Posttraumatic Headaches in Civilians, Soldiers, and Athletes

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KEYWORDS

• Headaches • Posttraumatic • Migraine • Tension-type • Occipital neuralgia • Soldiers • Athletes

KEY POINTS

- Mild head injury accounts.
- Headache is the most common symptom of the postconcussion syndrome (PCS) and develops in more than 50% of those who sustain mild head injuries.
- Tension, migraine, and occipital neuralgia are the most common types.
- There are few randomized placebo-controlled trial for treatment.

Headaches as a result of head trauma are one of the most common secondary headache types. Because of the medicolegal aspects, posttraumatic headaches have been one of the most controversial headache topics, and, for many physicians, one of their least favorite types to treat. In the past decade, however, there has been increasing interest in posttraumatic headaches among physicians and the public because of the headaches occurring in US soldiers with blast trauma and in athletes with concussions. This article reviews the PCS and posttraumatic 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 clinical syndrome of biomechanically induced alteration of brain function, typically affecting memory and orientation, that may or may not involve loss of consciousness.[2] A 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: headaches, dizziness, vertigo, tinnitus, hearing loss, blurred vision, diplopia, convergence insufficiency, light and noise sensitivity, diminished taste and smell, irritability, anxiety, depression, personality

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Neurol Clin 32 (2014) 283–303
http://dx.doi.org/10.1016/j.ncl.2013.11.010 **neurologic.theclinics.com**
0733-8619/14/$ – see front matter

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**).[3] Headaches, dizziness, fatigue, irritability, anxiety, insomnia, loss of concentration and memory, and noise sensitivity are the most common complaints.[4]

In a study of 118 patients who sustained a mild traumatic brain injury (TBI), symptoms were reported 1 month after the injury in the following percentages of patients: fatigue, 91%; headaches, 78%; forgetfulness, 73%; sleep disturbance, 70%; anxiety, 63%; irritability, 62%; dizziness, 59%; noise sensitivity, 46%; and light sensitivity, 44%.[5] PCS may be subdivided into early PCS and late or persistent PCS, which is when symptoms and signs persist for more than 6 months.[6] Among 53 patients referred to a headache clinic with chronic posttraumatic headaches, approximately half had cognitive complaints, a quarter had psychological complaints, and 17% had an isolated complaint of headache.[7]

HISTORICAL ASPECTS OF POSTCONCUSSION SYNDROME

PCS has been controversial for more than 150 years.[8,9] 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."[10] 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[11] raised the important issue of compensation neurosis when he described the increased incidence of posttraumatic invalidism after a system 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."[12] 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."[13]

In a survey performed among neurologists in the United States in 1992,[14] 25% believed that prolonged postconcussion symptoms were likely psychogenic in origin rather than due to any true pathology and 35% agreed that effective treatment of PCS was available. One respondent opined, "I am appalled at the number of groundless personal injury patients I see. Pain seems to occupy the position that

Box 1
Sequelae of mild head injury

- Headaches
  - Tension type
  - Migraine with and without aura
  - Medication overuse
  - Trigeminal autonomic cephalalgias
  - Hemicrania continua
  - Occipital neuralgia
  - C2-3 facet joint
  - Cervicogenic
  - Supraorbital and infraorbital neuralgia
  - Scalp lacerations and local trauma
  - Temporomandibular joint
  - Subdural or epidural hematomas
  - Low CSF pressure syndrome
  - Hemorrhagic cortical contusions
  - Carotid and vertebral artery dissections
  - Cerebral venous thrombosis
  - Carotid-cavernous fistula
- 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
  - Posttraumatic 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 posttraumatic seizures
Transient global amnesia
Tremor
Dystonia

From Evans RW. Post-concussion syndrome. In: Evans RW, Baskin DS, Yatsu FM, editors. Prognosis of neurologic disorders. 2nd edition. New York: Oxford University Press; 2000. p. 367; with permission.

hysteria did seventy years ago." Another, "Lawyers who focus in on these 'reported' injuries are causing a collapse in our medical and medico-legal systems."

