

Post-traumatic headaches

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Headaches as a result of head trauma are one of the most common secondary headache types. Because of the medicolegal aspects, post-traumatic headaches also are one of the most controversial headache topics, and, for many physicians, one of their least favorite types to treat. This article reviews the postconcussion syndrome (PCS) and post-traumatic headaches.

The postconcussion syndrome

PCS refers to a large number of symptoms and signs that may occur alone or in combination, usually after mild head trauma [1]. Concussion is a trauma-induced alteration in mental status that may or may not involve loss of consciousness [2]. The patient's account of loss of consciousness and duration may not be reliable. Loss of consciousness does not have to occur for PCS to develop.

The following symptoms and signs are associated with PCS, which develops in more than 50% of patients who have mild head injuries [3]: headaches, dizziness, vertigo, tinnitus, hearing loss, blurred vision, diplopia, convergence insufficiency, light and noise sensitivity, diminished taste and smell, irritability, anxiety, depression, personality change, fatigue, sleep disturbance, decreased libido, decreased appetite, posttraumatic stress disorder, memory dysfunction, impaired concentration and attention, slowing of reaction time, and slowing of information processing speed (Box 1). Headaches, dizziness, fatigue, irritability, anxiety, insomnia, loss of concentration and memory, and noise sensitivity are the most common complaints [4]. PCS may be subdivided into early PCS and a late or persistent PCS, which is when symptoms and signs persist for more than 6 months [5].

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Historical aspects of postconcussion syndrome

PCS has been controversial for more than 135 years [6,7] Erichsen, a London surgeon, beginning with a series of lectures in 1866, opined that minor injuries to the head could result in severe disability as a result of “subacute cerebral meningitis and arachnitis” [8]. Symptoms reported by these patients included headaches, memory complaints, nightmares, irritability, and light and noise sensitivity. Erichsen was defensive about these cases of cerebral concussion because many occurred after railway accidents in which litigation was involved. On the title page of his book, he quotes Montaigne, “Je raconte, je ne juge pas” (“I tell, I do not judge”). These injuries became known as “railway brain” and those of the spine as “railway spine.” He pointed out that earlier investigators had described the same symptoms in the prerailway era. He also was concerned about misdiagnosing these cases as hysteria: “Hysteria is the disease for which I have more frequently seen concussion of the spine, followed by meningo-myelitis, mistaken, and it certainly has always appeared extraordinary to me that so great an error of diagnosis could so easily be made).”

Railway spine and brain became topics of intense controversy. In 1879, Rigler [9] raised the important issue of compensation neurosis when he described the increased incidence of post-traumatic invalidism after a system

Box 1. Sequelae of mild head injury

Headaches

- Muscle contraction or tension type
- Cranial myofascial injury
- Secondary to neck injury (cervicogenic)
 - Myofascial injury
 - Intervertebral discs
 - Cervical spondylosis
 - C2-3 facet joint (third occipital headache)
- Secondary to temporomandibular joint injury
- Greater and lesser occipital neuralgia
- Migraine with and without aura
- Footballer’s migraine
- Medication rebound
- Cluster
- Supraorbital and infraorbital neuralgia
- Resulting from scalp lacerations or local trauma
- Dysautonomic cephalgia
- Orgasmic cephalgia
- Carotid or vertebral artery dissection
- Subdural or epidural hematomas
- Hemorrhagic cortical contusions

Low cerebrospinal fluid pressure syndrome
Hemicrania continua
Mixed

Cranial nerve symptoms and signs

Dizziness
Vertigo
Tinnitus
Hearing loss
Blurred vision
Diplopia
Convergence insufficiency
Light and noise sensitivity
Diminished taste and smell

Psychologic and somatic complaints

Irritability
Anxiety
Depression
Personality change
Post-traumatic stress disorder
Fatigue
Sleep disturbance
Decreased libido
Decreased appetite
Initial nausea or vomiting

Cognitive impairment

Memory dysfunction
Impaired concentration and attention
Slowing of reaction time
Slowing of information processing speed

Rare sequelae

Subdural and epidural hematomas
Cerebral venous thrombosis
Second impact syndrome
Seizures
Nonepileptic post-traumatic seizures
Transient global amnesia
Tremor
Dystonia

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of financial compensation was established for accidental injuries on the Prussian railways in 1871. In 1888, Strumpell discussed how the desire for compensation could lead to exaggeration. In 1889, Oppenheim popularized the concept of traumatic neurosis, in which a strong afferent stimulus resulted in impairment of function of the central nervous system. Charcot countered Oppenheim's work and suggested that the impairment described actually was the result of hysteria and neurasthenia.

