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Update on headaches associated with physical exertion

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Abstract

Background: Headaches associated with physical exertion include headache precipitated by coughing or other Valsalva maneuvers, headache brought on by prolonged physical exercise, sexual headaches and cardiac cephalalgia.

Objective: To review and update the clinical characteristics, etiologies, pathophysiology and management of these headaches related to exertion.

Methods: In depth review of the publications, both in PubMed and in the main textbooks, of the different headaches induced by physical exercise.

Results: Cough, exercise and sexual headaches can be primary or secondary; therefore, complementary studies are mandatory to rule out structural lesions. However, clinical characteristics, such as an old age and response to indomethacin for cough headache or being a young male and response to beta-blockers for exercise and sexual headaches, plus a normal examination are suggestive of a primary etiology. Etiology for secondary varieties, as posterior fossa lesions for cough headache or vascular malformations for exercise and sexual headaches, are also different. Finally, headache as a distant manifestation of myocardial ischemia, also known as "cardiac cephalalgia", appears at exertion in around two-thirds of cases and typically lasts less than 30 minutes and is relieved by nitroglycerine.

Conclusions: Primary and secondary cough headache can usually be suspected based on clinical characteristics and separated from exercise and sexual headaches, which share many aspects. Cardiac cephalalgia is not necessarily an exertional headache and should be considered in adult patients with short lasting headaches and patent vascular risk factors.

Keywords

Cardiac cephalalgia, cough headache, exercise headache, sexual headache

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Introduction

Headaches associated with physical exertion encompass those provoked by Valsalva maneuvers (cough headache), prolonged exercise (exercise headache) and sexual excitation (sexual headache). Even though they now appear in chapter 4 of the current International Headache Society (IHS) Classification ("Other primary headache disorders") (1), these headaches are a challenging diagnostic problem, as they can be either primary or symptomatic to serious conditions and their secondary etiologies differ depending on the headache type. In fact, for decades, cough and exertional headaches were considered as an ominous symptom.

In 1932, Tinel described a series of patients with paroxysmal headaches following exertion or maneuvers that increased intrathoracic pressure (2). It was neurologist Sir Charles Symonds who used the term "cough headache" and he was the first to show that it may be a benign syndrome without a structural lesion in the central nervous system (3). In the first big series of these headaches, Rooke proposed the term "benign exertional headache" for any headache precipitated

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by "exertion", including Valsalva-like maneuvers, prolonged physical efforts or mechanical factors related to the cervical spine, which meant that the term exertional headache actually encompassed cough headache, headache related to prolonged strenuous exercises and headache attributed to sexual activities (4). This concept remained until the nineties, as shown by the series by Sands et al., who still included all these headaches among the umbrella of "exertional headaches" (5). In this paper, they showed that one out of five of their 219 cases were secondary, usually to a posterior fossa lesion. In 1996 Pascual et al. reported our experience with 72 patients who had consulted due to these types of headaches (6), which was expanded in 2008 with the availability of modern magnetic resonance imaging (MRI) techniques (7). In addition to their different precipitants, we were able to separate the three headache subtypes clinically, and found that the clinical characteristics of those who have a primary type of these three varieties were different to those with secondary headaches, allowing us as clinicians to presume a preliminary etiological diagnosis from its clinical semeiology. In fact, these headaches were clearly defined and separated for the first time in the 2004 IHS Classification (8). In this manuscript we review the current clinical knowledge of the variety of headaches associated with physical exertion.

Cough headache

According to the current IHS Classification, cough headache is a headache precipitated by coughing or other Valsalva (straining) maneuvers, but not by prolonged physical exercise (1). Cough headache can be primary and secondary (6,7,9–14).

Epidemiology

Cough headache is rare in clinical practice. In terms of consultation, we found that 0.6% of new patients in a Headache Clinic are due to cough headache (15). Rasmussen and Olesen have shown that the lifetime prevalence of cough headache is around 1% (95% CI 0–2%) (16).

Clinical manifestations

Primary cough headache is defined as head pain precipitated by coughing or other Valsalva maneuvers in the absence of any intracranial disorder. According to current IHS diagnostic criteria (Table 1) primary cough headache is a sudden onset headache lasting from one second to 30 minutes, brought on by and occurring only in association with coughing, straining and/or other Valsalva maneuvers (1).

Table 1. Diagnostic criteria for primary cough headache (1).

A. At least two headache episodes fulfilling criteria B–D
B. Brought on by and occurring only in association with coughing, straining and/or other Valsalva maneuvers
C. Sudden onset
D. Lasting between 1 second and 2 hours
E. Not better accounted for by another ICDH-3 diagnosis

The clinical picture of primary cough headache usually allows its differentiation from secondary cases. The average age at onset of primary cough headache is around 70 years of age (range 44 to 81 years) and has a clear male predominance. Primary cough headache is an episodic disease with a symptomatic period ranging from a few weeks to a maximum of two years. The pain starts just after precipitants, which include coughing, sneezing, nose blowing, laughing, crying, singing, lifting a weight, defecation or stooping. However, prolonged physical exercise is not a precipitating factor for this entity. Primary cough headache is moderate-severe in intensity, with a stabbing, sharp, splitting or even explosive quality. Typically, headache is bilateral, maximal in the occipital region, but it can also be irradiated to vertex or frontal-temporal regions and lasts from a few seconds to several minutes. Some patients otherwise, describe a dull, aching pain during several hours following the initial paroxysm. Primary cough headache is not associated with other clinical manifestations, including nausea or vomiting, or with focal signs and responds to indomethacin (6,7,9–14,17).

