

OTHER PRIMARY HEADACHES

Chapter 99

Primary Stabbing, Cough, Exertional, and Thunderclap Headaches

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PRIMARY STABBING HEADACHE

International Headache Society (IHS) ICHD-II Code:
4.1 Primary stabbing headache

World Health Organization (WHO) ICD-10NA Code:
G44.800 Idiopathic stabbing headache

Short description: Transient and localized stabs of pain in the head that occur spontaneously in the absence of organic disease of underlying structures or of the cranial nerves

Previously used terms: Ice-pick pains; jabs and jolts; ophthalmodynia periodica

EPIDEMIOLOGY

Prevalence of the ultrashort paroxysms of head pain that characterize primary stabbing headache has been difficult to estimate and results have varied considerably. In two population-based studies, idiopathic stabbing headache was found to be less than 1 to 2% (48,65). However, in a large-scale, cross-sectional study of headache in a parish in the mountainous region of Vågå, Norway, primary stabbing headache was verified in 35.2% of the 1,838 adult parishioners (18 to 65 years of age) who were examined (79).

Prevalence estimates vary depending on the studied population. For example, primary stabbing headache is more prevalent in migraineurs and individuals with tension-type headache. Not surprisingly, given the prevalence of these primary headache disorders in the population, one would expect a prevalence of at least one third in a general unselected population. Indeed, the prevalence of primary stabbing headache in one study of migraine sufferers was 42% compared to only 3% in controls (64). The high prevalence of primary stabbing headache among migraineurs has been confirmed in subsequent studies (20).

The mean age of onset of primary stabbing headache is 47 years (range 12 to 70) (52). The female:male ratio has varied from 1:49 to 6.6.

CLINICAL FEATURES

Diagnostic Criteria for Primary Stabbing Headache (28):

- A. Pain occurring as a single stab or a series of stabs confined to the head and exclusively or predominantly felt in the distribution of the first division of the trigeminal nerve (orbit, temple, and parietal areas).
- B. Stabs last for up to a few seconds and recur with irregular frequency ranging from one to many per day.
- C. No accompanying symptoms.
- D. Not attributed to another disorder.

The painful attacks of primary stabbing headache are ultrashort, lasting only 1 second in more than two thirds of cases (52). In some patients, the pain can last up to 10 seconds. The pain is frequently unifocal, often in the orbital region, but multifocal patterns have been described with the pain quickly and erratically changing location. The pain has also been described in the facial, occipital, retroauricular, temporal, and even parietal regions. The attacks are often sudden, and moderate to high intensity, with a pricking or stabbing character. The frequency of attacks vary widely from 1 attack per year to 50 attacks daily. In one study, chronic attacks (>80% of days) occurred in 14% of 38 patients followed over a 1-year period. Most attacks are distributed throughout the day. The paroxysms generally occur spontaneously without provocation. There are seldom associated features such as lacrimation, rhinorrhea, conjunctival injection, nausea, or photophobia. Attack-related conjunctival hemorrhage and monocular visual loss have been described (52,94). Occasionally, bright

light, emotional stress, and postural changes are reported to trigger attacks. The ultrashort duration and lack of cranial autonomic features distinguish this disorder from short-lasting unilateral neuralgiform pain with conjunctival injection and tearing (SUNCT) syndrome, where attacks last from 15 to 180 seconds. The presence of trigger points, duration of a few seconds, and occurrence of pain in the second and third trigeminal distribution are characteristic of trigeminal neuralgia, with which primary stabbing headache can be confused. In addition to a high prevalence in patients with migraine, primary stabbing headache may also be seen in association with tension-type headache, cluster headache, cervicogenic headache, and during painful exacerbations in up to two thirds of patients with hemicrania continua.

Although invariably benign, primary stabbing headache may be associated with intracranial structural lesions such as meningioma and pituitary tumors (38,44). Primary stabbing headache has also been associated with onset of cerebrovascular diseases, cranial and ocular trauma, and herpes zoster (52). An association may also exist between new-onset primary stabbing headache and intraocular pressure elevation. Therefore, when clinically suspect, patients with new-onset primary stabbing headache should undergo a diagnostic evaluation to exclude organic pathology.