PCS also was controversial throughout the twentieth century. Miller, in 1961, summarized the viewpoint of those who believed that PCS actually was a compensation neurosis: "The most consistent clinical feature is the subject's unshakable conviction of unfitness for work" [10]. In 1962, Symonds took an equally strong opposing viewpoint: "It is, I think, questionable whether the effects of concussion, however slight, are ever completely reversible" [11].

Epidemiology

Head trauma is a cause of significant morbidity and mortality in all societies. Mild head injury accounts for 75% or more of all brain injuries [12]. The annual incidence of mild head injury per 100,000 population has been estimated at 149 for Olmsted County, Minnesota [13], 131 for San Diego County, California [14], and 511 for Auckland, New Zealand [15]. The incidence of mild head injury may be as high as 640 persons per 100,000 population, however, because many cases are unreported [16]. In addition, some patients may have hidden traumatic brain injury, where they develop PCS but do not make the causal connection between the injury and its consequences [17].

In an industrialized country, such as the United States, the relative causes of head trauma are approximately as follows: motor vehicle accidents, 45%; falls, 30%; occupational accidents, 10%; recreational accidents, 10%; and assaults, 5% [18]. Approximately one half of all patients who have mild head injury are between the ages of 15 and 34. Motor vehicle accidents are more common in the young and falls more common in the elderly [19]. Men are injured more frequently than women, with a 2:1 ratio. Approximately one half of all patients who have mild head injury are between the ages of 15 and 34. Approximately 20% to 40% of people who have mild head injuries in the United States do not seek treatment.

Headaches

Headaches are estimated as occurring variably in 30% to 90% of persons who are symptomatic after mild head injury [20]. Paradoxically, headache prevalence and lifetime duration is greater in those who have mild head injury compared with those who have more severe trauma [21]. Post-traumatic headaches are more common in those who have a history of headache [22].

Patients who sustain iatrogenic trauma when undergoing a craniotomy for brain tumor (other than acoustic neuroma) or intractable epilepsy often have a self-limited combination of tension-type and site-of-injury headache, if any headache at all [23]. Three months after removal of a vestibular schwannoma via the retrosigmoid approach, however, 34% of patients still complain of severe headaches.

According to the International Headache Society criteria, the onset of the headache should be less than 7 days after the injury [24]. The less than 7-day onset is arbitrary, particularly because the etiology of post-traumatic migraine is not understood. For example, post-traumatic epilepsy may have a latency of months or years. Similarly, it would not be surprising if there were a latency of weeks or months for post-traumatic migraine to develop. Conversely, because migraine is a common disorder, the longer the latency between the trauma and onset, the more likely the trauma may not have been causative. Consider the hypothetical case of a 27-year-old male who develops new-onset migraine 2 months after a mild head injury in a motor vehicle accident. The incidence of migraine in males under the age of 30 is 0.25% per year or, in this case, 0.042% per 2 months [25]. Was the new-onset migraine the result of the mild head injury or coincidence? Three months seems a more reasonable latency for onset than does 7 days [26].

Many patients have more than one type of headache or have headaches with tension and migraine features. Neck injuries commonly accompany head trauma and can produce headaches. Headaches commonly are associated with whiplash injuries [27,28], which are beyond the scope of this review. Although not part of PCS, headaches associated with subdural and epidural hematomas also are described.