Diagnosis of primary cough headache is confirmed by normal neuroimaging studies. From the old series, before computed tomography (CT) and MRI availability, it was thought that only one out of five cough headache cases were secondary to structural lesions (3,4). Recent studies have shown that about half of those patients consulting due to cough headache have structural lesions, usually (~90%) a Chiari type I deformity and in few cases space-occupying lesions in the posterior fossa/foramen magnum area, causing at least intermittent local blocking of cerebrospinal fluid (CSF) circulation (6,7,17,18) (Figures 1,2). Cough headache can be the only clinical manifestation of a tonsillar descent, even for several years, in up to one-fifth of patients, but most, if not all, patients with secondary cough headache finally develop posterior fossa symptoms or signs, mainly vertigo/dizziness, unsteadiness or syncope (16). In addition, symptomatic cough headache usually starts before the fourth decade of life (three decades earlier, on average, than the primary variant), has an evolution of years and does not respond to indomethacin (6,7,9–14). All these clinical clues must prompt us to

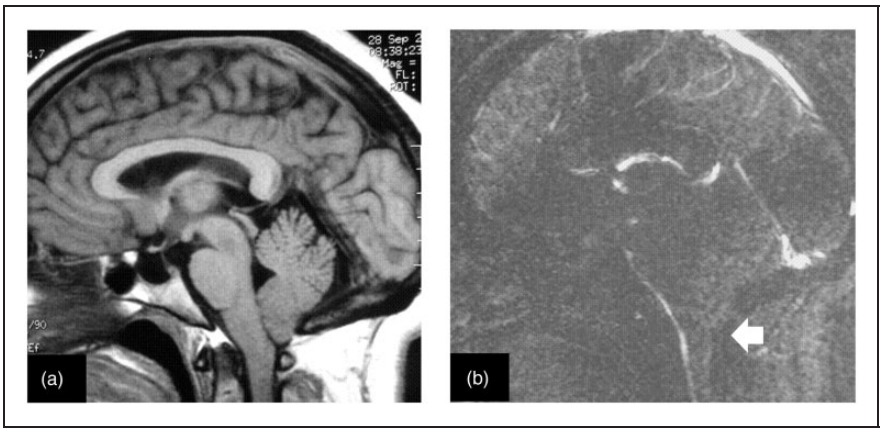

Figure 1. MRI (a) and cine-MRI (b) of a young woman with Valsalva-induced headaches plus cerebellar ataxia secondary to tonsillar descent. Notice the compromised cerebrospinal circulation in the foramen magnum area (arrow).

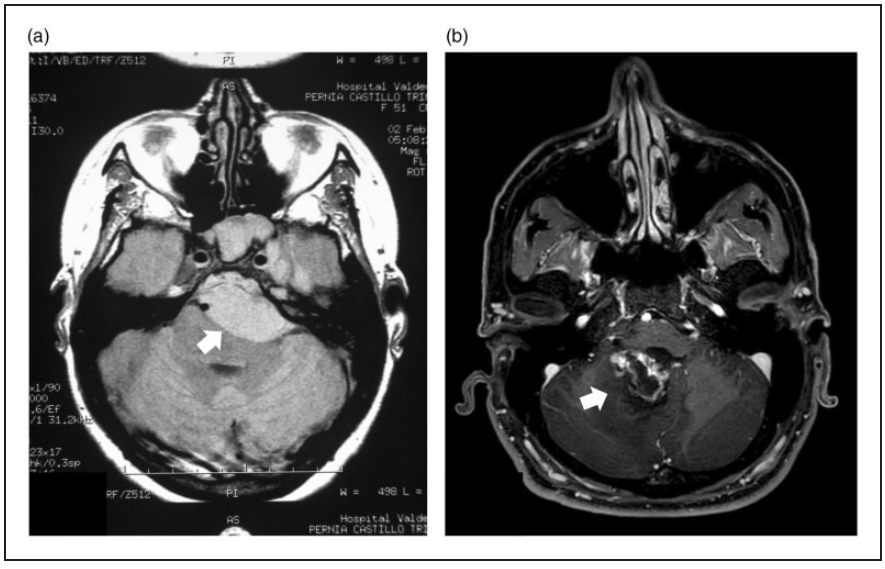

Figure 2. Two examples of MRI with structural posterior fossa lesions (arrows) in patients consulting due to cough headache. (a) one extrinsic meningioma and (b) one intraparenchymal subependymoma. Both patients showed nystagmus and instability.

ask for a cranio-cervical MRI study to rule out secondary cough headache (Figure 3).

Pathophysiology

As it was elegantly demonstrated in the classical works by Williams and Nightingale and Williams (19,20), secondary cough headache is due to a transient impact of the cerebellar tonsils below the foramen magnum disrupting the cerebrospinal normal dynamic, that is, provoking a selective obstruction of the CSF flow from the cranial cavity to the spine (Figure 1). It has been shown that in patients with Chiari type I, the presence of cough headache directly correlates with the degree of tonsillar descent and with the amplitude of tonsillar pulsation as well as with the severity of the arachnoid space reduction (21–23). Taken as a whole, these data confirm that symptomatic cough headache is due to compression or traction of the displaced cerebellar tonsils on pain-sensitive dura and other anchoring structures around the foramen magnum innervated by the first cervical roots.

