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Case Report
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Heart Attack Causes Head-Ache – Cardiac Cephalalgia

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Chest pain is the typical symptom of myocardial infarction (MI), and there are many atypical manifestations such as stomachache or dyspnea. Headache is a rare presentation of MI, which has specifically been termed "cardiac cephalalgia" or "cardiac cephalgia". In this article, we have reported a case of sudden onset headache and neck pain, of whom MI was confirmed by electrocardiography, cardiac markers, and coronary angiogram. The patient's headache subsided dramatically after coronary angioplasty, and it had not recurred in the following one year. Additionally, diagnostic clues and possible mechanisms of cardiac cephalalgia are discussed as well.

Key Words: Headache • Cardiac cephalgia • Cardiac cephalalgia • Myocardial infarction

INTRODUCTION

Retrosternal chest pain with or without diaphoresis is the typical symptom of myocardial infarction (MI); atypical manifestations such as stomach ache or dyspnea are also well-recognized by clinicians. Headache, however, could be the sole[1] or concomitant[2] presentation of MI, which is otherwise termed "cardiac cephalagia".[3] In these cases, it can be quite challenging to make the diagnosis of MI, yet delayed diagnosis could lead to serious complications. Here, we reported a case of MI presented with the rare symptom of headache.

CASE REPORT

A 70-year-old woman with the past history of hypertension, type 2 diabetes mellitus, and hyperlipidemia, presented to the emergency department (ED) with sudden-onset headache and nuchal pain. She had suffered from chronic headache for several years, which could be relieved after rest and oral analgesics. Sometimes, her headaches could be precipitated by anger and stress. On the day this patient was hospitalized to our facility, her headache recurred in the afternoon, which was similar in character but with longer duration and intensity. She had visited a local medical clinic, where antihypertensive agent was prescribed for elevated blood pressure (180 mmHg in systole). However, her headache and nuchal pain worsened in 2 hours, so she visited the ED in this hospital.

The character of this patient's headache was dull and squeezing pain over the bilateral posterior nuchal area, radiating to both temporal regions. She also reported dizziness, but there was no chest pain, dyspnea, cold sweating, stomachache, nausea, vomiting, or photophobia. She was not a cigarette smoker and had no family history of cardiovascular disease. The patient had tachycardia (106 beat-per-minute) and mildly elevated blood pressure (159/80 mmHg). Her consciousness was clear, and there was no evidence of neurological deficits. The neck was supple, and the jugular vein was not engorged. Her breath sounds showed bilateral basal fine crackles without wheezes, and there was no obvious murmur and gallop in heart sounds. Furthermore, neither lower leg was edematous.

Because the patient's headache and nuchal pain persisted, some analgesics were prescribed for the

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headache. Computer tomography of the brain was considered for further evaluation. However, electrocardiography (ECG) was performed because the image study was not available immediately. Surprisingly, the 12-lead ECG showed ST-T segment elevation over lead V2-V6 (Figure 1A). Cardiac markers were also elevated: creatinine kinase (CK): 234 IU/L, MB subtype of CK (CK-MB): 30 IU/L, and troponin-I: 0.858 ng/ml. A chest X-ray revealed bilateral pulmonary congestion (Figure 2A). A cardiologist was consulted immediately, and transthoracic echocardiography revealed left ventricular regional wall motion abnormality of the left anterior descending artery territory with fair left ventricle contractility. Dual antiplatelet agents and unfractionated heparin were administered under the impression of ST-elevation MI. Because headache was such an unusual manifestation, the patient and her family declined emergent coronary angiogram. Therefore, she was admitted to the intensive care unit (ICU) for medical therapy.

In ICU, dual antiplatelet agents, unfractionated heparin, statin, beta-blocker, and angiotensin-converting enzyme inhibitor were prescribed as the recommendation of clinical guidelines. A subsequent ECG performed 2 hours later (Figure 1B) showed obvious precordial leads ST-T segment dynamic change. The patient had no chest pain, but her headache was still noted. The headache was located over her bilateral temporal area, and there was some improvement of nuchal pain. Cardiac markers elevated gradually in serial follow-up, where the highest level was at the 19th-hour after the onset of headache (CK: 1373 IU/L, CK-MB: 105 IU/L, Troponin-I: 24.4 ng/ml). Due to an increasing confidence in the MI

