Chapter 106

Chronic Posttraumatic Headaches

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International Headache Society (IHS) codes and diagnoses:

5.2 Chronic posttraumatic headache
5.2.1 Chronic posttraumatic headache attributed to moderate or severe head injury
5.2.2 Chronic posttraumatic headache attributed to mild head injury
5.6.2 Chronic headache attributed to other head and/or neck trauma
5.7.2 Chronic postcraniotomy headache

World Health Organization (WHO) codes and diagnoses:

G44.3 Chronic posttraumatic headache
G44.30 Chronic posttraumatic headache attributed to moderate or severe head injury
G44.31 Chronic posttraumatic headache attributed to mild head injury
G44.88 Chronic headache attributed to other head and/or neck trauma
G44.30 Chronic postcraniotomy headache

Short description: Headache after head injury was described many centuries ago. Posttraumatic headache (PTHA) is a cardinal symptom of the "postconcussion syndrome" and may be accompanied by somatic, psychologic, or cognitive disturbances (15) (Table 106-1). PTHA is a new-onset headache resulting from brain, head, and sometimes neck injury, and can simulate the clinical characteristics of several primary headaches. Severe, moderate, and mild head injuries can cause PTHA (49).

Acute PTHA develops within 7 days after head trauma or regaining consciousness following head trauma and resolves within 3 months (22). If such headaches persist beyond the first 3 months postinjury, they are labeled chronic PTHA. It is easy to establish the relationship between a headache and head or neck trauma when the headache develops immediately or in the first days after trauma. On the other hand, such a relationship may be difficult to establish when headaches develop weeks or months after trauma.

Chronic PTHA could appear after mild, moderate, or severe head injury. Moderate or severe head injury is defined as head trauma with at least one of the following (22):

1. loss of consciousness for more than 30 minutes;
2. Glasgow Coma Scale (GCS) score of less than 13;
3. posttraumatic amnesia for more than 48 hours; or
4. imaging demonstration of a traumatic brain lesion (cerebral hematoma, brain contusion, or skull fracture).

Mild head injury (MHI) is defined as head trauma with all the following:

1. no loss of consciousness, or loss of consciousness of less than 30 minutes duration;
2. GCS *of more than or equal to* 13; and
3. symptoms or signs diagnostic of concussion.

The relationship between the severity of injury and headache is poorly defined. Surprisingly, the risk of developing chronic symptoms seems greater for mild or moderate head injury (9,57).

The mechanisms of PTHA are poorly understood; biologic, psychologic, and social factors have been invoked (33). Similarly, the pathophysiology of postconcussive symptoms is a matter of controversy (25). Neurologic factors have been implicated in the initial phase of chronic PTHA; psychologic and litigation factors are operant in the maintenance phase. Indeed, emotional, motivational, and premorbid personality factors contribute to chronic symptoms lasting 6 months and beyond and could explain some of the residual symptoms (30,57).

EPIDEMIOLOGY

Motor vehicle accidents are the most frequent cause of head injury (42%), and men 15 to 24 years old are at the highest risk. Other causes of head injury include falls (23%), assaults (14%), and sports accidents (6%) (58).

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TABLE 106-1 Features of the Postconcussion Syndrome

Symptoms	*Frequency*
Headache	>90%
Dizziness	>53%
Blurred vision	14%
Anosmia	5%
Photophobia	7%
Phonophobia	15%
Psychosomatic complaints	>85%
Fatigue	
Disturbed sleep	
Memory loss	
Poor concentration	
Impaired libido	
Apathy	
Anger	
Personality changes	
Depression	
Anxiety	
Irritability	

From Evans RW. The postconcussion syndrome and the sequelae of mild head injury. In: Evans RW, ed. *Neurology and trauma.* Philadelphia: WB Saunders, 1996:91–116.

Many of these patients suffer from posttraumatic syndrome and have additional somatic and neuropsychologic symptoms.