MANAGEMENT

Acute treatment of primary stabbing headache is not feasible given its ultrashort duration and repetitive nature. When attacks occur with a frequency that warrants prophylactic therapy, indomethacin is usually the treatment of choice (grade C recommendation). Indomethacin provides complete or partial improvement in two thirds of patients (16,46,52). The usual effective dose range from 25 to 150 mg per day. Recently, melatonin was described as completely effective in three patients at doses ranging from 3 to 9 mg at bedtime (69). A patient with primary stabbing headache and exploding head syndrome was reported to have responded to nifedipine (31). In three patients who began to experience primary stabbing headache after ischemic stroke, complete response was obtained with the use of celecoxib 100 mg twice daily (60).

PRIMARY COUGH HEADACHE

International Headache Society (IHS) ICHD-II Code: 4.2 Primary cough headache
World Health Organization (WHO) ICD-10NA Code: G44. 803 Benign cough headache
Short description: Headache precipitated by coughing or straining in the absence of any intracranial disorder

Previously used terms: Benign cough headache, valsalva maneuver headache

EPIDEMIOLOGY AND ETIOLOGY

Cough headache is considered a rare entity. Rasmussen and Olesen (66) have shown that the lifetime prevalence of cough headache is one percent (95% confidence interval [CI] 0 to 2%). Over 15 years, of the 3,498 patients attending a neurology department owing to headache, 20 (0.6%) consulted because of cough headache (54).

Cough headache can be either a primary benign condition or secondary to structural cranial disease. From older case series (prior to computed tomography [CT] and magnetic resonance imaging [MRI]), it was concluded that about 20% of patients with cough headache had structural lesions, most of them a Chiari type I deformity (49,54,68,70,84). However, with modern neuroradiologic techniques, it is clear that almost half of cough headache patients have symptomatic cough headache owing to tonsillar descent or, more rarely, to other space-occupying lesions in the posterior fossa/foramen magnum area (55). Around 30% of patients with Chiari type I malformation experience headache aggravated by Valsalva maneuvers, mainly cough (56) (Fig. 99-1). In summary, it can be concluded that about 50% of the patients with cough headache show no demonstrable etiology, and the other half are secondary to structural lesions, mostly at the foramen magnum level (57).

PATHOPHYSIOLOGY

Secondary cough headache seems to be caused by a temporary impaction of the cerebellar tonsils below the foramen magnum (50,71,90,91). In two patients with cough headache and tonsillar herniation, Williams demonstrated a pressure difference between the ventricle and the lumbar subarachnoid space during coughing (91). This craniospinal pressure dissociation displaces the cerebellar tonsils into the foramen magnum. Williams also observed that the headache disappeared after decompressive craniectomy. Subsequently, Nightingale and Williams described four more patients who had headache from episodic impaction of the cerebellar tonsils in the foramen magnum after abrupt Valsalva maneuvers (50). In our series, not only was it demonstrated that tonsilar descent is the actual cause of cough headache, but it was also shown that the presence of cough headache in Chiari type I patients only correlated with the degree of tonsilar descent (55,56). Pujol et al. (61), using cine phase-contrast MRI, were able to detect this abnormal pulsatile motion of the cerebellar tonsils in Chiari type I patients, but not in controls. This movement produced a selective obstruction of

FIGURE 99-1. Preoperative (A) and postoperative (B) T₂-weighted sagittal MR images from a 36-year old woman with secondary cough headache. (A) Note the presence of tonsillar descent (arrow) and flattening of posterior fossa (asterisks) as well as the absence of cisterna magna. (B) After posterior fossa reconstruction, note the appearance of cisterna magna with restitution of cerebrospinal fluid transit (asterisks) with upward migration of the tonsils. Reproduced with permission from Pascual J. Activity-related headache. In: Gilmar S, ed. *MedLink Neurol*. San Diego: MedLink Corporation.



the cerebrospinal fluid (CSF) flow from the cranial cavity to the spine. The amplitude of the tonsillar pulsation and the severity of the arachnoid space reduction were associated with cough headache (61). Collectively, these data confirm that symptomatic cough headache is usually secondary to Chiari type I deformity and that this pain is caused by compression or traction of the caudally displaced cerebellar tonsils on pain-sensitive dura and other anchoring structures around the foramen magnum that are innervated by the upper cervical nerve roots.