Tension-type headache

Eighty-five percent of post-traumatic headaches are tension type. These headaches occur in a variety of distributions, including generalized, nuchal-occipital, bifrontal, bitemporal, cap-like, or headband. The headache, which may be constant or intermittent with variable duration, usually is described as pressure, tight, or dull aching. The headache may be present on a daily basis. Temporomandibular joint injury can be caused by either direct trauma or jarring associated with the head injury. Patients may complain of jaw pain and hemicranial or ipsilateral frontotemporal aching or pressure headaches.

Occipital neuralgia

This term is in some ways a misnomer because the pain is not necessarily from the occipital nerve and usually does not have a neuralgic quality. Greater occipital neuralgia is a common post-traumatic headache and also is seen frequently without injury. The aching, pressure, stabbing, or throbbing pain may be in a nuchal-occipital or parietal, temporal, frontal, or periorbital or retroorbital distribution. Occasionally, a true neuralgia may

be present with paroxysmal shooting-type pain. The headache may last for minutes, hours, or days and be unilateral or bilateral. Lesser occipital neuralgia similarly can occur with pain generally referred more laterally over the head.

The headache may be the result of an entrapment of the greater occipital nerve in the aponeurosis of the superior trapezius or semispinalis capitis muscle or instead be referred pain without nerve compression from trigger points in these or other suboccipital muscles. Digital pressure over the greater occipital nerve at the mid-superior nuchal line (halfway between the posterior mastoid and the occipital protuberance) reproduces the headache. Pain referred from the C2-3 facet joint (third occipital headache) [29] or other upper cervical spine pathology and posterior fossa pathology, however, may produce a similar headache.

Migraine

Recurring attacks of migraine with or without aura can result from mild head injury. Impact also can cause acute migraine episodes in adolescents who have a family history of migraine. This originally was termed “footballer’s migraine” to describe headaches in young men who play soccer who had multiple migraines with aura attacks triggered only by impact [30]. Similar attacks can be triggered by mild head injury in any sport. The most famous example involves the running back of the Denver Broncos and was witnessed by hundreds of millions of people around the world during the 1998 Super Bowl. Terrell Davis, who had pre-existing migraine, developed a migraine with aura after a ding on the head at the end of the games’ first quarter. After successfully using dihydroergolamine (DHE) nasal spray, he was able to return for the third quarter, scored the winning touchdown, set a Super Bowl rushing record, and was voted “Most Valuable Player.”

After minor head trauma, children, adolescents, and young adults can develop a variety of transient neurologic sequelae that are not always associated with headache and are perhaps the result of vasospasm. Five clinical types can cause the following: hemiparesis; somnolence, irritability, and vomiting; a confusional state [31]; transient blindness, often precipitated by occipital impacts; and brainstem signs [32,33].

Cluster headaches

Cluster headaches rarely result from mild head injuries.

Supraorbital and infraorbital neuralgia

Injury of the supraorbital branch of the first trigeminal division as it passes through the supraorbital foramen just inferior to the medial eyebrow can cause supraorbital neuralgia. Similarly, infraorbital neuralgia can result

from trauma to the inferior orbit. Shooting, tingling, aching, or burning pain along with decreased or altered sensation and sometimes decreased sweating in the appropriate nerve distribution may be present. The pain can be paroxysmal or fairly constant. A dull aching or throbbing pain also may occur around the area of injury.

Scalp lacerations and local trauma

Dysesthesias over scalp lacerations occur frequently. In the presence or absence of a laceration, an aching, soreness, tingling, or shooting pain over the site of the original trauma can develop. Symptoms may persist for weeks or months but rarely for more than 1 year.

Subdural hematomas

Tearing of the parasagittal bridging veins (which drain blood from the surface of the hemisphere into the dural venous sinuses) leads to hematoma formation within the subdural space. Even minor injuries without loss of consciousness, such as bumps on the head or riding a roller coaster [34], can result in this tearing. Falls and assaults are more likely to cause subdural hematomas than motor vehicle accidents.

Subdural hematomas usually are located over the hemispheres, although other locations, such as between the occipital lobe and tentorium cerebelli or between the temporal lobe and base of the skull, can occur. A subdural hematoma becomes subacute between 2 and 14 days after the injury when there is a mixture of clotted and fluid blood and becomes chronic when the hematoma is filled with fluid more than 14 days after the injury. Rebleeding can occur in the chronic phase. Most patients who have chronic subdural hematomas are late middle aged or elderly. Subdural hematomas can be present with a normal neurologic examination.

