

## Cardiac cephalgia

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**Abstract** The purpose of this review was to provide a critical evaluation of medical literature on so-called “cardiac cephalgia” or “cardiac cephalalgia”. The 2004 International Classification of Headache Disorders codes cardiac cephalgia to 10.6 in the group of secondary headaches attributed to disorder of homeostasis. This headache is hardly recognizable and is associated to an ischaemic cardiovascular event, of which it may be the only manifestation in 27% of cases. It usually occurs after exertion. Sometimes routine examinations, cardiac enzymes, ECG and even exercise stress test prove negative. In such cases, only a coronary angiogram can provide sufficient evidence for diagnosis. Cardiac cephalgia manifests itself without a specific pattern of clinical features: indeed, in this headache subtype there is a high variability of clinical manifestations between different patients and also within the same patient. It “mimics” sometimes a form of migraine either

accompanied or not by autonomic symptoms, sometimes a form of tension-type headache; on other occasions, it exhibits characteristics that can hardly be interpreted as typical of primary headache. Pain location is highly variable. When the headache occurs as the only manifestation of an acute coronary event, the clues for suspicion are a) older age at onset, b) no past medical history of headache, c) presence of risk factors for vascular disorders and d) onset of headache under stress. Knowledge of cardiac cephalgia is scarce, due to its rare clinical occurrence and to the scant importance given to headache as a symptom concomitantly with an ischaemic cardiac event.

**Keywords** Cardiac cephalgia · Exertional headache · Secondary headache · Headache attributed to disorder of homeostasis · Acute myocardial ischemia

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### Introduction

The second edition of the International Classification of Headache Disorders (ICHD-II, 2004) [1] includes forms of primary headache in which the headache symptom is the disorder, and forms of secondary headache that are attributed to underlying pathological conditions. According to this classification, to establish a diagnosis of secondary headache it is necessary to demonstrate the presence of an underlying cause or disorder, the headache must occur in close temporal relation to the demonstrated pathological condition, and it must improve or disappear within 3 months from disappearance or spontaneous remission of the causative factor.

In a study conducted on the Danish general population, investigators found that over 70% lifetime prevalence rates of secondary headaches [2].

While there are currently many data available about the prevalence and triggering or relieving factors of primary headaches, little is known about clinical features of secondary headaches.

In ICHD-II, cardiac cephalgia has been coded as an autonomous clinical entity to 10.6 in Group 10 (“Headache attributed to disorder of homeostasis”).

The purpose of this paper is to provide an accurate review of present-day knowledge about cardiac cephalgia.

## Materials and methods

In this review of the literature, we considered only English-language articles published in scientific journals and chapters of English-language books. We searched for references by entering the key words “cardiac cephalgia” and “exertional headache” in the PubMed search tool, with no limitations to the year of publication. We first reviewed the abstracts of the publications considered for the purpose of our study in order to evaluate their relevance to the subject matter. We also reviewed the references of each publication in order to find other useful material. Overall, we included 30 cases in our review.

## Clinical features

A typical clinical feature of acute coronary syndromes is oppressive pain in the chest, possibly radiating to the left arm and the neck. Headache as the only symptom of an acute cardiac event is very infrequent and is generally associated with other typical symptoms. In 1971, Sampson reported that in a group of 150 patients with angina pectoris, only 6% complained headache and this was not the only symptom that they experienced [3].

The ICHD-II diagnostic criteria for cardiac cephalgia are:

- A. Headache, which may be severe, aggravated by exertion and accompanied by nausea and fulfilling criteria C–D.
- B. Acute myocardial ischemia has occurred (based on the presence of ST-segment elevations or depressions and of T-wave inversions on exercise electrocardiography and the presence of elevated cardiac enzymes).
- C. Headache develops concomitantly with acute myocardial ischaemia
- D. Headache resolves and does not recur after effective medical therapy for myocardial ischaemia or coronary revascularization.