EPIDEMIOLOGY OF TRAUMATIC BRAIN INJURY

Civilians

Traumatic brain injury is a cause of significant morbidity and mortality worldwide with approximately 54 to 60 million injuries annually.[15] Mild head injury accounts for 80% or more of all TBIs.[15] The Centers for Disease Control estimates that 1.4 to 3.8 million concussions occur per year in the United States,[16] resulting in more than 800,000 outpatient visits (most primary care) and 1.2 million emergency department visits for minor head injury or concussion.[17]

The annual incidence of mild head injury per 100,000 in the United States is between 100 and 300 (in a meta-analysis)[18] and 749 for New Zealand (95% of all brain injury cases).[15] The annual incidence may be as high as 600 per 100,000 in the United States[18] because many cases are unreported.[19] In addition, some patients may have hidden TBI, where they develop PCS but do not make the causal connection between the injury and its consequences.[20]

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%.[21,22] 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. Men are injured more frequently than women, with a 2:1 ratio.[23] 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.

US Military

Approximately 20% of veterans of Operations Enduring Freedom (Afghanistan) and Iraqi Freedom sustained a TBI, the signature wound of the conflicts.[24] According to the Congressional Research Service,[25] "Of the total 253,330 TBI cases between January 1, 2000, and August 20, 2012, 194,561 have been mild, 42,083 have been moderate, 6476 have been severe or penetrating, and 10,210 have not been classifiable." Blast exposure is the most common mechanism of injury, contributing to 75% of mild TBI.[26]

Sports

In the United States, 1.6 to 3.8 million persons per year sustain sport-related mild TBI, with many not obtaining immediate medical attention.[27] The incidences of concussion among high school and college football players per 1000 games were 1.55 and 3.02, respectively.[28] Female players have more concussions in high school and college basketball and soccer (highest rates) compared with male players. The rates per 1000 games for soccer are as follows: high school male players, 0.59; high school female players, 0.97; college male players, 1.38; college female players, 1.80.

Postcraniotomy

Iatrogenic trauma may also cause headaches. In the only prospective study of patients for the risk of headaches after treatment of intracranial aneurysms followed for 4 months after the procedure, the incidence of headache was 28 of 51 cases (54.9%) after surgery compared with 12 of 47 cases (25.5%) after embolization.[29] Less than a third had persistent headaches for more than 3 months. In another study of postcraniotomy headaches during the 6 months after craniotomy for the treatment of supratentorial intracranial aneurysms, there was an incidence of postcraniotomy headache of 40%, with 30% having migrainous headaches at 6 months.[30]

In a study of patients undergoing supratentorial craniotomy for epilepsy, 11.9% had ongoing headaches 1 year after surgery with 4% medically uncontrolled.[31] In another study of 107 patients who underwent craniotomies for brain tumors or epilepsy, no patients had debilitating headaches.[32] In a meta-analysis of 1653 patients who underwent resection of acoustic neuromas, long-term significant headaches were reported by 36% of those who underwent a retrosigmoid approach compared with 16% and 1% of those who underwent translabyrinthine and middle fossa approaches, respectively.[33] Some patients with chronic headaches after acoustic nerve resection have occipital nerve injuries improving after excision of the greater and lesser occipital nerves.[34] There is a single case report of a patient with new-onset hemicrania continua 2 days after resection of a large left-sided acoustic neuroma with complete resolution of headache on indomethacin (50 mg twice daily).[35]

HEADACHES

Headaches are estimated as occurring variably in 30% to 90% of persons who are symptomatic after mild head injury.[36] Head and neck injury account for approximately 15% of chronic daily headaches.[37] Paradoxically, headache prevalence and lifetime duration is greater in those who have mild head injury compared with those who have more severe trauma.[38,39] Posttraumatic headaches are more common in those who have a history of headache.[40]

Time of Onset

According to International Headache Society criteria, the onset of the headache should be less than 7 days after the injury.[41] The less than 7-day onset is arbitrary,

particularly because the cause of posttraumatic migraine is not understood. For example, posttraumatic 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 posttraumatic 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 man who develops new-onset migraine 2 months after a mild head injury in a motor vehicle accident. The incidence of migraine in men under the age of 30 is 0.25% per year or, in this case, 0.042% per 2 months.[42] Was the new-onset migraine the result of the mild head injury or coincidence?

