

A Case of Cardiac Cephalalgia Showing Reversible Coronary Vasospasm on Coronary Angiogram

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Received March 2, 2009

Revised June 11, 2009

Accepted June 11, 2009

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Background Under certain conditions, exertional headaches may reflect coronary ischemia.

Case Report A 44-year-old woman developed intermittent exercise-induced headaches with chest tightness over a period of 10 months. Cardiac catheterization followed by acetylcholine provocation demonstrated a right coronary artery spasm with chest tightness, headache, and ischemic effect of continuous electrocardiography changes. The patient's headache disappeared following intra-arterial nitroglycerine injection.

Conclusions A coronary angiogram with provocation study revealed variant angina and cardiac cephalalgia, as per the International Classification of Headache Disorders (code 10.6). We report herein a patient with cardiac cephalalgia that manifested as reversible coronary vasospasm following an acetylcholine provocation test.

J Clin Neurol 2010;6:99-101

Key Words cardiac cephalalgia, angina pectoris, headache.

Introduction

Cardiac ischemia typically causes chest pain, variously radiating elsewhere. Headache as a symptom of myocardial ischemia has been reported previously.^{1,2} The convergence of cardiac nerve fibers on central pathways receiving somatic afferents from the head is likely to be responsible for the perception of cardiac ischemic pain as headache.¹ Several reports have implicated headache, both with³⁻⁵ and without^{4,6} chest pain, as a clinical manifestation of coronary ischemia. In all previous reports, effect of continuous electrocardiography (ECG)-based testing was abnormal. The neuroanatomical substrates of headache induction by coronary ischemia are not well understood, although mechanisms involving increased intracranial pressure⁴ and convergence or overflow of somatic inputs from lower cervical levels into second-order neurons within the caudal trigeminal nucleus have been proposed.⁶

Case Report

A 44-year-old woman was admitted with exertional headache

that had developed over the previous 10 months. Her headache was located on the bifrontal area and was associated with substernal chest tightness after the onset of 5-10 minutes of exercise. Her headache gradually resolved over a period of minutes to an hour after the cessation of exercise and was not associated with aura, nausea, vomiting, photophobia, phonophobia, palpitation, or diaphoresis. Also, the headache was not provoked by cough, sneeze, or straining during bowel movement. The patient had suffered a severe headache 4 days before her admission to hospital that was biparietally and bioccipitally located, bursting in onset, and which was improved by laying down and resting. On a few occasions, her headaches were associated with chest discomfort and epigastric pain. Her history was negative for hypertension, diabetes, and heart disease, and she did not smoke. Neurological examination, routine laboratory tests, transcranial Doppler, brain imaging studies (brain CT, MRI, and magnetic resonance angiogram), ECG, EEG, and spinal tapping with cerebrospinal fluid studies produced normal results. The findings of a 24-hour Holter monitoring study were occasional periventricular complexes and one episode of nonsustained supraventricular tachycardia. During treadmill exercise