As it is associated to a cardiovascular event, cardiac cephalgia generally occurs after the fifth decade of life in

subjects at risk for cardiovascular disease who may not have previously suffered from headache [4–11].

Unlike primary headaches that have a well-defined pain type and location, in cardiac cephalgia the clinical picture is not homogeneous.

In our review we describe the characteristics of 30 cases reported in the literature (see Table 1).

Pain is not localized in a specific area but may involve the frontal, temporal, parietal, and occipital regions. Besides, the headache may be unilateral or bilateral. Pain is almost always severe or excruciating, constrictive and oppressive or resembling migraine. In some cases there aren't accompanying symptoms, while in others pain is accompanied by autonomic phenomena such as photophobia, phonophobia, osmophobia, and nausea (30% of cases).

Symptoms related to the underlying disorder, such as chest constriction, pain in the left arm radiating up to the mandible or epigastric pain are present in 50% of reported cases.

In 27% of the cases described in literature, cardiac cephalgia is the only manifestation of a cardiovascular ischaemic event.

The headache may start after physical exertion, even after a mild physical activity like walking. In so-called “walk headache” [4, 6, 12–14], for instance, pain appears almost instantly after starting exercise, but will disappear in a few minutes as soon as the patient stops exercising.

Cases have been reported (33%) in which the headache appeared at rest. The frequency of this headache is highly variable; it may appear only in concomitance with the acute cardiovascular event [10, 11, 15–20] or may be more frequent and even occur daily [4, 5, 9, 21] for periods lasting from a few weeks [5, 9, 12, 13, 22–25] to several years [6, 8, 14, 21, 26, 27].

Fifty-seven per cent of patients shows pathological alterations of the baseline ECG trace, such as ST-segment elevations or depressions and T-wave inversions [5, 8–11, 13, 15–20, 25, 27–29], as well as elevated cardiac enzymes [5, 7, 10, 11, 16–20, 26, 27].

However, there are cases in which the ECG may look perfectly normal at rest, with pathological alterations only under stress [4, 12–14, 22–26].

This form of headache does not respond to NSAID therapy [5, 13, 28] and triptans are contraindicated. By contrast, it responds to therapy with nitroderivatives, i.e. drugs that are used to treat cardiovascular disease.

In doubtful cases, the only test that can confirm the diagnosis of cardiac cephalgia is coronary angiography, a procedure that is capable of detecting coronary artery stenosis. Patients will then undergo an angioplasty or bypass surgery that will resolve their cardiac condition and their headache as well. Cardiac cephalgia may occur again in the event of coronary artery restenosis [7].

