

## Headache: An unusual presentation of acute myocardial infarction

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### Abstract

Acute myocardial infarction should be diagnosed as early as possible for the appropriate management to salvage ischemic myocardium. Accurate diagnosis is typically based on the typical symptoms of angina. Headache is an unusual symptom in patients with acute myocardial infarction. We report a patient with ST-segment elevation acute myocardial infarction who presented to the emergency department complaining of severe occipital headache without chest discomfort.

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**Key words:** Headache; Angina; Myocardial infarction

**Core tip:** The association of headache with myocardial ischemia is unusual and is accompanied by chest discomfort. The only symptom of this patient was occipital headache and this is extremely rare. Owing to the rare occurrence of headache as a symptom of myocardial ischemia, diagnosis may be extremely difficult since a brain computed tomography imaging is important to

rule out the possibility of hemorrhage.

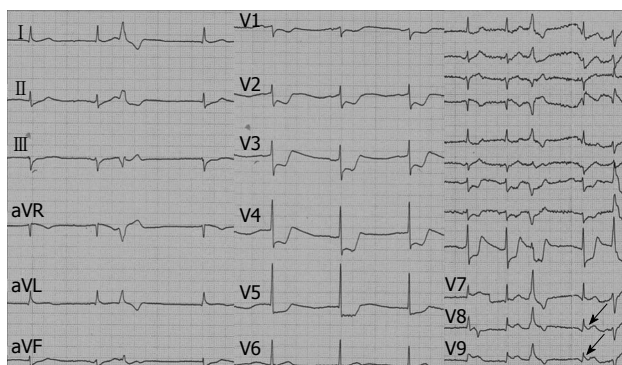
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### INTRODUCTION

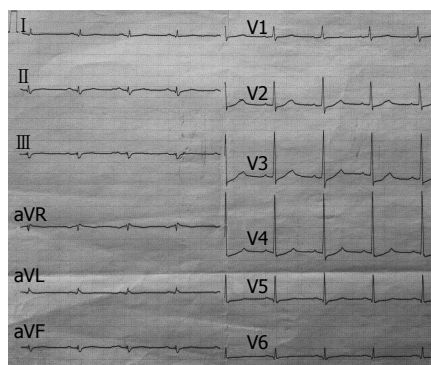
Atypical symptoms of myocardial infarction may delay the diagnosis, and therefore the proper management to rescue ischemic myocardium. Headache represents a rare symptom of myocardial ischemia<sup>[1-5]</sup>. We report a patient with ST-segment elevation acute myocardial infarction who presented to the emergency department complaining of headache without chest discomfort.

### CASE REPORT

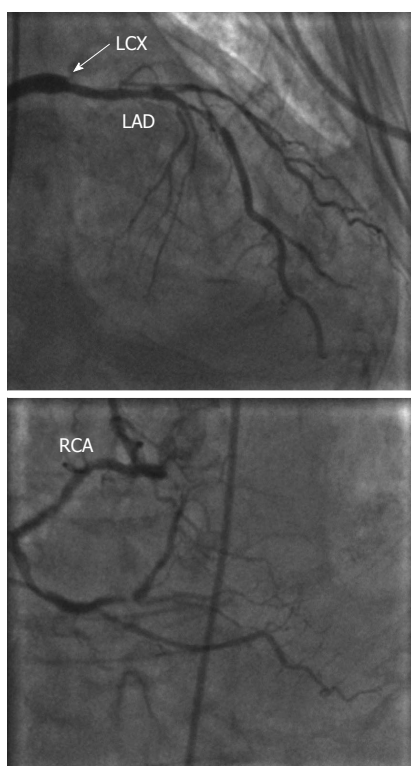
An 86-year-old man with a history of hypertension and tobacco use presented to the emergency department complaining of recent onset severe occipital headache. The patient did not report any chest pain, dyspnea, or other typical symptoms of angina. On admission the patient was pale with tachycardia (100 beats/min), and, while his blood pressure was within normal range (100/60 mmHg). At auscultation, a mild systolic murmur was audible. The electrocardiogram (ECG) showed sinus bradycardia, ST-segment depression in leads V1-V5 and ST-segment elevation in posterior leads (V7-V9) (Figure 1). Transthoracic echocardiography revealed an impaired left ventricular ejection fraction (40%-45%) along with mild mitral valve regurgitation. Initial laboratory examinations showed elevated levels of high-sensitivity cardiac troponin T (250 ng/L). Due to his clinical presentation, a brain computed tomography (CT) imaging was im-



**Figure 1** Electrocardiogram on admission demonstrating ST-segment depression in leads V1-V5 and ST-segment elevation in the posterior leads (V7-V9) (arrows).