The pathophysiology of primary cough headache is not known. The possibility of a sudden increase in venous pressure being sufficient in itself to cause headache caused by an increase in brain volume has been proposed (88). Other contributing factors, however, may be operant, such as a pressure receptor hypersensitivity hypothetically localized on the venous vessels (63). One of the potential etiologies for this transient receptor sensitization could be a hidden or previous infection (92). Finally, Chen et al. (9) recently found that patients with primary cough headache have a reduction in posterior fossa volume with subsequent crowding of structures, which may be a further contributing factor for the pathogenesis of this headache syndrome.

CLINICAL FEATURES

Diagnostic Criteria of Primary Cough Headache (28):

A. Headache fulfilling criteria B and C

B. Sudden onset, lasting from 1 second to 30 minutes

C. Brought on by and occurring in association with coughing, straining and/or Valsalva maneuver

The clinical picture of primary cough headache is very characteristic, which allows its differentiation from secondary cases (3,53,55,57) (Table 99-1). Primary cough headache does not begin earlier than 40 years; the mean age of onset in modern series is 67 years (range 44 to 81 years). Up to 80% of patients suffering from primary cough headache are men. Primary cough headache is an episodic disorder, ranging from 2 months to 2 years. The pain begins immediately or within seconds after the precipitants, which include cough, sneezing, nose blowing, laughing, crying, singing, lifting a burden, straining at stool, and stooping. Sustained physical exercise is not a precipitating factor for primary cough headache. Primary cough headache is moderate to severe in intensity, with a sharp, stabbing, splitting, or even explosive quality. Most patients have bilateral headaches all the time. The pain is usually maximal in the occipital region, but also in the frontal or temporal region or at the vertex. The pain typically lasts from seconds to several minutes. In a few patients, a dull, aching pain follows the paroxysm for several hours (13). Primary cough headache is not associated with other clinical manifestations, including nausea or vomiting, and responds to indomethacin.

The presence of a Chiari type I malformation or any other lesions causing obstruction of CSF pathways or displacing cerebral structures must be excluded before cough

TABLE 99-1 Clinical Characteristics of Cough and Exertional Headache Patients

	Primary Cough Headache	Symptomatic Cough Headache	Primary Exertional Headache	Symptomatic Exertional Headache
Mean age (range)	67 (44–81)	39 (15–63)	24 (10–48)	42 (18–61)
Gender, % male	77	59	88	43
Duration	Seconds–30 min	Seconds–days	5 min–48 hr	1 day–1 month
Frequency	One-multiple/daily	One-multiple/daily	One a day to 1 in 2 mo	One attack in SAH; multiple in patients with space-occupying lesions
Persistence	2–24 months	Persisting	15 days–10 years	1 day–1 month
Quality	Sharp, stabbing	Bursting, stabbing	Pulsating	Explosive, pulsating
Other manifestations	No	Posterior fossa signs	Nausea, photophobia	nausea, vomiting, neck rigidity
Diagnosis	Idiopathic	Chiari type I malformation	Idiopathic	SAH, sinusitis, brain metastases
Effective treatment	Indomethacin	Suboccipital craniectomy	Ergotamine, propranolol, indomethacin	Specific to diagnosis

Abbreviation: SAH: subarachnoid hemorrhage.
Adapted from 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.

headache is assumed to be benign. Cough headache may be the only clinical manifestation of Chiari type I malformation for several years in about one fifth of symptomatic patients. In our experience, however, most if not all patients with symptomatic cough headache finally develop posterior fossa symptoms or signs, mainly dizziness or vertigo, unsteadiness, and syncope (55). Symptomatic cough headache begins three decades earlier, on average, than primary cough headache, and does not show a clear male predominance or respond to indomethacin (57).