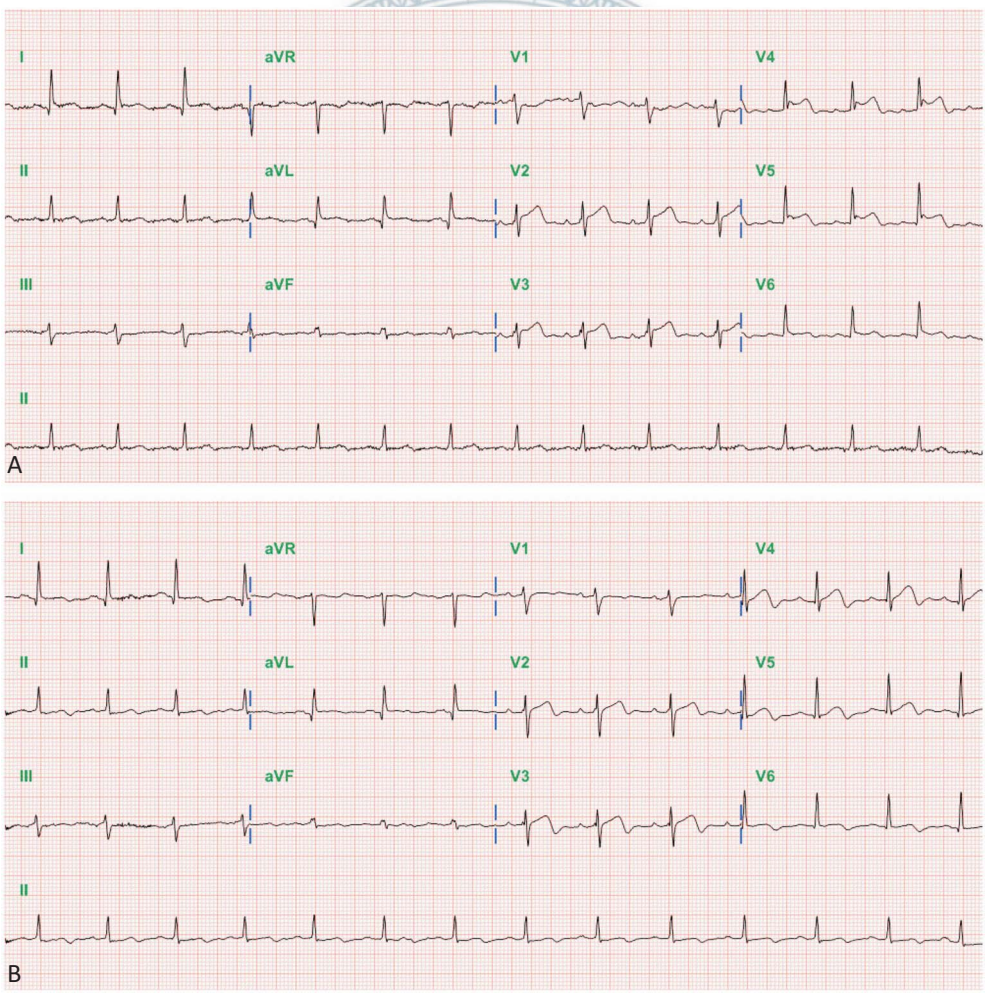

Figure 1. *Electrocardiography (A) 30 minutes (arrival at hospital) and (B) 2 hours after the onset of headaches. An evolutional change of ST-T segment over leads V2-V6 was demonstrated.*

diagnosis, coronary angiogram (Figure 2B, 2C) was performed the next day after customary permission was obtained. The test revealed a significant stenosis of the middle segment (#7) with intramural thrombus of left anterior descending (LAD) artery. Percutaneous coronary intervention (PCI) with drug-eluting stent (Everolimus-eluting stent/Xience PRIME^{TM}) was performed (Figure 2D). To our surprise, the patient said that her headache had disappeared after coronary angioplasty. Four days after PCI, she was discharged symptom-free. There were no more headaches in the next 1-year follow-up in cardiology clinic.

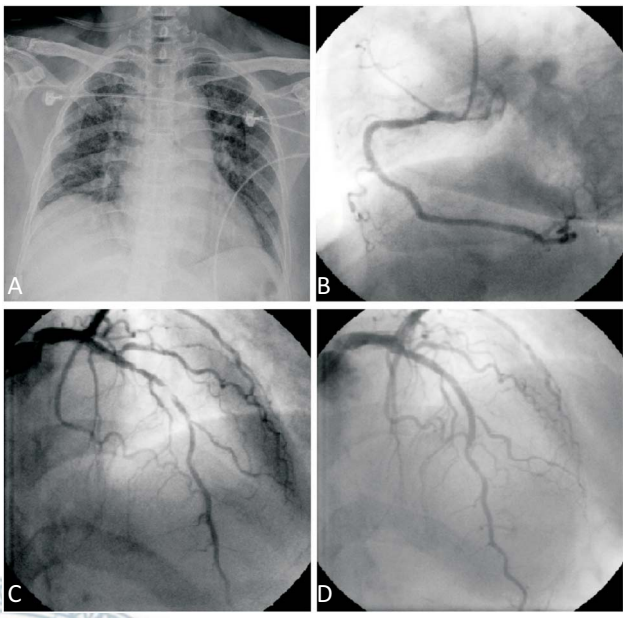

Figure 2. *(A) Chest X-ray at emergency department showed mild lung congestion, suggesting reduced cardiac contractility. CAG in the next day revealed (B) RCA atherosclerosis and (C) proximal-to-middle LAD atherosclerosis with more than 90% stenosis and intramural thrombus at middle segment. (D) After PCI with stenting to middle LAD. CAG, coronary angiogram; LAD, left anterior descending artery; PCI, percutaneous coronary intervention; RCA, right coronary artery.*