By contrast, the pathophysiology of primary cough headache and the explanation for the beneficial effect of indomethacin, is unknown. A sudden increase in venous pressure inducing an expanded brain volume or a hypersensitivity of pressure-sensitive receptors localized on the venous vessels have been proposed as potential mechanisms (14,24). A further predisposing factor suggested by Chen et al could be a crowded posterior cranial fossa, though none of these mechanisms have been fully confirmed (25).

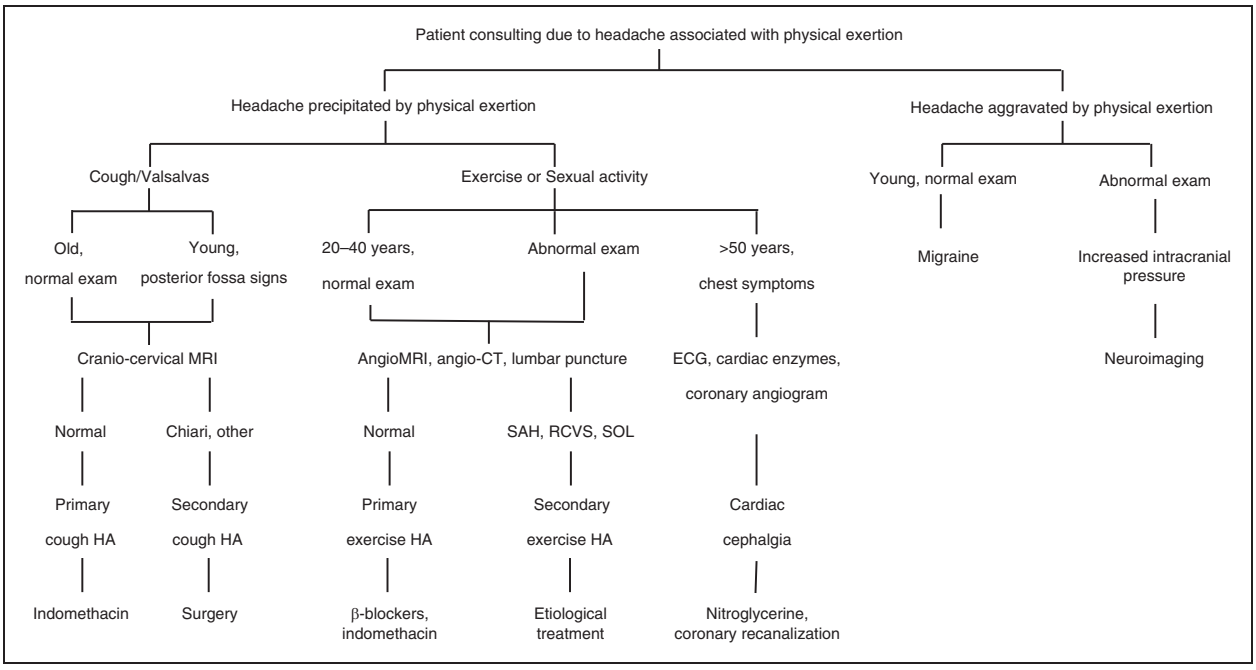

Figure 3. Schema of diagnostic and therapeutic approach in a patient with headache associated with physical exertion. See text for additional information. Abbreviations: HA: headache; RCVS: reversible cerebral vasoconstriction syndrome; SAH: subarachnoid hemorrhage; SOL: space occupying lesions.

Differential diagnosis

Cough headache is sometimes mistakenly included in the differential diagnosis of exertional headaches. However, exertional headaches are not precipitated by Valsalva maneuvers but by prolonged physical exercise. In addition, and in contrast to primary cough headache, primary exercise headache is more frequent in young people and include migraine properties. The same applies for primary sexual headache, which shares a lot of properties with exercise headache. However, as sexual intercourse is a prolonged exercise but also contains Valsalva maneuvers and orgasm, it may also be a precipitant factor for "cough" headache in some patients (14,15).

There are other headaches which can be aggravated (usually not elicited) by cough or other Valsalva maneuvers, which should be included in the differential diagnosis. Migraine and headache due to increased intracranial pressure are the two typical examples. There are scattered reports of patients with cough headaches due to unruptured aneurysms or carotid stenosis, but the relationship between these vascular lesions and cough headache is not established (6,7,9–14).

Cough headache due to low CSF pressure deserves specific mention (26). These patients complain of both orthostatic and cough headaches, which are related to a reversible pseudo-Chiari due to brain sagging (Figure 4). Low CSF pressure syndrome is the most common cause of acquired tonsillar descent. This syndrome can be secondary to CSF removal by lumbar shunting or by lumbar puncture or "spontaneous", usually due to a spinal leak located in the cervicothoracic region. Patients with spontaneous low CSF pressure often report a history of trivial trauma that preceded the onset of symptoms. Factors that predispose to the development of this syndrome include congenital weakness of the dural sac, meningeal diverticula (similar to Marfan syndrome), disorders of a connective tissue matrix, spondylotic spurs or disk herniation (27).

Treatment

The first step should be the treatment or withdrawal of potential precipitants, such as chronic lung diseases or cough-inducing medications, as popular antihypertensives containing angiotensin converting enzyme inhibitors. In our experience just changing to non-coughing drugs improves very significantly the clinical picture in some patients with primary cough headache. Primary cough headache responds to indomethacin, given prophylactically at doses usually ranging from 25–150 mg daily. A decrease in intracranial pressure has been proposed to explain its efficacy, but the actual mechanism of action of indomethacin is unknown (6,7,9–14).

