Case Reports

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Sensation of laryngeal obstruction as a manifestation of myocardial infarction: A case report

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Abstract

A rare case of a 62-year-old woman with an atypical cardiac symptom of sensation of laryngeal obstruction as a manifestation of acute coronary syndrome is described. Initially, the patient showed unremarkable test results and was diagnosed with laryngopharyngitis and discharged from hospital. However, 24 hours later she returned to the hospital with an abnormal electrocardiogram (ECG) and elevated blood troponin levels and was diagnosed with ST-segment elevation myocardial infarction (STEMI). She developed heart failure, cardiogenic shock and died. Clinicians should be aware that patients with an unexplained sensation of laryngeal obstruction should be considered for the presence of MI within their differential diagnosis since this may be the only symptom in some patients with life-threatening cardiac ischemia.

Keywords

myocardial infarction, laryngeal obstructed sensation, acute coronary syndrome

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Introduction

Acute coronary syndrome (ACS) is a common cardiac emergency with the potential for substantial morbidity and mortality.[1] ACS encompasses a spectrum of heart diseases that include ST-segment elevation myocardial infarction (STEMI), non-ST elevation myocardial infarction (NSTEMI)

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and unstable angina.[2] 'Pressure-like' retrosternal chest pain is a typical symptom of ACS.[2] Sometimes in the absence of chest pain, less common symptoms may include vomiting, nausea, sweating, neck/jaw/arm pain.[2] Misdiagnosis in life-threatening cases can lead to delayed and/or inappropriate treatment and increased mortality.[3] Herein, we report a rare case of acute myocardial infarction (MI) where the sensation of laryngeal obstruction was the only symptom on initial presentation.

Case report

A 62-year-old woman was admitted to the emergency department of our hospital complaining of a 24-hour history of a sensation of laryngeal obstruction. The patient did not have any chest pain, shortness of breath, sore throat, nausea, vomiting, heartburn, diaphoresis, fevers, or chills. She could not recall an obvious cause of her upper airways problem. She did not drink or alcohol or smoke cigarettes. Physical examination on presentation showed: body mass index (BMI), 25-kg/m^2; temperature, 35.9°C; respiration rate (RR), 16 breaths/min; oxygen saturation, 98%; blood pressure (BP), 117/80 mmHg; pulse rate (PR), 73 beats/min. A cardiovascular examination showed normal rate and rhythm with no murmurs or friction rub. A laryngopharyngeal examination was unremarkable with no evidence of discharge, hyperaemia or swelling and vocal cord function appeared normal. The patient's medical history was insignificant apart from a left nodular goitre excision she had undergone two years previously. A routine electrocardiogram (ECG) showed sinus rhythm and a chest computed tomography (CT) scan showed a small inflammatory lesion in the lower lobe of the right lung and confirmed the absence of an intra/extra-thoracic goitre. Her troponin I levels were <0.025 ng/ml (normal range <0.1 ng/ml) and her blood lipids were within normal range (triglycerides, 1.16 mmol/l [normal 0.00–1.70 mmol/l]; total cholesterol, 5.40 mmol/l [normal 3.00–5.70 mmol/l]; low density lipoprotein, 2.39 mmol/l [normal 1.89–3.37 mmol/L]). Therefore, because the patient exhibited no risk factors or evidence of ACS, a presumptive diagnosis of laryngopharyngitis was made and she was discharged from hospital with symptom-alleviating medication.

The patient returned to the emergency department 24 hours later. Her main complaint was that of a continuous sensation of laryngeal obstruction with accompanying mild fatigue. She reported that she had no chest pain or shortness of breath. Her vital signs were: BP, 104/77 mmHg, PR, 80 beats/min; RR, 15 breaths/min; oxygen saturation, 97%. A routine 12-lead ECG showed an anterior wall MI with ST segment elevation in leads V1-V4 (Figure 1). Bedside ECG showed a dramatically decreased ejection fraction of 35% with wall motion abnormalities. Laboratory data showed elevated levels of: troponin I, 13.2 ng/ml; B-type natriuretic peptide, 259 pg/ml (normal $\leq$100 pg/ml); D- Dimer, $>$3680 ng/ml (normal 0–243 ng/ml); creatine kinase, 2043 U/l (normal $<$140 U/l). On the basis of elevated troponin levels combined with ECG changes, her diagnosis was amended to acute anterior wall MI. After a MI protocol was initiated, a cardiology consultation was requested. However, after 10 minutes, her BP fell progressively to a minimum of 60/40 mmHg and she became unconscious. Ventricular fibrillation (VF) was detected in the ECG monitor and so cardiopulmonary resuscitation with advanced life support was initiated immediately. In spite of high doses of catecholamines and unsynchronous direct current defibrillation, her haemodynamics deteriorated dramatically. Refractory VF occurred repeatedly and her cardiac rhythm and function could not be restored. Her family consented to an autopsy that showed total