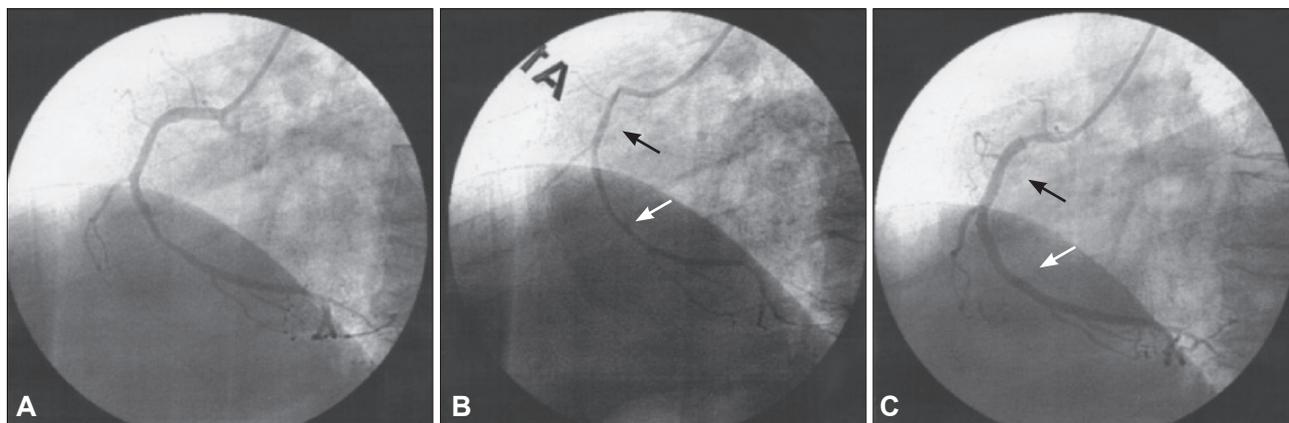


Fig. 1. Coronary angiogram findings of patient. A: Normal finding of the right coronary artery. B: Vasospasm of the right coronary artery in response to an intra-arterial injection of acetylcholine (provocation test). C: Recovery state of the right coronary artery following intra-arterial administration of nitroglycerine.

stress testing, the patient simultaneously suffered severe headache with chest tightness and ST-segment depression on ECG. Termination of the exercise-stress test was followed by resolution of her headache, chest pain, and ECG change. Her headache and chest tightness disappeared on resting. A coronary angiogram was performed the next day, revealing coronary artery spasm following an acetylcholine provocation study during which chest tightness, headache, and ischemic ECG changes appeared (Fig. 1). The symptoms completely disappeared after the patient was administered nitroglycerine by intra-arterial injection. Because of the reversible coronary changes on coronary angiogram (the headache and chest tightness disappeared after intra-arterial nitroglycerine injection), the patient was diagnosed as having variant angina. After 5 days of nitrate and calcium-channel-blocker medication, the patient was discharged without headache or adverse medication effect.

Discussion

Headaches that appear upon exertion are usually benign if structural lesions can be excluded. Organic causes of exertional headache usually result from intracranial structural lesions, but may also occur in association with myocardial ischemia.⁷ Headache as a rare symptom of myocardial infarction, and cardiac cephalalgia has been reported previously.^{2,7-9} Cardiac cephalalgia has only recently been recognized as a distinct entity, being first proposed as 'cardiac cephalgia' by Lipton et al.⁴ in 1997. This condition is classified under the grouping '10. Headache attributed to disorder of homeostasis,' which is coded and named '10.6 cardiac cephalalgia' in the second edition of the International Classification of Headache Disorders (ICHD-II).³ The diagnostic criteria are as follows:³

- 1) Headache, which may be severe, aggravated by exertion, and accompanied by nausea, and fulfilling criteria C and D.
- 2) Acute myocardial ischemia occurs.

- 3) Headache develops concomitantly with acute myocardial ischemia.

- 4) Headache resolves and does not recur after effective medical therapy for myocardial ischemia or coronary revascularization.

Our patient experienced cardiac cephalalgia showing a reversible coronary vasospasm in coronary angiogram according to the ICHD-II: 10.6. The concomitant occurrence of headache and myocardial ischemia is the key criterion (criterion C) for diagnosis. The proposed headache features (criterion A) are not generally satisfactory; in particular, nausea was the least commonly fulfilled criterion in the review study of the literature with the new ICHD-II criteria revisited,¹⁰ in which the proposed mechanisms of cardiac cephalalgia are described. One possible explanation is an anatomical connection. The heart's sympathetic fibers are supplied by cervical and thoracic ganglia. Because fibers from these ganglia also supply the structures of the eye, face, neck, and cerebrovasculature, referral of pain along these pathways might account for headache symptoms. A second possible mechanism is that the decrease in cardiac output and increase in left-ventricular and right-atrial pressures associated with angina pectoris causes a decrease in venous return from the brain and subsequently an increase in intracranial pressure. A sudden and transient increase in intracranial pressure is also proposed as an explanation for cough headache. A third explanation posits an as yet unidentified mediator that is released secondary to cardiac ischemia and which might act on intracranial pain-sensitive structures. Serotonin, bradykinin, histamine, and substance P have been proposed as mediators of ischemic pain and might also have distant intracranial effects. The increase in intracardiac pressure associated with angina may also induce the release of atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP), a response to increased right-atrial and left-ventricular pressures. ANP and BNP are potent vasodilators and thus could produce headache by dilation

of the cerebrovasculature.⁴ The differential diagnosis with migraine is crucial to avoid the administration of vasoconstrictors. In addition, exercise-induced headache relieved by rest should raise a high suspicion of the headache's cardiac cause. Response of a headache to nitrates provides a strong clue as to the myocardial ischemic cause of the symptom.¹¹

Conflicts of Interest

The authors have no financial conflicts of interest.

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