



Kasuistika | Case report

Non-ST-segment elevation myocardial infarction without culprit lesion – Role of coronary artery spasm

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A 68-year-old white woman was referred to our hospital by her general practitioner, with symptoms of acute coronary ischemia: she reported severe chest pain at rest that had started 12 hours ago accompanied by palpitations. She has been suffering from angina on exertion as well as at rest for several months. Her cardiovascular risk factors were arterial hypertension, hypercholesterolemia (low density lipoprotein [LDL] of 4.1 mmol/l [74 mg/dl] under treatment with simvastatin 30 mg o.d.) as well as a positive family history for cardiovascular disease (her

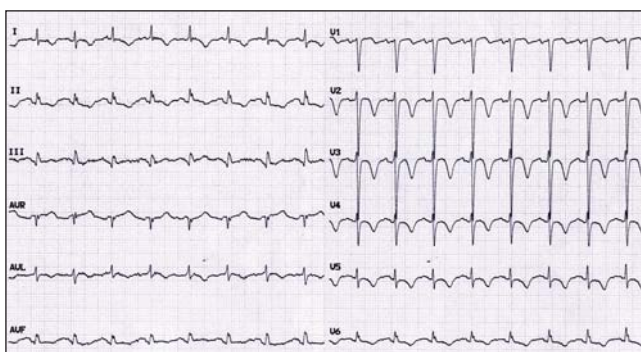


Fig. 1 – ECG on admission showing sinus rhythm with a heart rate of 104 bpm and negative T-waves in lead I, II, aVL, V₂–V₆.

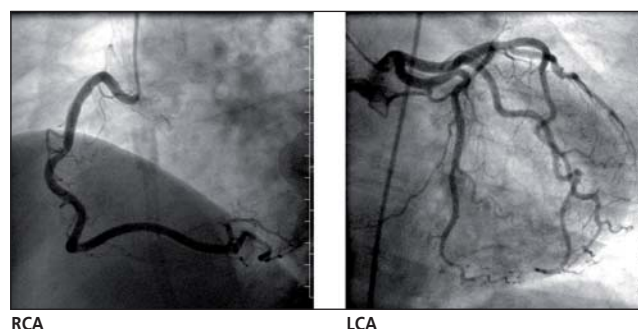


Fig. 2 – Coronary angiography of the right coronary artery (left) and the left coronary artery (right) showing no flow-limiting epicardial stenosis.

mother had suffered from angina pectoris, her grandfather had a myocardial infarction at the age of 76). In addition, she had been a cigarette smoker until 1999. The initial electrocardiogram (ECG) showed negative T-waves in leads I, II, aVL and V₂–V₆ (see Fig. 1). Laboratory analysis showed an elevation of high-sensitive cardiac troponin of 27 pg/ml ($n < 14$ pg/ml), the creatinkinase (CK) was normal (i.e. < 180 U/l).

A diagnosis of non-ST-segment elevation myocardial infarction was made and the patient underwent emergent coronary angiography. Surprisingly, coronary angiography did not reveal any relevant epicardial stenosis (see Fig. 2). Left ventricular angiography showed normal

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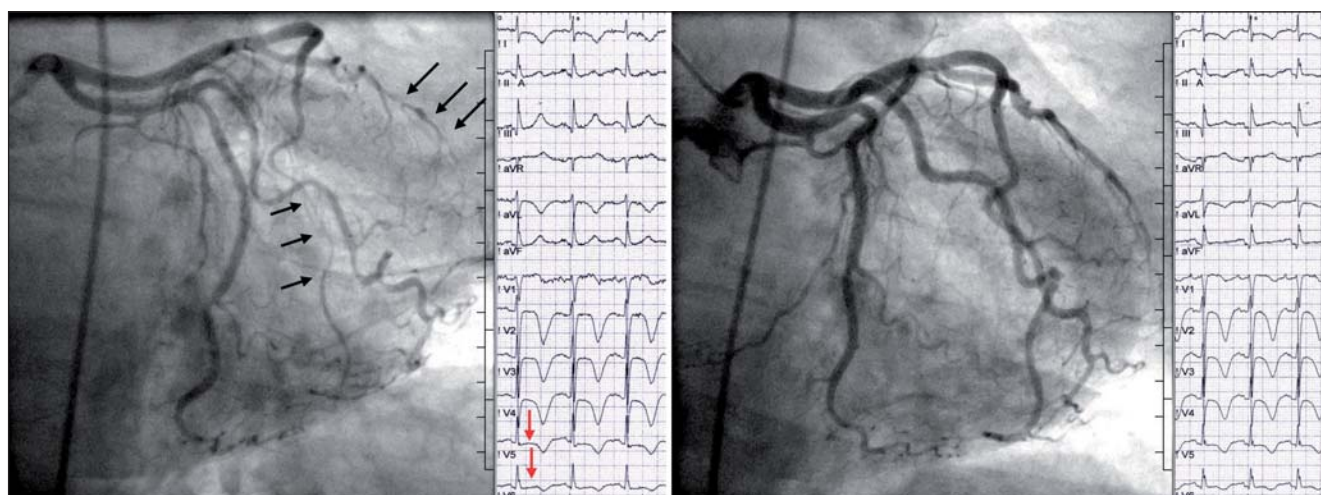