Consider the increased incidence of new or worse headaches found with onset after 7 days at 3-month assessment in 3 studies. In a prospective study of 212 subjects hospitalized with mild TBI for observation or other injuries, an additional 59% of subjects reported new or worse headache (compared with preinjury) at 3 months who had not previously reported headache within the first 7 days after injury (baseline assessment).[43] Two other studies found a high percentage of new-onset headache with onset after 7 days: 23% additional headaches at the 3-month assessment in consecutive patients with moderate to severe TBI[44] and 19% in a retrospective cohort of US Army soldiers.[45] Three months seems a more reasonable latency for onset than 7 days[43,46] although a small percentage of patients with new-onset primary headaches are misdiagnosed as having posttraumatic headaches.

Epidemiology of Phenotypes

Civilians

In a meta-analysis[47] of 5 studies of posttraumatic headaches,[7,48–51] most headaches were of the tension type (ranging from 6.9% to 85.7%, mean 33.6%) and the second-most type had migraine characteristics (ranging from 1.9% to 40.7%, mean 28.6%). The following features were present: mild to moderate intensity pain, approximately 60%; bilateral, 72.5%; nonthrobbing, 83%; light sensitivity, 35.8%; noise sensitivity, 29.1%; and aggravation by routine physical activities, 71.1%. Analgesic overuse was reported as present in 18.8% to 45.8%. The percentage of mixed or unclassifiable headaches ranged from 4.2% to 36.5%.

In a prospective 1-year study of 212 subjects that included headaches with onset at any time, up to 49% of headaches met criteria for migraine and probable migraine, up to 40% met tension-type criteria, 4% were cervicogenic, and up to 16% were unclassified.[43] Of the up to 27% of subjects who reported having headaches several times per week to daily, 62% of the headache types were migraine in this highest frequency group at 1 year. Individuals over age 60 were significantly more likely to report no headaches over time and at all time points.

In another prospective 1-year study (that included headaches with onset at any time) of 378 subjects who sustained moderate to severe TBIs, migraine occurred in up to 38%, probable migraine in up to 25%, tension-type headache in up to 21%, cervicogenic headache in up to 10%, and unclassifiable headache in up to 30%.[52] Women were more likely to have preinjury migraine than men and to have migraine or probable migraine at all time points after injury.

US military

Migraine is the most common type of posttraumatic headache, occurring in 60% to 97% of cases.[53] Blast trauma had been sustained by 77% of soldiers with chronic posttraumatic headaches.[54] The onset of posttraumatic headaches after injury was within 1 week for nearly 40%, within 1 month for 20%, and beyond 1 month for 40%.[55]

There was a high prevalence of migraine in US soldiers deployed to combat in Iraq without physical trauma, with 17.4% of men and 34.9% of women reporting a headache consistent with migraine during the prior year, much greater than a civilian population.[56]

Athletes

There are few studies describing the types of headaches among athletes. In a study of 296 student athletes ages 12 to 25 years who sustained sport-related concussions, migraines occurred in 52, headache in 176, and no headache in 68.[57] Female athletes were 2.13 times more likely than male athletes to report posttraumatic migraine characteristics. Those with migraine characteristics had prolonged symptom recovery, including cognitive, neurobehavioral, and somatic symptoms. Only 1 patient reported migraine at 90 days. Another study of high school and college athletes found that those with posttraumatic migraines had significantly greater neurocognitive deficits compared with those who had concussions with nonmigraine headaches and controls.[58]

Possible overdiagnosis of migraine

Tension-type, cervicogenic headaches, and occipital neuralgia have the potential for being misdiagnosed as migraine because light and noise sensitivity are commonly associated with PCS[5] and nausea may also be present in early PCS and in those with associated dizziness.