**Figure 3** Electrocardiogram demonstrating resolution of the ST-segment depression in leads V1-V5 after revascularization.



**Figure 2** Coronary angiography showing total obstruction of the proximal left circumflex artery (arrow) and severe stenosis in left anterior descending artery and right coronary artery. LCX: Left circumflex artery; LAD: Left anterior descending artery; RCA: Right coronary artery.

mediately performed. The CT imaging was negative for intracerebral or subarachnoid hemorrhage. Following CT imaging, the patient prepared for cardiac catheterization and received aspirin (500 mg), clopidogrel (600 mg) and unfractionated heparin (70 U/kg). Coronary angiography was performed 60 min after admission and demonstrated a three-vessel coronary artery disease [the proximal left circumflex artery (LCX) was totally obstructed, the left anterior descending artery (LAD) displayed a severe stenosis and the right coronary artery was also severely diseased] (Figure 2). Proximal LAD lesion was directly stented, while the blood flow was restored in LCX artery revealing a severe stenosis of more than

90%. We attempted to insert the guidewire into the LCX but failed to cross the proximal part of LCX. Following revascularization, the patient was totally asymptomatic without headache, while the ECG was normalized (Figure 3). During the following days, the myocardial enzymes (CK-MB, hs-troponin T) followed the classic rise and fall kinetic pattern. He discharged 6 d later under dual anti-platelet (aspirin, clopidogrel),  $\beta$ -blocker and angiotensin converting enzyme inhibitor therapy.

## DISCUSSION

Myocardial infarction should be diagnosed as early as possible for the appropriate management to salvage ischemic myocardium. Accurate diagnosis is based on both ECG and clinical presentation of the patient. Ischemia and myocardial infarction typically causes chest pain variously radiating elsewhere (shoulders, upper extremities and epigastrium). The association of headaches with myocardial ischemia is unusual and is accompanied by chest discomfort. The only symptom of this patient was occipital headache and this is extremely rare. Owing to the rare occurrence of headache as a symptom of myocardial ischemia, diagnosis may be extremely difficult since a brain CT imaging is important to rule out the possibility of hemorrhage.

The incidence of headache as a symptom of myocardial ischemia may be underestimated<sup>[1-5]</sup>. Culić *et al*<sup>[6]</sup> reported that headache is present (along with other symptoms) in 5.2% of patients with acute myocardial infarction. Moreover, in 3.4% of these patients headache was the primary complaint<sup>[6]</sup>. Cardiac cephalalgia or headache angina is a recognized phenomenon, but the pathophysiological mechanism is still unclear<sup>[7-8]</sup>. There is a connection between the central cardiac pathway and the cranial pain afferents. The cardiac sympathetic fibers originate from cervical lymph nodes which also innervate pain sensitive cranial structures<sup>[9-10]</sup>. Furthermore, it is hypothesized that chemical mediators like bradykinin, serotonin and histamine can induce pain in shoulders, arms, neck and in this case headache. Another mechanism is based on the elevated intracranial pressure associated in the case of decreased cardiac output during myocardial

infarction and elevated venous pressure<sup>[11,12]</sup>. Finally, increased levels of atrial and brain natriuretic peptides may be involved in intracranial pressure regulation<sup>[13]</sup>. Even though the occurrence of headache as a sole manifestation of angina or myocardial infarction has been previously described, many clinicians ignore this unusual manifestation. The diagnosis of “cardiac headache” is difficult and requires a high degree of suspicion.

## COMMENTS

### Case characteristics

An 86-year-old man presented to the emergency department complaining of recent onset severe occipital headache.

### Clinical diagnosis

The patient was pale with tachycardia and electrocardiogram (ECG) signs suggestive of myocardial infarction.

### Differential diagnosis

The differential diagnosis included intracerebral or subarachnoid hemorrhage and myocardial infarction.

### Laboratory diagnosis

Elevated levels of high-sensitivity cardiac troponin T were initially recorded.

### Imaging diagnosis

Brain computed tomography imaging excluded intracerebral or subarachnoid hemorrhage, while coronary angiography demonstrated a three-vessel coronary artery disease.

### Treatment

Proximal left anterior descending artery lesion was directly stented, while the blood flow was restored in left circumflex artery revealing a severe stenosis of more than 90%.

### Experiences and lessons

Careful ECG interpretation in the setting of acute headache is of major importance.

### Peer review

Asvestas *et al* report a rare case of a patient who presented with headache as the sole symptom of an acute myocardial infarction. The mechanisms by which headache is linked to ischemic vascular disease remain uncertain and are likely to be complex. The paper is generally well-written and interesting.

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