Migraine, cluster headache, post-lumbar puncture headache, and idiopathic intracranial hypertension can be aggravated but not elicited by cough. Given the differential diagnosis outlined, every patient with cough headache should have a brain MRI to rule out tonsillar descent or a posterior fossa lesion. In spite of scattered reports, there is not enough scientific background to support unruptured aneurysms (81), carotid stenosis (6,67), or vertebrobasilar disease (72) as specific causes for cough headache. Therefore, a magnetic resonance angiography (MRA) study is not felt to be mandatory in such patients.

MANAGEMENT

Acute treatment is impractical because of the short duration and multiplicity of cough headaches. Primary cough headache responds to indomethacin, given prophylactically at doses usually ranging from 25 to 150 mg per day (14,16,45). The mechanism of action of this drug is unknown, but could include a decrease in intracranial pres-

sure (80). This would explain the benefits seen with lumbar puncture or acetazolamide in patients with primary cough headache (8,13,62).

Patients with symptomatic cough headache do not consistently respond to any known pharmacologic treatment, including indomethacin, and need specific surgical treatment. It has been shown that suboccipital craniectomy relieves cough headache in patients with Chiari type I malformation (55,56).

PRIMARY EXERTIONAL HEADACHE

- International Headache Society (IHS) ICHD-II Code:
4.3 Primary exertional headache
- World Health Organization (WHO) ICD-10NA Code:
G44. 804 Benign exertional headache
- Short description: Headache precipitated by any form of exercise. Subforms such as weightlifters’ headache are recognized.
- Previously used terms: Benign exertional headache

PATHOPHYSIOLOGY

The pathophysiology of primary exertional headache remains speculative. The development of headache after sustained exertion, particularly after a hot day, is more likely caused by arterial dilatation, but objective evidence is lacking. Because patients with primary exertional headache show an increased frequency of both migraine and orgasmic headache, they could also share some

pathophysiologic mechanisms (25,76). The acute onset of headache with straining and breath holding, as in weightlifter’s headache, is most likely explained by acute venous distension similar to what happens in cough headache (34).

CLINICAL FEATURES

- Diagnostic Criteria of Primary Exertional Headache (28):
- A. Pulsating headache fulfilling criteria B and C
 - B. Lasting from 5 minutes to 48 hours
 - C. Brought on by and occurring only during or after physical exertion

Contrary to primary cough headache, primary exertional headache is typical of young people (range 10 to 48 years in our series) (see Table 99-1). As occurs with primary cough headache, primary exertional headache is more common in men and boys. The majority of cases occur in patients who have a personal and/or family history of migraine (56,90). Primary exertional headache occurs in both nonathletes and trained athletes. Heat, humidity, barometric changes, high altitude, caffeine, hypoglycemia, and alcohol use have been described as contributing factors (10). This headache may be triggered by any kind of prolonged physical exercise (10,30,58), or at least exercise sufficient to double the resting pulse for longer than 10 seconds, but ordinarily for minutes or even hours. Headache usually occurs at the peak of the exercise and subsides when the activity ceases, even though on some occasions headache can last up to 2 days. Exertional headache is described as aching, pounding, or throbbing and has many migraine characteristics, with associated nausea, vomiting, photophobia, and some phonophobia. It may be bilateral (about 60% of the cases) or unilateral (56,90).

Even in the presence of a typical clinical picture, the diagnosis of primary exertional headache can be made only after a thorough investigation. For typical patients (middle-aged men with normal examination), it is mandatory to exclude any kind of intracranial space-occupying lesion and sentinel hemorrhage from vascular malformations (56). Very rarely, exertional headache is a symptom of middle cerebral artery dissection or pheochromocytoma (59). Nowadays, MRI followed by MRA should be the screening procedures. In doubtful cases, a lumbar tap could also be considered. A number of papers have documented exertional (2,5,24,35,37,41,87)—and recently nonexertional (26)—vascular headaches as the presenting symptoms of cardiac ischemia (*cardiac cephalgia*). In these rare cases, cardiac enzymes and an electrocardiogram are indicated.