Secondary cough headache does not respond to indomethacin or other pharmacological treatments. Suboccipital craniectomy with posterior fossa reconstruction, for those cases secondary to Chiari type I malformation, and tumor resection for those cases

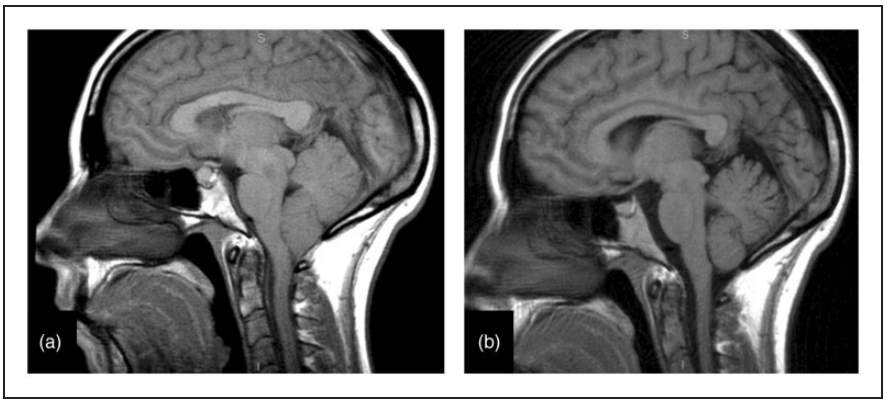

Figure 4. (a) Acquired tonsillar descent in a young woman consulting due to cough plus orthostatic headaches after a lumbar puncture and (b) Resolution of the tonsillar descent after an epidural lumbar patch.

secondary to mass lesions, have been shown to relieve symptomatic cough headache (14).

Exercise headache

Headaches precipitated by prolonged physical exercise are included in the ICHD-3 as "exercise" headache (1). As occurs with cough headache, they can be primary or secondary and investigation of secondary cases in mandatory for patients with these headaches.

Epidemiology

It has been shown that exercise headache accounts for 1–2% of the consultations in general neurological clinics (1,28) and for 5.3% of consultations in a headache clinic (29). Epidemiological studies in the general population have found a higher than expected prevalence of exercise headache: 12.3% in Norway (16) and up to 30.4% in Taiwanese teenagers (30). This discrepancy between hospital-based and general population studies might be due to the fact that most of the exertional headaches are not severe and the sufferers usually do not seek medical help. Possible explanations for the lower prevalence in adults than adolescents might be recall bias of early-life experiences and being less physically active in adulthood. Most of the hospital-based studies found that exertional headaches are more prevalent in males (6,7,9–14), but two large-scaled field studies as well as one study performed in a headache clinic found a female predominance (16,30,31).

Clinical manifestations

Current IHS criteria for the diagnosis of primary exercise headache appear in Table 2 (1).

Contrary to cough headache where the probability of primary and secondary cases is similar, around 80% of cases consulting due to exercise headache are primary (6,7). Also, in contrast to primary cough headache, primary exercise headache is typical of younger people (below 50 years of age), is four times more frequent in males and most of the patients experience a few or multiple headache attacks precipitated by exertion for a few months, usually between two and six months. Around half of the cases occur in patients with a personal history of migraine. Primary exercise headache occurs in both trained and untrained athletes, particularly in hot weather or at high altitude. This headache may be triggered by any kind of prolonged exercise, at least enough to double the resting pulse for over 10 seconds, but ordinarily for minutes or even hours. Headache usually occurs at the peak of the exercise and subsides when the activity stops, even though is some occasions can last for up to two days. Exercise headache is described as aching, pounding or throbbing and has many migraine properties, such as nausea, vomiting, photo and sonophobia. It may be bilateral (about 60% of cases), lasts between 10 minutes and three days (average three to four hours) and is not accompanied by any other neurological (or systemic) symptom/sign (6,7,9–14,31). In spite of a similar age and male predominance, the absence of cranial autonomic symptoms together with duration (up to 48 h) and the usual bilateral pain location clinically differentiate primary exercise headache from cluster headache and other trigeminal-autonomic headaches.

Secondary exercise headache should be suspected if: i) the patient has had only one episode; ii) the consulting

Table 2. Diagnostic criteria for primary exercise headache (1).

A. At least two headache episodes fulfilling criteria B and C
B. Brought on by and occurring only during or after strenuous physical exercise
C. Lasting <48 hours
D. Not better accounted for by another ICDH-3 diagnosis

patient is female; iii) there are other symptoms (such as loss of consciousness) or signs (for instance, neck rigidity), iv) in people older than 40–50 years; and v) if it lasts longer than 24 hours (9).

Primary exercise headache is a diagnosis of exclusion. Even with a typical clinical picture, diagnosis of primary exercise headache can be only reached after a thorough investigation. For typical patients (young males with normal exam), it is mandatory to exclude any kind of intracranial space occupying lesion and sentinel subarachnoid hemorrhage. Therefore, emergency CT and angioCT or MRI with magnetic resonance angiography (MRA), if necessary, should be the screening procedures. In doubtful cases, a lumbar puncture could also be considered. ECG and myocardial enzymes may be required in suspected exercise-induced cardiac cephalgia (see below) (6,7,9–14) (Figure 3, Table 3).