Neck injuries commonly accompany head trauma and can produce headaches, such as those associated with whiplash injuries,[59] discussion of which is beyond the scope of this review. Although not part of PCS, headaches associated with subdural and epidural hematomas also are described.

CASES

Case 1. Migraine from Blast Trauma

A 39-year-old US male army soldier was standing outside of his truck in Afghanistan when 3 rocket-propelled grenades hit the truck. The blast threw him 25 to 30 feet, resulting in loss of consciousness for 3 to 4 minutes and confusion following. He had a mild headache immediately following and developed severe headaches 2 days later, which were initially 2 to 3 times per week and then became daily 10 months after the trauma. He was started on amitriptyline (50 mg) at bedtime and the headaches decreased to 1 to 2 times per week. He described a right-sided, especially frontoparietal, throbbing with an intensity ranging from 3 of 10 to 10 of 10 associated with nausea, vomiting at times, and light and noise sensitivity but no aura. The headache would resolve in approximately 40 minutes with an oral triptan but without medication could last 24 hours. Weightlifting and stomach crunches were triggers. There was no prior history of headaches. An MRI of the brain was normal. He also had posttraumatic stress disorder.

Comment

This is a typical case of posttraumatic migraine with onset within 1 week after blast trauma in a soldier associated with comorbid posttraumatic stress disorder.

Case 2. Footballer's Migraine

Late in the first quarter of Super Bowl XXXII on January 25, 1998, Terrell Davis, a 25-year-old running back for the Denver Broncos with a history of migraine with and without aura since age 7, was unintentionally kicked in the helmet by a Green Bay Packers defender.[60] A few minutes later, he went to the sidelines with a migraine visual

aura. Coach Shanahan sent him back in for one more play that was a fake where Elway kept the ball and ran into the end zone. Davis was given his usual migraine medication, dihydroergotamine nasal spray, on the sideline by the trainer. He went into the locker room and his severe headache was gone by the start of the third quarter, with the benefit of the extra Super Bowl halftime minutes. When he returned for the second half, he had 20 carries for 90 yards, including the winning touchdown, and won the game's most valuable player (MVP) award.[61] He had a Super Bowl–record 3 rushing touchdowns.

Comment

This is the most famous example of footballer's migraine, witnessed by 800 million viewers and occurring in American football rather than in soccer, as originally described. Early treatment of migraine can get patients back to school or work and even enable them to be a Super Bowl MVP.

TYPES AND FEATURES OF HEADACHES

Tension-Type Headache

Tension-type headaches occur in a variety of distributions, including generalized, nuchal-occipital, bifrontal, bitemporal, caplike, or headband. The headache, which may be constant or intermittent with variable duration, usually is described as pressure, tight, or dull aching. Temporomandibular joint injury can be caused by either direct trauma or jarring associated with the head injury. Patients may complain of temporomandibular joint area pain with chewing and hemicranial or ipsilateral frontotemporal aching or pressure headaches, although the pain may be referred anywhere in the trigeminal and cervical complex.[62]

Occipital Neuralgia

The term, occipital neuralgia, 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 posttraumatic headache[63] and also is seen frequently without injury. The aching, pressure, stabbing, or throbbing pain may be in a nuchal-occipital or parietal, temporal, frontal, 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.

Occasionally, referred ipsilateral facial paresthesias or subjective numbness, especially in the cheek, which is a diagnosis of exclusion, may be present due to convergence of the C2 afferents, which supply the greater occipital nerve and trigeminal afferents on second-order neurons within the trigeminocervical complex.[64] Lesser occipital neuralgia similarly can occur with pain generally referred more laterally over the head with reproduction of symptoms by digital pressure over the nerve.