**Table 1** Clinical features of cardiac cephalgias

Author	Age/ sex	Site	Quality	Intensity	Onset	Duration	Associated autonomic signs	Symptoms of angina	Trigger	Therapy	Follow-up	Risk factors
Caskey [21]	47/M	Right eye	Pressing	Severe	NA	30–40 s	None	Chest pain, left arm pain	Rest, mild exercise	Nitrates	Resolution	A
Lefkowitz [23]	62/M	Bregmatic	Explosive	Severe	NA	NA	NA	Retrosternal pain, arm numbness	Stress, exertion	Bypass surgery	Resolution	A
Fleetcroft [27]	78/F	Frontal	NA	NA	NA	NA	None	Chest tightness	Mild exercise, cold, meal	Nitrates	Resolution	A
Blacky [24]	40/M	Bitemporal	NA	NA	NA	NA	None	None	Vigorous exercise	Nitrates	Resolution	A
Vernay [14]	71/M	Occipital parietal frontal	NA	NA	NA	NA	None	Shoulder pain radiating to arms	Exertion, exercise, meal	Nitrates	Resolution	A, S
Bowen [9]	59/M	Bitemporal	NA	Severe	Sudden	10–30 min	None	Chest pressure, left arm pain	NA	PTCA	Resolution	A, S, O, H, L
Ishida [17]	64/M	Occipital	Throbbing	Severe	Sudden	10 h	Nausea	Shoulder pain	Rest	PTCA	Resolution	A, S
Lipton [22]	57/M	Vertex	Sharp or shooting	Severe	Gradual	Minutes–hours <sup>a</sup>	Nausea	Abdominal or chest pain	Vigorous exercise, sexual activity	Bypass surgery	Resolution	A, S
Lipton [22]	67/M	Bifrontal	Squeezing, steadily pressing	Severe	Gradual	Minutes–hours <sup>a</sup>	None	None	Vigorous exercise	PTCA	Resolution	A, H
Grace [7]	59/M	Vertex occipital	Bursting	Severe	Sudden	Seconds <sup>a</sup>	None	None	Mild exercise	Bypass surgery	Relapse	A
Lance [12]	67/M	Right frontal	NA	NA	Gradual	Minutes <sup>a</sup>	None	Chest pain	Mild exercise	Bypass surgery	Resolution	A, S, L
Lanza [26]	68/M	Occipital	NA	NA	NA	NA	None	Shoulder pain	Rest	Bypass surgery	Resolution	A, S, L
Amendo [10]	77/F	Right frontal and maxillary	NA	Severe	NA	Hours	None	None	NA	NA	NA	A
Amendo [10]	78/F	Bitemporal	NA	Severe	NA	Hours	Vomiting	None	NA	Bypass surgery	Resolution	A, H, L
Auer [15]	47/M	Occipital	NA	NA	NA	Minutes–2 h	NA	NA	NA	Advanced life support	Death	A, S, L
Rambihar [28]	56/F	Occipital	NA	NA	NA	NA	NA	Shoulder and left arm pain	Exercise, meal	Bypass surgery	Partial resolution	A
Famularo [16]	70/M	Fronto-parietal bilateral	Sharp or shooting	Severe	NA	2 days	None	Midepigastric pain	NA	Nitrates	Resolution	A, S, H
Gutierrez [8]	59/M	Vertex occipital bilateral	Dull and throbbing	Moderate–severe	Rapidly progressive	1 day	Nausea photophobia phonophobia	Chest pain	Rest	Nitrates	Resolution	A, H, L
Martinez [4]	68/F	Left hemicranial	Shooting	Severe	Gradual	1 h <sup>a</sup>	None	None	Mild exercise, exertion	PTCA	Resolution	A, S, D, H

Table 1 continued

Author	Age/ sex	Site	Quality	Intensity	Onset	Duration	Associated autonomic signs	Symptoms of angina	Trigger	Therapy	Follow-up	Risk factors
Sathirapanya [13]	58/M	Left occipital	Sharp or shooting	Severe	NA	15–20 min	None	Chest tightness	Rest, exercise	Bypass surgery	Resolution	A, S
Chen [25]	76/M	Bitemporal	Non-throbbing	Mild-severe	NA	5 min	None	Chest pain	Rest, exertion	Nitrates	Resolution	A, H
Gutierrez [5]	74/F	Bitemporal	Pulsating	NA	NA	Minutes–hours	Nausea	Chest tightness	Rest	Nitrates	Resolution	A, O, D
Gutierrez [5]	64/F	Uni- or bilateral	Oppressive	Severe	Sudden	1 h	None	None	Rest, mild exercise	NA	Death	A, H, L
Korantzopoulos [11]	73/F	Occipital	Sharp	Severe	Sudden	1 h	Nausea vomiting	None	Rest	Nitrates	Resolution	A, H, L, O
Cutrer [6]	55/M	Biparietal	Non-throbbing	NA	Gradual	Minutes <sup>a</sup>	None	None	Mild exercise, sexual activity	PTCA	Resolution	A, H, L, O, S
Greiner [18]	50/F	NA	NA	Severe	Sudden	NA	Collapse	None	NA	NA	Death	NONE
Seow [19]	35/M	NA	Explosive	Severe	Gradual	1 day	Vomiting cold sweating	None	NA	NA	Resolution	S
Broner [20]	72/F	Occipital frontal bilateral	Sharp and throbbing	Mild-severe	Sudden	Hours	Nausea vomiting pallor	None	Rest, exercise	Heparin	Resolution	A, D, L
Wei [29]	36/M	Vertex occipital bilateral	Dull	Severe	Rapidly progressive	NA	NA	NA	NA	PTCA	Resolution	S
Wei [29]	85/F	Right eye	NA	NA	NA	NA	NA	Chest pain	Exercise	Nitrates	Resolution	A