MHI accounts for more than 75% of brain injuries (26) and its annual incidence varies from 131 to 511 per 100,000 inhabitants (15). PTHA follows head trauma and whiplash injury in 30 to 90% of patients (14,25). The reported incidence of chronic PTHA also varies from one study to another, in part because of disagreement on the duration of symptoms from the time of injury. The new IHS diagnostic criteria define chronic PTHA when symptoms persist beyond 3 months of the original insult (22). Up to 32% of patients with head injury report persistent headaches 3 months after trauma, and approximately one in four continues to report headache at 4 years (4,6, 13,25).

The IHS criteria require the onset of PTHA to be within 1 week of head trauma (22). However, some studies indicate that late-acquired headaches (starting more than 2 weeks after the trauma) may be as common (6,29) or more common (10) than those of early onset. Until well-designed case-control studies are performed, it is difficult to ascertain that these "late-onset headaches" are, indeed, posttraumatic in nature.

PATHOPHYSIOLOGY

Posttraumatic syndrome is probably not a single pathologic entity but a group of traumatically induced disorders with overlapping symptoms. Headache is mainly a manifestation of brain dysfunction with occasional contributions from persistent musculoskeletal injuries. Axonal injury, soft tissue damage, cerebral metabolic derangements, and altered cerebral hemodynamics have been implicated in the genesis of symptoms, including headache, following head trauma.

Neck, jaw, and scalp tissue injuries may contribute to acute PTHA. Most of these injuries heal completely and cannot, by themselves, account for chronic PTHA. However, soft tissue or skeletal injuries may initiate or trigger a transformation process in headache-prone patients somewhat akin to the process of chronic migraine that evolves from an episodic disorder. Perhaps these injuries induce wind-up and sensitization, resulting in a permanently altered neuronal function (44).

Shear forces are applied to the brain during head injury, resulting in diffuse axonal insult, which varies in severity from functional abnormalities to widespread structural damage. Experimental and clinical data suggest that diffuse axonal injury occurs with MHI, but its extent and relevance remain elusive. A better understanding of the nature of neuroimaging abnormalities after MHI could shed light on the pathologic basis for the long-term neurologic disability that some patients with chronic PTHA develop. Indeed, newer magnetic resonance imaging (MRI) techniques (e.g., diffusion tensor imaging and magnetization transfer ratio) (1,23) may better delineate small parenchymal brain lesions, diffuse axonal injury secondary to disruption of axonal membranes, or delayed cerebral atrophy. Furthermore, magnetic resonance spectroscopy studies may detect metabolic brain changes (an early reduction in N-acetyl aspartate and an increase in choline compounds) that correlate with head injury severity (17,50). Lastly, positron emission tomography (PET), single-photon emission computed tomography (SPECT), and ^{133}Xenon inhalation techniques provide additional insight into brain perfusion abnormalities after MHI and chronic posttraumatic symptoms (45,52).

The mechanisms of PTHA may mirror those of primary headaches. Neurochemical changes that are observed in typical migraine and in experimental traumatic brain injury include excessive release of excitatory amino acids (primarily glutamate); increase in extracellular potassium, intracellular sodium, or calcium; and accumulation of serotonin. Also, changes in levels of catecholamines and endogenous opioids, decline in magnesium levels, abnormalities in nitric oxide formation, and alterations in neuropeptides are observed (41,48). Furthermore, patients with PTHA may develop a central sensitization phenomenon as a result of traumatic focal lesions (27). Finally, both central and peripheral sensitization have been proposed to occur in PTHA (5,31,39).

Many patients with posttraumatic migraine (PTMA), and some with tension-type PTHA, suffer frequent generalized headaches and analgesic overuse (54,55). Analgesic overuse may perpetuate chronic PTHA.