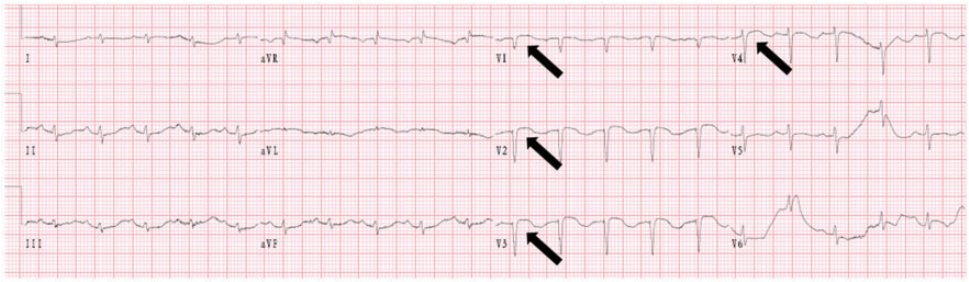

Figure 1. A routine 12-lead electrocardiogram (ECG) showed an anterior wall myocardial infarction with ST segment elevation (STEMI) in leads V1-V4 (arrows).

occlusion of the left anterior descending artery with thrombus below the proximal segment and 80% stenosis of the left circumflex artery middle part. Definitive histology using haematoxylin and eosin (H&E) staining confirmed anterior wall myocardial necrosis (Figure 2a and b), which was consistent with our diagnosis of acute anterior wall MI.

Written informed consent for the publication of this report was obtained from the patient's husband and the study was reviewed and approved by the local ethics committee of Ningbo First Hospital. This report adheres to CARE guidelines.[4]

Discussion

Typically, acute MI is characterized by chest pain, which often radiates out to the neck, shoulder, and jaw and down the left arm. However, in some instances cardiac pain may be experienced in regions outside the thorax such as the jaw, arms, neck, and epigastrium.[5,6] Because its clinical manifestations vary considerably, symptom recognition in the context of ACS is a challenging task. Moreover, previous studies have reported that up to 33% of patients with ACS present with atypical symptoms.[7,8] Furthermore, research has shown that the general population as well as many clinicians have insufficient knowledge of the atypical symptoms of AMI.[9] One study

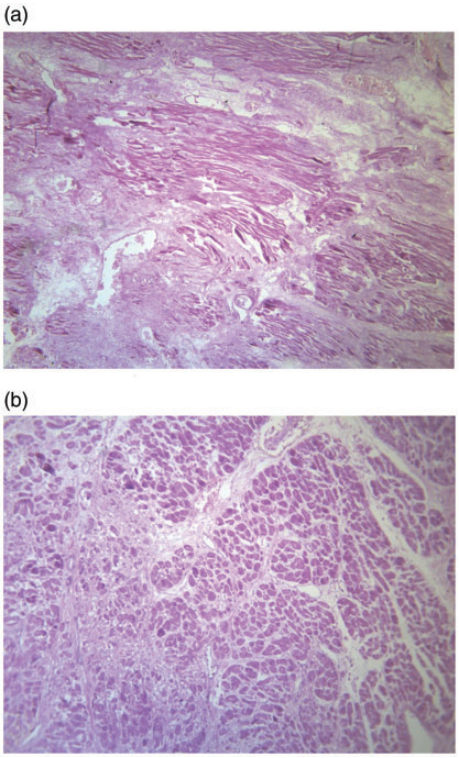

Figure 2a. Histopathological appearance showing necrosis of the myocardial cells. Myocardial fibres were wavy and sarcoplasmic aggregation formed red transverse bands of varying thickness. Haematoxylin and eosin (H&E) stain with 5× magnification.
2b. Cross section of myocardial cells in the infarct area shows sarcoplasmic aggregation (stained red). Myocardial cells collapse into irregular coarse granules or masses, with nuclear concentration and fragmentation. Haematoxylin and eosin (H&E) stain with 5× magnification.

that compared risk factors in patients with typical and atypical MI found that older patients and those with low cholesterol levels were more likely to present with atypical MI compared with younger patients.[10] In addition, another study reported that that old age (≥75 years), female sex, diabetes mellitus, history of acute MI, and absence of hyperlipidaemia were five independent predictors for atypical symptom presentation of ACS. Consistent with these findings, our patient was a 62-year-old female without hyperlipidaemia.

To our knowledge, the sensation of laryngeal obstruction as a manifestation of acute MI is rare. From our literature search, we only found one other report of a NSTEMI presenting as a sore throat with an obstructed sensation.[11] Similar to our report, the correct diagnosis of cardiac ischemia was missed and the patient was initially treated for primary laryngopharyngitis. Interestingly, the underlying pathophysiology of the sensation of laryngeal obstruction is thought to be related to the neuroanatomy of the nerves innervating the heart and larynx. These nerves enter the spinal tract nucleus near the medulla and their stimulation can cause a sensation of laryngeal obstruction based on the convergence-projection theory.[12] This theory suggests that the central nervous system cannot differentiate between stimuli that converge in a common sensory pathway. Therefore, the anatomic convergence of cardiac nerve fibres and somatic afferents from the larynx in central pathways is likely to be responsible for the perception of cardiac ischemic pain as a sensation of laryngeal obstruction.

Our report highlights the diagnostic challenge for clinicians and emergency room physicians. A delay in the diagnosis of ACS is associated with poor clinical outcome and increased mortality[3] Our patient developed heart failure and cardiogenic shock and died 24 hours after her initial presentation. Therefore, in patients with an unexplained sensation of laryngeal obstruction, the presence of MI should be considered within the differential diagnosis because this may be the only symptom in some patients with life-threatening cardiac ischemia.

Declaration of conflicting interests

The authors declare that there are no conflicts of interest.

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