Pathophysiology

The pathophysiology of primary exercise headaches remains obscure. It is believed to be "vascular" in origin due to a hypothesized impaired myogenic cerebrovascular autoregulation, which would lead to headache through an aberrant vasodilation during or following exercise (32). An incompetent internal jugular valve or a stenosis of intracranial venous sinuses, which would lead to transient retrograde venous flow and increased intracranial pressure, have also been proposed to play a role in the pathogenesis of exercise headache (33,34), but it is yet to be demonstrated convincingly in the majority of primary exercise headache cases.

Treatment

Non-pharmacological interventions include proper warm-up before exercise, adequate hydration and regular sports training. If attacks are predictable in patients who do not exercise regularly, preemptive treatment can be given just before exercise. Ergotamine, triptans or indomethacin can be of help. For subjects with frequent attacks, preventive therapy with beta-blockers (for instance, metoprolol 1–2 mg/kg/daily) or indomethacin (25–150 mg/daily) administered during several months have shown (uncontrolled) efficacy (6,7,28,35). In cases with high disability and known venous stenosis, direct retrograde cerebral venography with manometry and even stenting in experienced hands might be considered.

Table 3. Clues for the differential diagnosis of primary versus secondary exercise/sexual headache.

Primary exercise/ sexual headache	Secondary exercise/ sexual headache
Age <40 years	Age >40 years
More than one episode	Only one episode
Men	Women
Pain lasting <24 h	Pain lasting >24 h
No other symptoms/signs	Other neurological symptoms/signs

Headache associated with sexual activity

Primary sexual headache is precipitated by sexual activity, usually starting as a dull bilateral ache as sexual excitement increases and suddenly becoming intense at orgasm, in the absence of any intracranial disorder (1). As with other exertional headaches, headache developed by sexual excitation can be primary or secondary.

Sexual activity can be associated with at least three different headaches. Lance was the first to propose two different subtypes of headaches related to sexual activities (36). First type was a headache related to muscle contraction appearing in the pre-orgasmic phase, actually similar to tension-type headache. Second type would be the explosive, true sexual headache occurring at orgasm (Table 4). Also, a third type of post-orgasmic headache postural (orthostatic) headache related to low CSF pressure has been recognized (1). Silbert et al. already noticed in 1991 that almost half of the patients with true sexual (orgasmic) headache simultaneously experienced exercise headache (28) and this observation was confirmed in subsequent studies (6,7), indicating that exercise and sexual headaches share many aspects or that they are in fact the same entity, which – in terms of clinical expression – depends in the type of exercise performed by the patient when he/she is experiencing such headache. Being this way, the nosology of sexual headaches is even more complicated as some patients experience, coinciding with the Valsalva maneuvers in the orgasm, short lasting headache episodes meeting the criteria for cough headache.

Epidemiology

Concurring with exercise headache, the prevalence of true headache associated with sexual activity is around

Table 4. Diagnostic criteria for primary headache associated with sexual activity (1).

A. At least two episodes of pain in the head and/or neck fulfilling criteria B-D
B. Brought on by and occurring only during sexual activity
C. Either both of the following:
   1. increasing in intensity with increasing sexual excitement
   2. abrupt explosive intensity just before or with orgasm
D. Lasting from 1 minute to 24 hours with severe intensity and/or up to 72 hours with mild intensity
E. Not better accounted for by another ICDH-3 diagnosis

1–2% of the general population (36,37). Sexual headache is two to five times more frequent in males and its peak age at onset is around the forties, though adolescent cases are being increasingly recognized (6,7,9,27,38,39).

Clinical manifestations

Sexual headaches are mostly bilateral and predominantly occipital or holocephalic in location. Most of the sexual headaches are explosive at onset (orgasmic type), but some have gradual onset with increasing sexual excitement (pre-orgasmic type). However, except for the mode of onset, these two subtypes do not have significant differences in demographics, clinical features, comorbidities, or prognosis. Therefore, in the ICHD-3, these two subtypes are no longer described separately (1). These headaches usually occur during sexual intercourse or orgasm, but also can occur during masturbation. Comorbidity with migraine (or exercise headaches) is common.

Primary sexual headache has a clear male predominance (around 4:1) and usually begins between 20–45 years of age. Sexual headaches are usually severe in intensity, lasting from minutes to hours, with a median duration of 30 minutes. Nausea, vomiting, and mood disturbance are only occasionally noted. The location of the pain is bilateral in 75% of patients. Most patients with primary sexual headache have a favorable prognosis as they suffer a bout for several weeks/months, that then disappears even with no treatment. Only in a minority of patients does it follow a chronic course (6,7,9,28,38,39).

As stated for exercise headache, and despite that less than one out of five cases are secondary, explosive sexual headache should be considered as a warning symptom and investigated. The secondary causes of sexual headache include subarachnoid hemorrhage, reversible cerebral vasoconstriction syndrome, cervical or intracranial arterial dissection, hypertensive crisis and hydrocephalus (6,7,38–40). In addition to structural (CT/MRI) brain imaging studies, considering the high proportion of vascular lesions in secondary sexual headache, comprehensive vascular imaging of the head and neck is highly recommended. Studies have shown that subarachnoid hemorrhage precipitated by coitus occurs in 4–12% of all cases with saccular aneurysm and in 4.1% of patients with arterio-venous malformations (11,41). When the patient experiences several episodes of explosive headache during sexual activities, reversible cerebral vasoconstriction syndrome should always be considered (40). Because vasoconstrictions may not be observed at the early stage of this syndrome, follow-up studies may be needed (Figure 3, Table 3).