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To date, the cause of chronic PTHA remains elusive, but electrophysiologic, hemodynamic, and neuroimaging studies are indicating distinct pathophysiologic disturbances such as cortical dysfunction with resultant alteration in neuronal threshold for pain, axonal injury with subsequent dysregulation of brainstem nociceptive pathways, and unstable cerebral hemodynamics. It is thought that an interplay between the physical injury of the brain, however minimal, psychologic disturbances that are generated by the physical and emotional stresses of the accident and perpetuated by persistent individual concerns regarding the injury suffered and the ability to work, and the patient's premorbid disposition all contribute to chronic PTHA (15,19,33,42,49). These factors are compounded by the desire for financial compensation in some cases.

RISK FACTORS FOR CHRONIFICATION

One study indicated that incident headaches (i.e., before head trauma) were not a risk factor for a chronic PTHA (24). However, the prevalence of headache increased from 40% pretrauma to 64% after the injury, and the largest increase was in the cohort of patients reporting one or more headache days per week. Also, the study showed that more women (49%) than men (30%) develop chronic PTHA, in line with the results of an earlier study (6) but not concordant with a later study of 35 patients (56).

Older age was not a risk factor for chronic PTHA in one study (24) and was associated with slower and incomplete recovery in two subsequent ones (34,43).

A commonly held view is that chronic PTHA is more common in people who sustain mild injury compared with those who suffer a moderate or severe insult (14,36,42). Cartlidge and Shaw found that, among 372 patients, 34% with short-duration amnesia and 19% of those with prolonged amnesia reported headache at 6 months (6). At 1 year, the proportions were 21% in those with short-duration amnesia and 14% in the long-duration amnesia group. The numbers were 24% and 19%, respectively, at 2 years. Similar findings were reported in a later study where 42% of 48 patients who had not lost consciousness developed chronic PTHA, as compared to 38% of 74 patients who lost consciousness for less than 15 minutes and 23% of 30 patients who lost consciousness for 15 minutes to 24 hours. These studies are in contrast with an old report that indicated that headaches lasting longer than 2 months after accidents affected 10 to 20% of patients who were merely dazed by their trauma and 34% of 175 patients rendered unconscious (4).

Mechanical impact factors such as an abnormal position of the head (rotation or inclined) also increase the risk of PTHA (25,30,53). Other predictors of bad prognosis are low intellectual, educational, and socioeconomic level; history of alcohol abuse; presence of skull fracture; reduced value of GCS; elevated serum protein S-100B; and dizziness, headache, and nausea in the emergency room (11,46).

LITIGATION IN CHRONIC POSTTRAUMATIC HEADACHES

The role of litigation in the genesis and perpetration of PTHA remains highly debated. Generally, there is no firm evidence that litigation and economic expectation is associated with prolongation of headaches. Indeed, studies have shown that some patients improve when still in litigation, and conversely, the resolution of litigation or compensation claims do not coincide with headache resolution in others (14,38,56). These beliefs are not accepted universally since some authors contend that insurance and compensation claims have a large impact on recovery from posttraumatic and postwhiplash headaches (8). In support of this alternative hypothesis are (a) observations of self-limiting and brief symptoms after an acute whiplash injury in a country where there is no preconceived notion of chronic pain arising from rear-end collision, (b) insurance companies or litigation (16,37), and (c) no clear evidence that acute symptoms evolve into the so-called late whiplash syndrome (7,47). However, it can be argued that intensive and early treatment of these patients may be the main factor in preventing more frequent chronic posttraumatic symptoms rather than the absence of litigation issues (3).