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 reproduces the headache. Pain referred from the C2-3 facet joint[65] 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 or preexisting migraine may be exacerbated. Medication overuse for treatment of other

types of posttraumatic pain can also increase the frequency of migraine, whether de novo or preexisting.

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.[66] Similar attacks can be triggered by mild head injury in any sport (see Case 2).

After minor head trauma, children, adolescents, and young adults can develop a variety of transient neurologic sequelae that are not always associated with migraine and are perhaps the result of vasospasm. Five clinical types can cause the following: hemiparesis; somnolence, irritability, and vomiting; a confusional state[67]; transient blindness, often precipitated by occipital impacts; and brainstem signs.[68]

Trigeminal Autonomic Cephalalgias and Hemicrania Continua

Cluster headaches rarely result from mild head injuries, with 19 reports in the literature.[69] There are case reports of posttraumatic chronic paroxysmal hemicranias with aura[70]; short-lasting unilateral neuralgiform headache attacks with conjunctival injection, tearing, sweating, and rhinorrhea[71]; short-lasting unilateral headache with cranial autonomic symptoms[72]; and hemicrania continua.[73]

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.[74] 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,[75] 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.[76] 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 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%; 95% 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 and 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 score 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.[77] The patient often is a child or young adult who sustains what seems to be a trivial injury often without loss of consciousness.[78] 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.

Other Causes

Trauma can cause a cerbrospinal fluid (CSF) leak through a dural root sleeve tear or a cribiform plate fracture and result in a low CSF pressure headache with the same features as a post–lumbar puncture headache.[79] Hemorrhagic cortical contusions can cause a headache resulting from subarachnoid hemorrhage. Headaches can be the only symptom of posttraumatic carotid and vertebral artery dissections. Cerebral venous thrombosis[80] and carotid-cavernous fistulas[81] are other rare causes.

PATHOGENESIS

Neurobiologic Factors

Mild TBI may result in cortical contusions after coup and contrecoup injuries or diffuse axonal injury resulting from sheer and tensile strain damage.[82] Release of excitatory neurotransmitters, including acetylcholine, glutamate, and aspartate, may be a neurochemical substrate for mild TBI. Impairment in cerebral vascular autoregulation can occur. TBI can also lead to neuroinflammation with activation of glial cells, disruption of the blood-brain barrier leading to extravasation of cytoxic peripheral blood components, and activation of cytokines.[83] Neuroimaging studies, including MRI, single-photon emission CT, positron emission tomography, magnetic source imaging, magnetic resonance diffusion tension imaging, functional MRI, and MRI spectroscopy can demonstrate structural and functional deficits.[84–86] Although these findings may

help explain cognitive deficits, the causes of posttraumatic headaches are poorly understood.

Nonorganic Explanations

There are several nonorganic explanations for PCS that suggest an origin for subjective symptoms other than TBI for some people, including psychogenic, sociocultural, and psychosocial factors; base rate misattribution, expectation as etiology, chronic pain, compensation and litigation, and malingering.[87]

Psychogenic factors

A psychogenic contribution to PCS is suggested by several empiric and clinical observations. The symptom complex of PCS (headache, dizziness, and sleep impairment) is similar to the somatization seen in psychiatric disorders, including depression, anxiety, and posttraumatic stress disorder. In addition, anxiety and depression can produce subjective and objective cognitive deficits that are similar to those seen in PCS and that improve with antidepressant treatment.[88]

Several studies suggest that both psychiatric predispositions (poor coping skills, limited social support, and negative perceptions) and psychiatric comorbidity (depression, anxiety and panic, and acute and posttraumatic stress disorder) are more prevalent in patients with PCS compared with general population controls and/or with head-injured patients who do not develop persistent PCS.[26,89–92]