A age, S smoke, D diabetes, L hyperlipidaemia, H hypertension, O obesity, NA Not available, PTCA Percutaneous Transluminal Coronary Angioplasty

<sup>a</sup> Duration after end of exercise

## Pathogenesis

There are a variety of theories about pathogenesis of cardiac cephalgia.

The first theory is based on the fact that anginal pain is mediated by sympathetic fibres in 50–60% of cases and by vagal fibres or both in the remaining cases (10–20% and 30–40%, respectively) [30]. According to this theory, cardiac pain referred to somatic structures arises when afferent autonomic fibres (relaying visceral information from the heart through dorsal roots from C8 to T5) and somatic fibres (innervating the chest and arms) converge on sensory neurones in the spinal cord (at the posterior horns) or sympathetic trunk or at the thalamus. This convergence of autonomic sensory fibres and of trigeminal somatic fibres in the descending trigeminal nucleus is less frequent, but is nonetheless responsible for pain in the lower dental arch and in the head, and might then explain the occurrence of cardiac cephalgia. In the cases of cardiac cephalgia with pain in the occipital region, the cause could be the convergence of autonomic and somatic sensory fibres in the upper cervical spinal cord. Sympathetic and somatic sensory afferents converge into common neurones in the posterior horns of the spinal cord. Parasympathetic sensory impulses travelling through the vagal nerve probably converge with somatic sensory impulses at the thalamus. Based on this “convergence” theory, afferent somatic and visceral fibres converge on the same neurones and therefore, when the neurones are stimulated by visceral afferents, the information for the higher centres of the central nervous system is relayed also to the corresponding somatic region [13, 16, 22, 30].

The second pathogenetic theory postulates that the sudden reduction of cardiac output associated with cardiac ischaemia increases pressure in the left ventricle and in the right atrium. The result is a reduction of venous blood flow from the brain, an elevation of intracranial pressure [22], and nociceptive distension of the intracranial structures.

The third pathogenetic theory attributes the pain of cardiac cephalgia to the release of neurochemical mediators as a result of myocardial ischaemia. These mediators—serotonin, bradykinin, histamine, substance P, and atrial natriuretic factor—exhibit a potent vasodilating action on the brain [22, 30, 31].

Finally, there is a fourth pathogenetic theory assuming that cardiac cephalgia could be due to the concomitant presence of vasospasm in both coronary and cerebral vessels [32–34].

## Diagnosis and differential diagnosis

Cardiac cephalgia can be suspected when a patient complains of headache in concomitance with other symptoms accompanying a cardiac ischaemic event.

In “typical” cases, to establish a diagnosis of “cardiac cephalgia” it is necessary to have a patient’s past medical history demonstrating exactly the onset of headache in concomitance with acute myocardial ischaemia verified by stress tests or a radionucleotide scan. In particular, ECGs must be performed at rest and under stress in order to reveal any possible abnormalities that are typical of cardiovascular ischaemic events. An assessment of cardiac markers (CPK-MB, myoglobin, and troponin) is also useful.

Diagnosis is much more difficult when the headache occurs as the only manifestation of an acute coronary event. In such cases, the clinical clues for suspicion that may lead to an accurate diagnosis are older age at onset (which is always indicative of a secondary form), the presence of risk factors for vascular disorders, and onset of headache under stress.