Pathophysiology

The pathophysiology of primary sexual headaches is unknown. Sexual activities are a form of exercise and thus many aspects of the speculated pathophysiology of sexual headaches are similar to exertional headaches (see below). An impaired metabolic cerebrovascular autoregulation (42), an aberrant central sympathetic response (39) or the existence of venous stenosis (33) have been proposed to explain the pathophysiology of these headaches, but none has been definitely demonstrated.

Treatment

Treatment strategies for sexual headaches are quite similar to those for exercise headache. Because of the variable clinical course, treatment should be individualized. Some patients can improve by transiently stopping or by taking a passive role in sexual activities. Daily beta-blockers may be effective for preventing sexual headache (6,7,28,38,39,43,44). A few cases have been reported a response to topiramate (45). Indomethacin, 25–100 mg one to two hours before intercourse, is effective as a pre-emptive treatment and also as short-term prevention. Ergotamine, triptans and recently erenumab have also shown positive pre-emptive results (44,46).

Cardiac cephalalgia

This headache has been included in the ICHD-3 to describe a migraine-like headache relieved by nitroglycerine occurring during an episode of myocardial ischemia (1). Its current diagnostic criteria appear in Table 5

Epidemiology

This secondary headache is rare. In a very recent study performed in China, the authors were able to identify 30 cases in 12,000 patients hospitalized over two years in their cardiology department, which would mean one case of cardiac cephalgia per 400 patients with myocardial ischemia (47).

Clinical manifestations

After the first reported cases, cardiac cephalalgia was considered an exercise-induced headache (48). However, in 2002, our group reported the first cardiac cephalgia patient with headache at rest and proposed that this headache should not be necessarily restricted within the frame of exercise headaches (48). This has been confirmed in later studies (49–51). Xu et al. and

Table 5. Diagnostic criteria for cardiac cephalalgia (1).

A. Any headache fulfilling criterion C
B. Acute myocardial ischemia has been demonstrated
C. Evidence of causation demonstrated by at least two of the following:
1. headache has developed in temporal relation to the onset of acute myocardial ischemia
2. either or both of the following:
a. headache has significantly worsened in parallel with worsening of the myocardial ischemia
b. headache has significantly improved or resolved in parallel with improvement in or resolution of the myocardial ischemia
3. headache has at least two of the following four characteristics:
a. moderate to severe intensity
b. accompanied by nausea
c. not accompanied by photophobia or phonophobia
d. aggravated by exertion
D. Not better accounted for by another ICDH-3 diagnosis

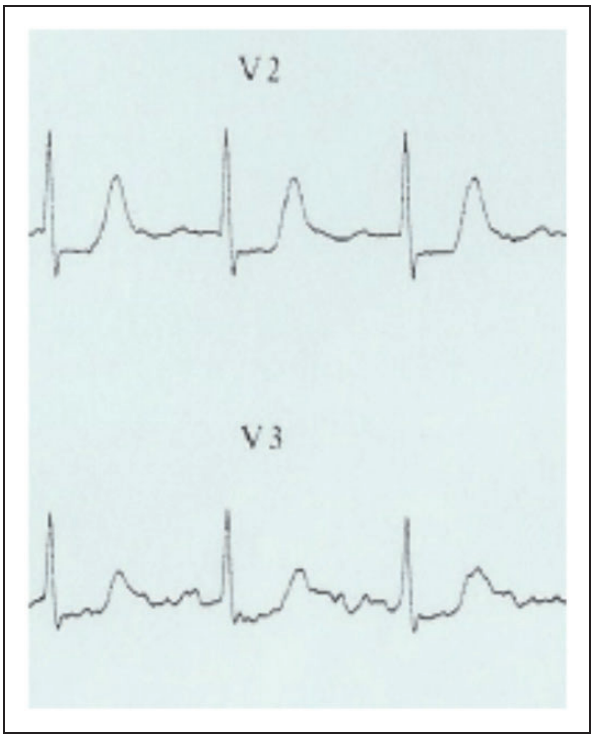

Figure 5. Detail of ECG showing right-inferior ST descent in a 78 years-old woman admitted due to multiple episodes of short-lasting (around 20 minutes), pulsating headache relieved by nitroglycerine. The headache was accompanied by nausea and chest discomfort and was shown to be secondary to myocardial ischemia.

Wang et al. found that in around one-third of patients cardiac cephalgia was not triggered by exercise (47,51).

Cardiac cephalalgia typically appears after the age of 50, in subjects with vascular risk factors and with atherosclerotic heart disease, though it has also been described in patients with normal coronary studies in the context of Prinzmetal angina (52). Location of cardiac cephalgia is not specific, as it can be uni- or bilateral, fronto-temporal or occipital. Vomiting, photo and phonophobia are not frequent (47–50). Usually pain has a throbbing component, is moderate to severe, lasts less than 30 minutes and is accompanied by chest pain/tightness and sweating, though headache can be the only symptom of myocardial ischemia (53). Some patients can exhibit autonomic symptoms (sweating, tearing, runny nose, salivation or defecation), which could lead to a trigeminal autonomic cephalgia misdiagnosis. Myocardial enzymes or ECG are usually abnormal, but they were within normal limits in the study by Xu et al. in up to 20% of cardiac cephalgia cases (47) (Figure 5). Therefore, other studies, such as cardiac monitoring, exercise test or coronary angiography are necessary to confirm the existence of vascular heart disease.