Headaches associated with subdural hematomas are nonspecific, ranging from mild to severe and paroxysmal to constant [35]. Unilateral headaches usually are the result of ipsilateral subdural hematomas. Headaches associated with chronic subdural hematomas have at least one of the following features in 75% of cases: sudden onset; severe pain; exacerbation with coughing, straining, or exercise; and vomiting and or nausea.

Epidural hematomas

Bleeding into the epidural space from a direct blow to the head produces an epidural hematoma. The source of the bleeding is variable and can be arterial or venous or both. In the supratentorial compartment, bleeding is of the following origins: middle meningeal artery, 50%; middle meningeal veins, 33%; dural venous sinus, 10%; and other sources, including

hemorrhage from a fracture line, 7%. Most epidural hematomas in the posterior fossa are to the result of dural venous sinus bleeding. The locations of epidurals are as follows: temporal region (usually under a fractured squamous temporal bone), 70%; frontal convexity, 15%; parieto-occipital, 10%; and parasagittal or posterior fossa, 5%. Ninety-five percent of epidurals are unilateral.

Epidural hematomas usually occur between the ages of 10 and 40 and much less frequently in those under 2 or over 60. Motor vehicle accidents or falls are the most common causes. Trivial trauma without loss of consciousness can be a cause.

Forty percent of patients who have an epidural hematoma present with a Glasgow Coma Scale of 14 or 15. Less than one third of patients have the classic lucid interval (initially unconscious, then recovery, and then unconscious again).

Up to 30% of epidural hematomas are of the chronic type [36]. The patient often is a child or young adult who sustains what seems to be a trivial injury often without loss of consciousness [37]. A persistent headache then develops, often associated with nausea, vomiting, and memory impairment, which might seem consistent with PCS. After the passage of days to weeks, focal findings develop. The headaches of acute and chronic epidural may be unilateral or bilateral and can be nonspecific.

Low cerebrospinal fluid pressure headache

Trauma can cause a CSF leak through a dural root sleeve tear or a cribriform plate fracture and result in a low CSF pressure headache with the same features as a post-lumbar puncture headache [38].

Dysautonomic cephalgia

Dysautonomic cephalgia is a rare headache resulting from injury of the anterior triangle of the neck or carotid sheath. Acute local pain and tenderness in the anterior triangle can be followed weeks or months later by severe unilateral frontotemporal headache, ipsilateral increased sweating of the face, dilation of the ipsilateral pupil, blurred vision, ipsilateral photophobia, and nausea. The headache can occur a few times per month and last hours to days.

Other types

New-onset orgasmic cephalgia can follow mild head trauma within 3 to 4 weeks. Hemorrhagic cortical contusions can cause a headache resulting from subarachnoid hemorrhage. Rare types of posttraumatic headaches can be the result of carotid and vertebral artery dissections and hemicrania continua [39].

Pathophysiology

Mild head injury (MHI) may result in cortical contusions after coup and contre-coup injuries or diffuse axonal injury resulting from sheer and tensile strain damage [40]. Release of excitatory neurotransmitters, including acetylcholine, glutamate, and aspartate, may be a neurochemical substrate for MHI. Impairment in cerebral vascular autoregulation can occur. Neuroimaging studies, including MRI, single photon emission computerized tomography (SPECT), positron emission tomography (PET), and magnetic source imaging, can demonstrate structural and functional deficits [41]. Although these findings may help to explain cognitive deficits, the etiology of post-traumatic migraine is poorly understood.

Treatment of headaches

There is a dearth of randomized placebo-controlled trials of medications for post-traumatic headaches. There are anecdotal reports of tension- and migraine-type headaches treated with the usual symptomatic and preventative medications used for chronic daily headaches and migraine. The physician should be concerned about the potential for medication rebound headaches with the frequent use of over-the-counter medications, such as acetaminophen, aspirin, and combination products containing caffeine, and prescription drugs containing narcotics, butalbital, and benzodiazepines. Habituation also is a concern with narcotics, butalbital, and benzodiazepines. Post-traumatic chronic daily headache may respond to an intravenous DHE regimen. Botulinum toxin injections also may be beneficial for post-traumatic headaches [42].