Studies of the interaction of depression, anxiety, and cognitive performance in TBI are limited, however. Some studies have not found a substantial correlation between the level of depressive symptoms and cognitive deficits in patients with mild TBI,[93] whereas others have found a correlation in the response to antidepressant treatment in a subset of patients.[94]

The association of psychiatric disease and PCS is not established. Limitations in methodology, including cross-sectional design and patient and control group selection bias, preclude firm conclusions. Also, such an association could have several explanations. Patients with premorbid psychiatric disease may be more likely to suffer head injury as a result of more prevalent alcoholism, motor or physical impairments resulting from their disease or medications, and other reasons. Alternatively, patients with psychiatric disease may be more prone to develop PCS after head injury. Finally, head injury may cause or precipitate psychiatric disease in susceptible individuals.

Sociocultural and psychosocial factors

The very low, even absent, rates of postconcussion symptomatology in some countries (Lithuania) and in children sometimes reported might suggest a prominent role for sociocultural factors in the pathogenesis of PCS, perhaps because of misattribution or litigation.[95,96] The Lithuanian studies have been criticized, however, because of the high incidence of chronic daily headache in the control group.[97] Another prospective study of 100 patients with acute mild head injury in Austria found none developed posttraumatic headaches at follow-up at 90 to 100 days.[98] Some studies have found poor social support and increased social adversity among patients who suffered prolonged symptoms than among those whose symptoms had remitted.[99]

Base rate misattribution

A high base rate level of PCS symptoms in the general population can lead to misattribution of symptoms to PCS. In one study of 104 healthy university community adults (61% women) with a mean age of 23 years, the following percentages endorsed the following symptoms from the *International Classification of Diseases, Tenth Revision*

criteria for PCS as present in the prior 2 weeks: fatigue, 76%; irritable, 72%; nervous or tense, 63%; poor sleep, 62%; poor concentration, 61%; sad, 61%; temper problems, 53%; headaches, 52%; memory problems, 51%; dizziness, 42%; extrasensitive to noises, 40%; nausea, 38%; and difficulty reading, 36%.[100] Several studies have compared patients with mild TBI to non–head-injured controls, finding a high prevalence of the same symptoms in both groups, indicating a high prevalence of base rate symptoms in the general population[101] similar to those with persistent PCS.[102]

Expectation as etiology

Volunteers with no history of head trauma can correctly identify the symptoms of persistent PCS present 6 months after the injury. Because patients may expect PCS symptomatology after an injury, they and their physicians may mistakenly attribute their common base rate complaints to the head injury, when they are actually unrelated.

Chronic pain

Patients with chronic pain have symptoms of PCS at a rate similar to a comparison group of patients after head injury.[103,104] Similar patterns of cognitive deficits may be seen in patients with chronic pain and PCS. It is not clear whether this reflects a shared prevalence of psychiatric disorders among sufferers of PCS and chronic pain syndromes, suggests that PCS is a manifestation of a chronic pain syndrome, or reflects the ubiquitous nature of these symptoms.[88]

Effects of compensation and litigation

Patients who have litigation are similar to those who do not in the following respects: symptoms that improve with time,[105] types of headaches, cognitive test results,[105] and response to migraine medications.[106] Symptoms usually do not resolve with the settlement of litigation.[107] 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.

Malingering

There are, however, some patients who have persistent complaints resulting from secondary gain,[108] malingering, and psychological 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"); postmorbid complaints (describing events surround 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).[109]

In a study of mild head-injured litigants, Andrikopoulos[110] compared 72 patients who had no improvement or worsening headache with 39 patients who had improving headache. Those who had no improvement or worsening performed worse on cognitive tests and had greater psychopathology on the Minnesota Multiphasic Personality Inventory-2 than those who had improving headaches, suggesting the possibility of malingering.