MANAGEMENT

For nonincapacitating cases or for those with a low exercise frequency, the first, and sometimes the only recommendation should be transient exercise moderation or abstinence. Lambert and Burnett described how a prescribed warm-up period prevented swimmer’s headache (33). There is no absolute evidence of the value of pharmacologic treatments in the management of primary exertional headache. In general, however, antimigraine preventive medications show some benefit. For most patients, β -blockers at the usual antimigraine doses seem useful (23,55). There are well-documented cases with exertional headache who did not improve or could not tolerate β -blockers. Some of these cases seem to improve on indomethacin in doses varying from 25 to 150 mg per day (13). There is no consensus on the treatment duration in these cases. Primary exertional headache is usually transient, lasting less than 3 months and rarely longer than 6 months. Therefore, we recommend stopping the preventive treatment after 3 to 6 months to check for headache recurrence.

Acute therapy, immediately before physical exercise, may be a reasonable alternative for some patients. Simple analgesics and nonsteroidal anti-inflammatory drugs do not seem to prevent the development of exertional headaches. Ergotamine may be useful, and triptans could theoretically be an alternative treatment to ergotamine, but, again, there is no scientific evidence to support the use of triptans in the acute or preemptive treatment of exertional headache (23).

PRIMARY THUNDERCLAP HEADACHE

- International Headache Society (IHS) ICHD-II Code:**
4.6 Primary thunderclap headache
- World Health Organization (WHO) ICD-10NA Code:**
G44.80 Benign thunderclap headache
- Short description:** High-intensity headache of abrupt onset mimicking that seen in the case of ruptured cerebral aneurysm
- Previously used terms:** Benign thunderclap headache

EPIDEMIOLOGY

The epidemiologic characteristics of primary thunderclap headache are unclear. The recognition that thunderclap headache may be a primary headache disorder has only recently been considered, and the International Headache Society argues that the evidence for its existence is preliminary (28). Thunderclap headache is frequently associated with secondary causes, particularly subarachnoid

TABLE 99-2 Primary Thunderclap Headache: Differential Diagnosis

Primary	Secondary
Primary sexual headache	Subarachnoid hemorrhage
Primary cough	Cerebral venous sinus thrombosis
Primary exertional	Arterial dissection
	Pituitary apoplexy
	Hypertensive encephalopathy (+ pheochromocytoma)
	Posterior leukoencephalopathy syndrome
	Spontaneous intracranial hypotension (CSF leak)
	Retroclival hematoma
	Benign angiopathy of the central nervous system
	Call-Fleming syndrome
	Drug induced (SSRIs, cocaine, amphetamine, MAOI)

Abbreviations: CSF, cerebrospinal fluid; MAOI, monoamine oxidase inhibitor; SSRIs, selective serotonin reuptake inhibitors

hemorrhage and other serious vascular disorders, as well as several primary headache disorders, such as cough, exertional, and sexual headache (Table 99-2). Distinguishing between primary and secondary thunderclap headache cannot be done on the basis of the headache characteristics alone. Accordingly, an exhaustive and swift search for an underlying cause in all patients presenting with thunderclap headache is warranted. To gain insights into the epidemiology, clinical spectrum, pathophysiology, appropriate management, and prognosis of patients with primary thunderclap headache, clinical criteria needed to be established to create a homogeneous patient population that can be studied in a prospective fashion (28).

PATHOPHYSIOLOGY

The pathophysiology of primary thunderclap headache is unclear. The occurrence of the headache during physical activity, in patients with pheochromocytoma or acute hypertensive crises, and in patients who ingest sympathomimetic drugs (amphetamine, cocaine) or foods containing tyramine while concurrently using monoamine oxidase inhibitors, suggests the possibility that excessive sympathetic activity or an abnormal vascular response to circulating catecholamines may be involved (1,7,17,18,32,42,51,83,86).