Occipital neuralgia may improve with local anesthetic nerve blocks, which are effective alone or combined with an injectable corticosteroid (eg, 3 mL of 1% xylocaine or 2.5 mL of 1% xylocaine and 3 mg of betamethasone). Before injection, the physician should aspirate to avoid inadvertent injection into the occipital or vertebral artery. Nonsteroidal anti-inflammatory drugs and muscle relaxants may also be beneficial. If there is a true occipital neuralgia with paroxysmal lancinating pain, baclofen, carbamazepine, tizanidine, or gabapentin may help. Physical therapy and transcutaneous nerve stimulators may help some headaches.

Education

One of the most important roles of the physician is education of the patient and family members, other physicians, and when appropriate, employers, attorneys, and representatives of insurance companies. There is widespread ignorance about the potential effects of mild head injury because of what Evans has termed “the Hollywood head injury myth” [43]. Patients

complaints of chronic daily headaches of any type, especially post-traumatic, often are met with skepticism by much of the public, who cannot imagine that headaches occur with such frequency.

Most people's knowledge of the sequelae of mild head injuries largely is the result of movie magic. Some of the funniest scenes in slapstick comedies and cartoons depict the character sustaining single or multiple head injuries, looking dazed, and then recovering immediately. In cowboy movies, action and detective stories, and boxing and martial arts films, seemingly serious head trauma often is inflicted by blows from guns and heavy objects, motor vehicle accidents, falls, fists, and kicks, all without lasting consequences. Our experience is minimal compared with the thousands of simulated head injuries seen in the movies and on television.

The physician can provide education by summarizing the literature and using vivid examples from sports. The public is familiar with dementia pugilistica, or punch-drunk syndrome, of cumulative head injury in boxers. The examples of Joe Louis and Muhammad Ali are well known. Many have witnessed powerful punches resulting in dazed, disoriented boxers or knock-outs. There also is growing awareness of the effects of cumulative concussions in professional football (eg, quarterbacks Steve Young, Troy Aikman, and Stan Humphries) and hockey (eg, Pat Lafontaine). Reports of headaches preventing athletes from returning to play now make the sports pages.

Prognosis

The percentage of patients who have headaches at 1 month varies from 31.3% [41] to 90% [44]; at 3 months from 47% [45] to 78% [46]; and at 1 year from 8.4% [47] to 35% [44]. Twenty-four percent of patients have persisting headaches at 4 years [48].

Effect of litigation

Patients who have litigation are similar to those who do not in the following respects: symptoms that improve with time [49], types of headaches [27], cognitive test results [49], and response to migraine medications [50]. Symptoms usually do not resolve with the settlement of litigation [51]. Pending litigation may increase the level of stress for some claimants and may result in increased frequency of symptoms after settlement. Skepticism of physicians also may accentuate the level of stress and compel some patients to exaggerate so that the doctors take them seriously.

There certainly are, however, some patients who have persistent complaints resulting from secondary gain [52], malingering, and psychologic disorders. Potential indicators of malingering after mild head injury include the following: premorbid factors (antisocial and borderline personality traits, poor work record, and prior claims for injury); behavioral characteristics (uncooperative evasive or suspicious); neuropsychologic test performance

(missing random items, giving up easily, inconsistent test profile, or stating frequently, “I don’t know”); postmorbidity complaints (describing events surrounding the accident in great detail or reporting an unusually large number of symptoms); and miscellaneous items (engaging in general activities not consistent with reported deficits, having significant financial stressors, resistance, and exhibiting a lack of reasonable follow-through on treatments) [53].

In a study of mild head injured litigants, Andrikopoulos compared 72 patients who had no improvement or worsening headache with 39 patients who had improving headache [54]. Those who had no improvement or worsening performed worse on cognitive tests and had greater psychopathology on the MMPI-2 than those who had improving headaches, suggesting the possibility of malingering.

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