DISCUSSION

The third edition of the International Classification of Headache Disorders (ICHD-III beta)[3] proposed diagnostic criteria for cardiac cephalalgia. Basically, headache is regarded as an atypical presentation of angina, which would be aggravated by exertion and not accompanied by photophobia or phonophobia. Pathological alterations as noted by ECG and elevated cardiac markers are regarded as objective evidence of MI, and the causal relationship between headache and MI should be demonstrated. This patient had a history of exertional headache, and she suffered another even more severe episode on the day of her heart attack. The ST-T segment elevation and regional wall motion abnormality were also found, as well as cardiac markers elevation. Her symptoms disappeared parallel to the resolution of myocardial ischemia after PCI, without recurrence thereafter. Her manifestation is consistent with the criteria of cardiac cephalagia, but there are still some questions that require further elucidation for a better understanding and reliable diagnosis of this disease.

Researchers have proposed several theories about the pathogenesis of cardiac cephalalgia. Since the afferent autonomic fibers from the heart relay their signals through cervical dorsal roots, converging with somatic fibers innervating the neck and face (i.e. trigeminal nerve), referred pain is believed to play a role in cardiac cephalalgia.[2,4,5]. Secondly, a sudden reduction of cardiac output secondary to MI reduces cerebral venous return and increases intracranial pressure, which produces a feeling of nociceptive distention.[6,7] The third mechanism postulates that neurochemical mediators released during MI – such as serotonin or bradykinin – result in cerebral vasodilatation/spasm and subsequent headaches.[4,8] In this case, the headache and nuchal pain could be explained as a referred pain to the dermatome of somatic nerves. The patient's dizziness, tachycardia, and elevated blood pressure suggested a reduced cardiac output and hyper-sympathetic tone secondary to various vasoactive mediators.

The diagnostic challenge of cardiac cephalalgia is that its clinical presentations are highly variable. The headache may be unilateral[1] or bilateral,[4,6] and may involve one or more of the frontal, temporal, parietal, or occipital regions.[2,4,6,8] Most patients described it as "explosive" or "thunderclap",[4,8] but some reported only throbbing or squeezing pain.[6] Most headaches were precipitated by exertion and relieved by nitroglycerin or rest.[6,9] Previous analyses found that 27% of patients presented with only headache, while about 30% of them also had nausea or other autonomic phenomenon.[5,7] Since it is unrealistic to evaluate all patients with headaches as possible cases of MI, cardiovascular risk factors

provide a simple way to screen for patients with "cardiac headache".[5-8] Most cases (> 80%) reported in the literature have at least one cardiovascular risk factor, such as older age at onset.[5,7,8] If we apply this knowledge to our case, multiple cardiovascular risk factors (age, hyperlipidemia, hypertension, diabetes mellitus) would prompt physicians to include MI as one of the differential diagnoses of headache, and further diagnostic studies would not be delayed.

Another challenge in cardiac cephalalgia is the choice of diagnostic tool. The use of ECG and cardiac markers are simple and widely available, but only about a half (57%) of the cases had pathological alterations of ECG or elevated cardiac markers at presentation.[5] Nonetheless, ECG and cardiac markers may appear perfectly normal in some patients,[9,10] and diagnosis could only be made by stress test or coronary angiogram – just like in angina. The testing for cardiac markers is not available universally, and the process is always time-consuming. Meanwhile, delayed diagnosis of MI may lead to rapid clinical deterioration in an emergency setting.[1,2,7] According to the American Heart Association's guideline recommendations for management of ST-elevation MI, transthoracic echocardiography provides the evidence of focal left ventricle wall motion abnormality, and could be a revealing triage in a patient with ECG findings that are difficult to interpret, just as this case.

In summary, cardiac cephalalgia is a rare but serious cause of secondary headaches. In the few cases of myocardial ischemia that presented with only headache, an elevated awareness of cardiovascular risk factors would enable physicians to more effectively identify these patients.[1,7,8] Although ECG and cardiac markers are not always abnormal, they are the simplest and most easily available methods to evaluate these patients.[5] In those whom delayed diagnosis would cause rapid deterioration, echocardiography could be another available imaging modality for the diagnosis of cardiac cephalalgia.

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