Pathophysiology

Several mechanisms have been proposed to explain headache in cardiac cephalalgia (50). Cardiac pain requires autonomic fibers for transportation to the dorsal horn of the spinal cord, where convergence with the neural somatic structures of neck, face and eyes can occur giving rise to referral pain. The tenth cranial nerve could also be a source for referral pain via its synaptic inputs with the pars caudalis of the trigeminal nerve. Other proposed mechanism of cardiac cephalgia includes transient increase in intracardiac pressure, which would cause intracranial hypertension, though the absence of headache in subjects with increased intracardiac pressure of other etiologies makes this possibility very unlikely. Finally, it is possible that CGRP or other vasodilating peptides are acutely released as a result of the ischemic event either from the ischemic tissue or, as a compensatory vasodilating mechanism, from other sources (54). It is tempting to propose that such release could induce distant, short-lasting, migraine-like headache in subjects with a predisposed trigemino-vascular system.

Treatment

Typically, pain is acutely relieved by nitroglycerine or its derivatives and disappears after successful angioplasty and/or stent implantation (1,49–51).

Article highlights

- Cough, exercise and sexual headaches can be primary or secondary; therefore, complementary studies are mandatory to rule out structural lesions.
- Clinical characteristics, such as old age and the response to indomethacin for cough headache or being a young male and esponding to beta-blockers for exercise and sexual headaches, together with a normal examination, point out a primary etiology.
- The etiologies for secondary varieties, mainly with posterior fossa lesions for cough headache and vascular malformations for exercise and sexual headaches, are also different.
- Headache as a distant manifestation of myocardial ischemia, also known as "cardiac cephalalgia", is induced by exertion in around two-thirds of cases, usually lasts less than 30 minutes and is relieved by nitroglycerine.

Authors contribution statement

Vicente González-Quintanilla and Jorge Madera: literature review and paper revision. Julio Pascual: literature review and preparation of the manuscript.

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References

1. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition. *Cephalalgia* 2018; 38: 1–211.
2. Tinel J. La céphalée a l'effort. Syndrome de distension douloureuse des veines intracraniennes. *Médecine (Paris)* 1932; 13: 113–118.
3. Symonds C. Cough headache. *Brain* 1956; 79: 557–568.
4. Rooke ED. Benign exertional headache. *Med Clin North Am* 1968; 52: 801–808.
5. Sands GH, Newman L and Lipton R. Cough, exertional and other miscellaneous headaches. *Med Clin North Am* 1991; 75: 733–747.
6. Pascual J, Iglesias F, Oterino A, et al. Cough, exertional, and sexual headaches. An analysis of 72 benign and symptomatic cases. *Neurology* 1996; 46: 1520–1524.
7. Pascual J, González-Mandly A, Martín R, et al. Headaches precipitated by cough, prolonged exercise or sexual activity: a prospective etiological and clinical study. *J Headache Pain* 2008; 9: 259–266.
8. Headache Classification Subcommittee of the International Headache Society. The International Classification of Headache Disorders. 2nd Edition. *Cephalalgia* 2004; 24: 1–160.
9. Pascual J. Other primary headaches. *Neurol Clin* 2009; 27: 557–571.
10. Chen PK, Fuh JL and Wang SJ. Cough headache: a study of 83 consecutive patients. *Cephalalgia* 2009; 29: 1079–1085.
11. Allena M, Rossi P, Tassorelli C, et al. Focus on therapy of the Chapter IV headaches provoked by exertional factors: primary cough headache, primary exertional headache and primary headache associated with sexual activity. *J Headache Pain* 2010; 11: 525–530.
12. Cordenier A, De Hertog W, De Keyser J, et al. Headache associated with cough: A review. *J Headache Pain* 2013; 12: 42.
13. Bahra A. Other primary headaches-thunderclap-, cough-, exertional-, and sexual- headache. *J Neurol* 2020; 267: 1554–1566.
14. Pascual J and van den Berg P. Cough headache. In: Ferrari M, Charles A, Dodick D, et al. (eds) *Oxford Textbook of Headache Syndromes*. Oxford University Press, Oxford. 2020, pp. 220–224.
15. Pascual J, Combarros O, Leno C, et al. Distribución por diagnósticos del dolor de cabeza como motivo de consulta neurológica. *Med Clin (Barc)* 1995; 104: 161–164.
16. Sjaastad O and Bakketeig LS. Exertional headache. I. Vaga study of headache epidemiology. *Cephalalgia* 2002; 22: 784–790.
17. Ozge C, Atiş S, Ozge A, et al. Cough headache: frequency, characteristics and the relationship with the characteristics of cough. *Eur J Pain* 2005; 9: 383–388.
18. Pascual J, Oterino A and Berciano J. Headache in type I Chiari malformation. *Neurology* 1992; 42: 1519–1521.
19. Williams B. Cough headache due to craniocerebrospinal pressure dissociation. *Arch Neurol* 1980; 37: 226–230.