Therefore, it is important to differentiate between cardiac cephalgia as the only manifestation of an acute coronary syndrome and an attack of migraine without aura. Migraine occurs between the second and the third decade of life is more frequent in women, and often develops around the time of the first menstrual cycle. It is often preceded by warning symptoms, such as mood change, shivering, yawning, and hunger. The headache phase is represented by a progressively worsening headache, which usually appears when the patient awakes in the morning and can be triggered by hormonal factors, weather changes, travel, or stressful events. Pain intensity increases progressively for a few hours until it peaks and remains stable throughout the attack.

Both migraine and cardiac cephalgia may occur as a severe headache accompanied by autonomic phenomena, most notably nausea, and can both be worsened by exercise or may develop at rest. It is extremely important to differentiate this condition from other forms, especially migraine without aura. In fact, vasoconstricting agents (such as triptans or ergot derivatives) are indicated for treatment of migraine but should be avoided in patients with cardiac ischaemia because they would worsen their clinical condition.

Triptans are selective agonists of the 5-HT<sub>1B/1D</sub> receptors localized in meningeal arteries, perivascular trigeminal nerve terminals and the central nervous system. They carry out their anti-migraine action by constricting extracerebral intracranial arteries and by inhibiting peripheral neuronal activity and neurotransmission by second-order neurones of the trigeminal-cervical complex [35, 36]. Although these drugs exhibit their vascular effect mainly at the cranial level, a minimal vasoconstricting effect was also observed in the coronary artery district. For that reason, they are not indicated in patients with cardiovascular disease [37, 38].

Other differential diagnoses with cardiac cephalgia, when there are no accompanying symptoms typical of myocardial ischaemia, are primary and secondary forms of exertional headache, thunderclap headache, orgasmic headache, and cough headache. A common feature of this group of headaches is the sudden onset of pain. Primary, or benign, cough headache is precipitated by coughing, is bilateral, and usually resolves in 5 min. Primary, or benign, exertional headache may be precipitated by any form of exercise, is usually bilateral and pulsating, and lasts from a few minutes to 24 h. The orgasmic subtype of primary, or benign, headache associated with sexual activity is characterized by sudden severe pain occurring at orgasm. Apart from an accurate evaluation of the patient's past medical history, it is always necessary to perform a brain MRI to exclude any underlying pathological conditions.

Another differential diagnosis should be with headache triggered by angina treatment such as nitroglycerine, which may precipitate a headache or a migraine attack with its vasodilating action.

Nitric oxide (NO) donor-induced headache is a form of secondary headache that appears following administration of nitroderivatives. Both headache and non-headache patients may develop pain soon after receiving medication (within 10 min). This headache is bilateral, throbbing and exacerbated by physical activity, and it disappears after about 1 h from medication intake. Late NO donor-induced headache occurs more frequently in headache patients. It appears with the same features as primary headache and disappears about 72 h after medication intake [1]. In this case, for differential diagnosis between cardiac cephalgia and NO donor-induced headache, it is important to determine the temporal relation between the administration of nitroderivatives and the appearance of cranial pain in patients with a verified or suspected diagnosis of acute ischaemic coronary disease.

Failure to recognize and correctly diagnose cardiac cephalgia may have severe consequences.

In cases of de novo headache in patients over 50 years of age at risk for cardiovascular disease, some authors believe that it is necessary to perform an ECG and an assessment of cardiac markers [15, 16].

## Conclusion

Headache is a non-specific symptom of various conditions and is rarely considered in medical textbooks as an accompanying symptom of acute coronary syndromes.

In all likelihood, then, cardiac cephalgia is an underestimated symptom in emergency departments but also in neurology or internal medicine departments, where

physicians probably have little knowledge of this subtle form of headache.

**Conflict of interest** None.

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