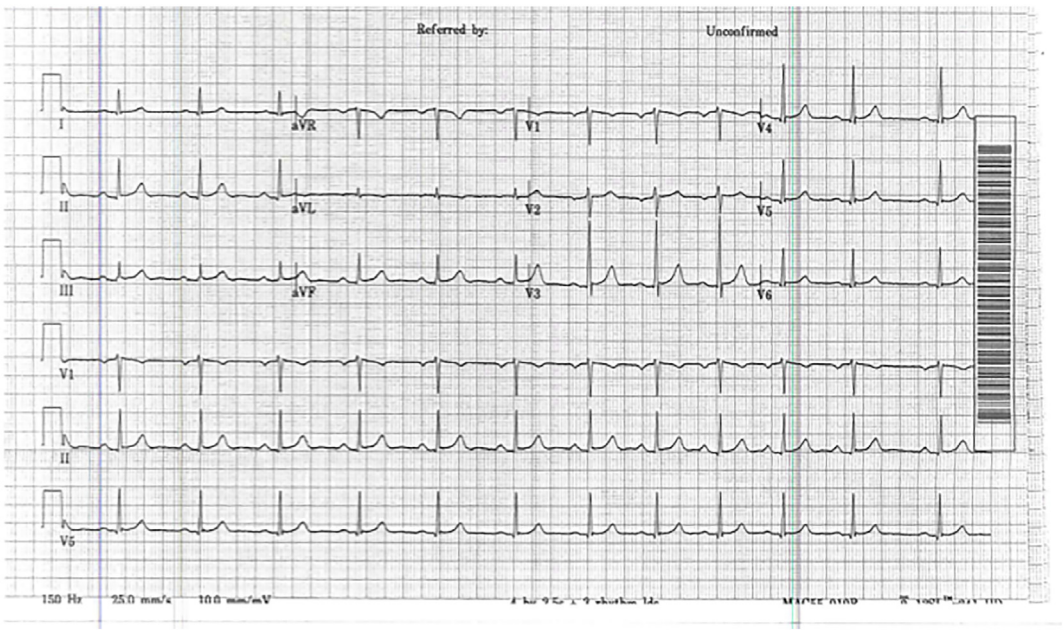

Figure 1. ECG: normal sinus rhythm with <1 mm ST elevation in the inferior leads. ECG = electrocardiogram.