Enddiastolic view

Endsystolic view

Fig. 3 – Left ventriculography with normal systolic left ventricular function and no regional wall motion abnormalities (enddiastolic view on the left and endsystolic view on the right). Although the clinical history and the mild troponin elevation as well as the deep negative T-waves suggest takotsubo cardiomyopathy, the normal LV angiogram is not compatible with this diagnosis.

artery the patient experienced the same angina that had led to admission and the 12-lead ECG showed additional ST-segment depression in leads I, aVL and V₅–V₆. After 200 µg of ACH the patient had full reproduction of the symptoms leading to admission and coronary angiography revealed epicardial coronary spasm of the distal left anterior descending artery and the first diagonal branch (see Fig. 4). The ST-segment depression was unchanged but the negative T-waves became deeper (Fig. 4). After intracoronary nitroglycerine injection, the symptoms as well as the epicardial spasm resolved. The patient was treated with amlodipine and molsidomine. Under this treatment, the ECG abnormalities almost disappeared only a few days after the acute event (see Fig. 5). Only the negative T-waves in I and aVL remained. Two months later the patient reported a marked improvement of her symptoms.



LCA after 200 µg ACH i.c.

LCA after 200 µg nitroglycerine

Fig. 4 – Left coronary artery and 12-lead ECG after injection of 200 µg acetylcholine (left) showing epicardial coronary spasm of the distal left anterior descending artery and the first diagonal branch, accompanied by ST-segment depression and deepening of the negative T-waves in leads I, aVL, V₅–V₆. The patient had full reproduction of her symptoms that led to admission. After intracoronary nitroglycerine injection (right) the symptoms as well as the epicardial spasm resolved and the ischemic ECG shifts dwindled.

left ventricular function without wall motion abnormalities (see Fig. 3). To further investigate the cause of the patient's symptoms, intracoronary acetylcholine (ACH) provocation testing with simultaneous 12-lead ECG registration was performed as reported previously [1]. After the injection of 100 µg acetylcholine in the left coronary

This case illustrates the causative role of functional coronary artery abnormalities as the potential cause of an acute coronary syndrome (ACS) without culprit lesion. Coronary artery spasm has been shown to be a frequent cause of acute coronary syndrome. About every fourth patient with ACS has no culprit lesion upon coronary

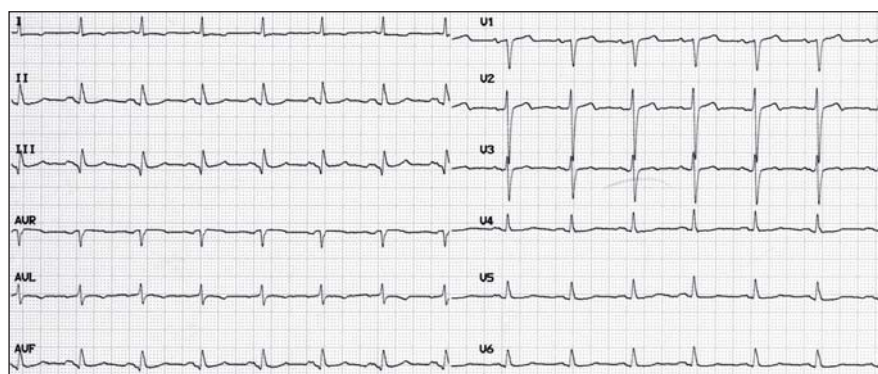


Fig. 5 – 12-lead resting ECG a few days after the acute event. The ECG abnormalities almost disappeared under treatment with amlodipine and molsidomine, only slight negative T-waves in I and aVL remained.

angiography [2]. In ACS patients without culprit lesion who underwent ACH provocation testing, nearly 50% had coronary spasm as the cause of the ACS [1]. Therefore it is important to consider coronary artery spasm as a differential diagnosis in patients with acute coronary syndrome. Interestingly, the negative T-waves in I and especially in aVL are often seen in patients with symptoms pointing to the presence of coronary vasospasm. The clinical presentation with angina pectoris on exertion as well as at rest is often seen in patients with coronary vasomotor disorders pointing towards a concomitant dysfunction of the coronary microcirculation as previously shown by our group [3]. In the present case this is supported by the fact that symptoms and ECG changes preceded the angiographically documented vasospasm.

Fortunately, the prognosis for patients with ACS due to coronary spasm is rather good if they are treated with calcium channel blockers and nitrates: mortality and coronary events are significantly lower after 3 years compared to patients with obstructive ACS [4]. However, persistent angina, which can lead to repeated coronary angiography is a frequent problem in these patients leading to impaired quality of life and prognosis may be worse than in patients with normal coronary arteries but without angina [5].

Conflict of interest

The authors declare that there is no conflict of interest in conjunction with this manuscript.

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Ethical statement

The authors declare that the research was done according to ethical standards.

Informed consent

The authors declare that the patient agreed to participate in the research.

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