20. Nightingale S and Williams B. Hindbrain hernia headache. *Lancet* 1987; 1: 731–734.
21. Sansur CA, Heiss JD, DeVroom LH, et al. Pathophysiology of headache associated with cough in patients with Chiari malformation. *J Neurosurg* 2003; 98: 453–458.
22. Pujol A, Roig C, Capdevila A, et al. Motion of the cerebellar tonsils in Chiari type I malformation studied by cine-phase contrast MRI. *Neurology* 1995; 45: 1746–1753.
23. Quigley MF, Iskandar B, Quigley ME, et al. Cerebrospinal fluid flow in foramen magnum: temporal and spatial patterns at MR imaging in volunteers ad in patients with Chiari type I malformation. *Radiology* 2004; 232: 229–326.
24. Wang SJ, Fuh JL and Lu SR. Benign cough headache is responsive to acetazolamide. *Neurology* 2000; 55: 149–150.
25. Chen YY, Lirng JF, Fuh JL, et al. Primary cough headache is associated with posterior fossa crowdedness: a morphometric MRI study. *Cephalalgia* 2004; 24: 694–699.
26. Ramón C, González-Mandly A and Pascual J. What differences exist in the appropriate treatment of congenital versus acquired adult Chiari type I malformation? *Curr Pain Headache Rep* 2011; 15: 157–163.
27. Mokri B. Spontaneous intracranial hypotension. *Continuum* 2015; 21: 1086–1108.
28. Silbert PL, Edis RH, Stewart-Wynne EG, et al. Benign vascular sexual headache and exertional headache: interrelationships and long term prognosis. *J Neurol Neurosurg Psychiatry* 1991; 54: 417–421.
29. Tugba T, Serap U, Esra O, et al. Features of stabbing, cough, exertional and sexual headaches in a Turkish population of headache patients. *J Clin Neurosci* 2008; 15: 774–777.
30. Chen SP, Fuh JL, Lu SR and Wang SJ. Exertional headache–a survey of 1963 adolescents. *Cephalalgia* 2009; 29: 401–407.
31. Sjaastad O and Bakketeig LS. Exertional headache–II. Clinical features Vaga study of headache epidemiology. *Cephalalgia* 2003; 23: 803–807.
32. Heckmann JG, Hilz MJ, Muck-Weymann M et al. Benign exertional headache/benign sexual headache: a disorder of myogenic cerebrovascular autoregulation? *Headache* 1997; 37: 597–598.
33. Doepp F, Valdueza JM and Schreiber SJ. Incompetence of internal jugular valve in patients with primary exertional headache: a risk factor? *Cephalalgia* 2008; 28: 182–185.
34. Donnet A, Valade D, Houdart E, et al. Primary cough headache, primary exertional headache, and primary headache associated with sexual activity: a clinical and radiological study. *Neuroradiology* 2013; 55: 297–305.
35. Diamond S. Prolonged benign exertional headache: its clinical characteristics and response to indomethacin. *Headache* 1982; 22: 96–98.
36. Lance JW. Headaches related to sexual activity. *J Neurol Neurosurg Psychiatry* 1976; 39: 1226–1230.
37. Rasmussen BK and Olesen J. Symptomatic and nonsymptomatic headaches in a general population. *Neurology* 1992; 42: 1225–1231.
38. Frese A, Eikermann A, Frese K, et al. Headache associated with sexual activity: demography, clinical features, and comorbidity. *Neurology* 2003; 61: 796–800.
39. Yeh YC, Fuh JL, Chen SP, et al. Clinical features, imaging findings and outcomes of headache associated with sexual activity. *Cephalalgia* 2010; 30: 1329–1335.
40. Lin PT, Wang YF, Fuh JL, et al. Diagnosis and classification of headache associated with sexual activity using a composite algorithm: A cohort study. *Cephalalgia* 2021; 41: 1447–1457.
41. Mauri G, Vega P, Murias E, et al. Fusiform aneurysms of the vertebral artery: a hidden cause of exertional headache? *Cephalalgia* 2012; 32: 715–718.
42. Evers S, Schmidt O, Frese A, et al. The cerebral hemodynamics of headache associated with sexual activity. *Pain* 2003; 10: 73–78.
43. Frese A, Rahmann A, Gregor N, et al. Headache associated with sexual activity: prognosis and treatment options. *Cephalalgia* 2007; 27: 1265–1270.
44. Evans RW and Pascual J. Expert opinion: orgasmic headaches: clinical features, diagnosis, and management. *Headache* 2000; 40: 491–494.
45. Evans RW. Topiramate for the prevention of primary headache associated with sexual activity: the third and fourth case reports. *Headache* 2020; 60: 1800–18002.
46. Makarevicious G and Ryliskine K. Sucessful treatment of primary headache associated with sexual activity using erenumab: Case report. *Cephalalgia* 2022; 42: 680–683.
47. Wang C, Xu J, Mao N, et al. Clinical analysis of 30 cases of cardiac cephalgia. *Cephalalgia* 2022; 42: 926–932.
48. Lipton R, Lowenkopf T, Bajwa Z, et al. Cardiac cephalgia: A treatable form of exertional headache. *Neurology* 1997; 49: 813–616.
49. Gutiérrez-Morlote J and Pascual J. Cardiac cephalgia is not necessarily an exertional headache: case report. *Cephalalgia* 2002; 22: 765–766.
50. Wei J and Wang H. Cardiac cephalalgia: Case reports and review. *Cephalalgia* 2008; 28: 892–896.
51. Wang SJ, Chen PK and Fuh JL. Cough headache: a study of 83 consecutive patients. *Cephalalgia* 2009; 29: 1079–1085.
52. Yang YS, Jeong D, Jin DG, et al. A case of cardiac cephalgia showing reversible coronary vasospasm on coronary angiogram. *J Clin Neurol* 2010; 6: 99–101.
53. Martínez HR, Rangel-Guerra RA, Cantú-Martínez L, et al. Cardiac headache: hemicranial cephalalgia as the sole manifestation of coronary ischemia. *Headache* 2003; 42: 1029–1032.
54. Mair J, Lechleitner P, Länge T, et al. Plasma CGRP in acute myocardial infarction. *Lancet* 1990; 335: 168.