Because sympathetic tone and sympathetic receptor sensitivity are in part responsible for vascular caliber and vasomotor tone, one possible mechanism proposed is an

aberrant central sympathetic neurogenic reflex (15). Sympathetic nerves containing neuropeptide Y richly invest and cause concentration dependent constriction of human intracerebral arteries (21,22). Furthermore, the abrupt onset of both the headache and vasospasm imply a neurogenic, rather than a mechanical or biochemical mechanism.

CLINICAL FEATURES

- Diagnostic Criteria
- A. Head pain is severe and of sudden onset reaching maximum intensity in less than 1 minute.
 - B. Pain lasts 1 hour to 10 days.
 - C. Headache may recur within the first week after onset, but does not recur regularly over subsequent weeks or months.
 - D. Not attributed to another disorder; normal CSF and normal brain imaging are required.

Thunderclap headache may occur as a benign and potentially recurrent headache disorder in the absence of organic intracranial pathology. Before coining the term *thunderclap headache* in 1986, the same clinical syndrome was described under various terms such as *benign vascular headache*, *migrainous vasospasm*, and *crash migraine* (47,74,82). Although some patients with primary thunderclap headache may have a history of migraine, the clinical presentation and temporal profile of thunderclap headache is distinctive from migraine headache. The headache, which is sudden and reaches maximum intensity within 30 seconds, usually lasts several hours, but a persistent less severe headache may persist for weeks. Nausea, prostration, and recurrent emesis may be present simultaneously with or shortly after headache onset. The headache may be diffuse, but is often occipital in location. Repeated paroxysms of primary thunderclap headache may occur over a period of 7 to 14 days, and these may be spontaneous or provoked by minimal exertion or exercise (15). Primary thunderclap headache may recur over subsequent months to years in up to one-third of patients. These headaches may occur spontaneously when patients are at rest or during light activities, or may be precipitated by intense exertion (weight lifting), vigorous exercise, bathing in hot water, hyperventilation, or sexual activity (15,29,93).

Although the term *thunderclap headache* was used initially to describe a headache that had characteristics of an unruptured cerebral aneurysm (11), a number of other disorders may present with a headache that has features indistinguishable from primary thunderclap headache, including subarachnoid hemorrhage, cerebral venous sinus thrombosis, pituitary apoplexy, spontaneous

intracranial hypotension, hypertensive encephalopathy, posterior leukoencephalopathy, retroclival hematomas, Call-Flemming syndrome, and benign angiopathy of the central nervous system (4,7,12,17–19,40,51,73,75,77, 83,85). Thunderclap headache with reversible vasospasm has also been described as a complication of selective serotonin reuptake inhibitors (78). Therefore, a careful clinical evaluation and diagnostic assessment with CSF analysis and imaging of the brain and cerebral arteries and venous sinuses must be performed in all patients who present for the first time with a thunderclap headache (see Table 99-2). In this regard, primary thunderclap headache is truly a diagnosis of exclusion.

PROGNOSIS

Limited data suggest that primary thunderclap headache is a benign and self-limited syndrome. A benign outcome has been demonstrated in five prospective studies involving 296 patients with thunderclap headache who were followed for 1 to 3.5 years after a subarachnoid hemorrhage was excluded by at least a CSF analysis and CT brain scan (27,36,39,43,89). However, several of these studies have reported repeated thunderclap headache in 8 to 44%, days to years after the index event, indicating an individual susceptibility and the potential for this headache syndrome to recur over time. These studies also reported the development of migraine headache in up to one-third of patients with primary thunderclap headache, suggesting that these patients were at an increased risk of developing migraine headache in the future.

MANAGEMENT

Primary thunderclap headache is a self-limited syndrome and treatment consists of symptomatic analgesics if necessary. Because thunderclap headache has been associated with serotonergic drugs and reversible cerebral vasospasm, the safety of serotonin agonists (triptans or ergots) in this group of patients has not been evaluated and should avoided. Because of the potential for recurrence within the first 1 to 2 weeks, patients may be cautioned to avoid vigorous physical activity and sympathomimetic drugs. The majority do not experience recurrent headaches and are not at increased risk for serious neurologic events over months or years; therefore, patients can and should be reassured.

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