CLINICAL PRESENTATION

The revised criteria for the diagnosis of chronic posttraumatic headache (Revised International Classification for Headache Disorders–ICHD-II) are as follows:

5.2.1 Chronic posttraumatic headache with moderate or severe head injury

A. New headache appearing after head trauma and fulfilling criteria B through D

B. Head trauma with at least one of the following:
- **1.** Loss of consciousness >30 minutes
- **2.** GCS <13
- **3.** Posttraumatic amnesia >48 hours
- **4.** Imaging demonstration of a traumatic brain lesion (cerebral hematoma, brain contusion, or skull fracture)

C. Headache occurs <7 days after head trauma or after regaining consciousness or memory.

D. Headache lasts >3 months after regaining consciousness or memory.

5.2.2 Chronic posttraumatic headache with mild head injury

A. New headache appearing after head trauma and fulfilling criteria B through D.

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B. Head trauma with all of the following:
  1. No loss of consciousness, or loss of consciousness of <30 minutes duration
  2. GCS ≥13
  3. Symptoms or signs diagnostic of concussion
C. Headache occurs <7 days after head trauma or after regaining consciousness or memory.
D. Headache lasts >3 months after regaining consciousness or memory.

A variety of headache patterns occur after head injury and may closely resemble primary headache disorders (18), particularly tension-type headache (TTH) (10). In one of the first prospective studies on chronic PTHA, 48 patients and an equal number of controls were followed (20). Of those patients with PTHA, 75% had TTH, 21% had migraine without aura, and 4% had an unclassified headache. Other prospective, but uncontrolled, studies showed that the headache pattern after head injury may be tension-type, cervicogenic, or migrainous in the majority (2,12).

PTMA varies in incidence between 3% and 28% of all cases of PTHA and usually it is a migraine without aura. PTMA often occurs in children, adolescents, and young adults with familial history of migraine but without personal history of previous headache (15,20,32). In other words, trauma acts as a triggering factor of migraine (15,21) and family history is a predisposing factor. Migraine with visual or sensory aura, dizziness, or mental confusion have been described rarely (18,56).

Clusterlike headache, supraorbital neuralgia, occipital neuralgia, exertional headache, and headache associated with sexual activity have been described occasionally (2,15,20,41).

PTHA is diagnosed only when the headache develops within 7 days of head trauma or regaining consciousness following head trauma (12,53).

Mild, moderate, and severe head injuries can be associated with PTHA. Clinical quantification of traumatic brain injury patients should be based on admission GCS; presence or absence of loss of consciousness; duration of unconsciousness, if any; presence or absence of PTA; and any focal neurologic findings. Nausea and dizziness frequently occur with head trauma and may persist for weeks to months postinjury. Patients with chronic PTHA continue to report postconcussion symptoms, which might indicate that the headache is a manifestation of a more generalized posttraumatic syndrome.

MANAGEMENT STRATEGIES

Trauma-induced headaches are usually heterogeneous in nature, including episodic tension-type migraine and with time, chronic daily headache. Anecdotal evidence supports the notion that aggressive and early treatment may prevent chronicity. Delayed recovery from PTHA may be a result of inadequate or ineffective treatment, overuse of analgesic medications resulting in analgesic-rebound phenomena, or comorbid psychiatric disorders (posttraumatic stress disorder, insomnia, substance abuse, depression, or anxiety) (28).

In general, therapeutic strategies are aimed at symptomatic treatment of the particular headache type that develops after trauma (i.e., migraine, tension-type). Treatment of acute exacerbations is necessary and includes antiinflammatory agents, ergotamine, or triptans. More importantly, the use of prophylactic therapy in the early phase after head trauma may prevent chronification. Amitriptyline or propanolol, used alone or in combination, and verapamil improve all symptoms of the postconcussive syndrome, especially migraine (36,49,51). Also, very good results may be achieved with divalproex sodium (40). Physical therapy, biofeedback, appropriate educational support (42), and cutaneous stimulation are good complementary measures, especially in patients with risk factors for poor prognosis (15,35). Sometimes, a peripheral trigger can be identified and targeted specifically, which could resolve the pain (27). Finally, management strategies should address cognitive difficulties and neuropsychiatric symptoms (e.g., depression, anxiety) that coexist in many patients with chronic PTHA.

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