TREATMENT OF HEADACHES

There is a dearth of randomized placebo-controlled trials of medications for posttraumatic headaches. Only 5 studies, all done without controls, have been performed for the prevention of posttraumatic headaches. Three studies in civilians, which involved either monotherapy or combined therapy with propranolol, amitriptyline,[106,111] or valproate,[112] showed efficacy although a small study showed no benefit with amitriptyline.[113]

There are 2 open-label retrospective studies of chronic posttraumatic headaches among US soldiers. One found no significant benefit of treatment with low-dose tricyclic antidepressants but improvement with preventive treatment with topiramate. The second found benefit from onabotulinum toxin A injections, with 56% reporting more than one headache type.[114] Triptans may be effective for posttraumatic migraine.[51,115]

There are anecdotal reports of posttraumatic tension-type and migraine-type headaches treated with the usual symptomatic and preventative medications used for nontraumatic headaches. Physicians should be concerned about the potential for medication rebound headaches with the frequent use of over-the-counter medications, such as acetaminophen, aspirin, combination products containing caffeine, and prescription drugs containing narcotics, butalbital, and benzodiazepines. In one survey, more than 70% of those with headache during the first year after mild TBI used acetaminophen or a nonsteroidal antiinflammatory drug (NSAID), which was usually not effective.[116] Habituation also is a concern with narcotics, butalbital, and benzodiazepines. Although chronic posttraumatic migraine may respond to onabotulinum toxin A, this treatment is not effective for cervicogenic headaches.[117] Posttraumatic chronic daily headache may respond to an intravenous DHE regimen.

No strong evidence from clinical trials supports the use of biofeedback, cognitive behavioral therapy, physical therapy and manual therapy, immobilization devices, and ice.[118] A small study suggests benefit from cognitive behavioral training.[119]

Occipital neuralgia may improve with local anesthetic nerve blocks, which are effective alone or combined with an injectable corticosteroid if patients do not respond adequately to local anesthetics alone (eg, 3 mL of 1% xylocaine or 2.5 mL of 1% xylocaine and 3 mg of betamethasone).[63,120] Natsis and colleagues,[121] based on a cadaver study, recommend injecting approximately 20 mm to 25 mm below the external occipital protuberance and approximately 15 mm lateral from the midline, starting infiltration shortly after the injection needle has overcome the resistance of the trapezius muscle aponeurosis. Other investigators recommend injecting one-third of the way laterally along an imaginary line connecting the occipital protuberance to the mastoid process.[120] Before injection, physicians should aspirate to avoid inadvertent vascular injection.

Anecdotally, NSAIDs and muscle relaxants may also be beneficial. If there is a true occipital neuralgia with paroxysmal lancinating pain, baclofen, tizanidine, carbamazepine, gabapentin, or pregabalin may help. Physical therapy and transcutaneous nerve stimulators may help some headaches. A variety of other treatments have been proposed for refractory cases, including pulsed radiofrequency therapy[122,123] and occipital nerve stimulation.[124]

There are studies suggesting benefit from occipital nerve decompression, including one of 76 patients with complete benefit in 89.5% with patient selection based on complete but temporary improvement after an occipital nerve block.[125] There remain questions about the efficacy of decompression because of differences in definitions and diagnosis of occipital neuralgia and suggestions for a sham surgery comparison group.

Treatment of posttraumatic headaches arising from the neck and temporomandibular joint are discussed in other articles in this issue.[62,65]

EDUCATION

One of the most important roles of physicians is education of patients 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]. Patient complaints of chronic daily headaches of any type, especially posttraumatic, 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 a 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.

Physicians 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 Floyd Patterson are well known. Many have witnessed powerful punches resulting in dazed, disoriented boxers or knockouts. 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) and the fear of chronic traumatic encephalopathy. Reports of headaches preventing athletes from returning to play make the sports pages on a regular basis.

PROGNOSIS

The percentage of patients who have headaches at 1 month varies from 31.3%[33] to 90%,[126] at 3 months from 47%[127] to 78%,[128] and at 1 year from 8.4%[129] to 35%[126]; 24% of patients have persisting headaches at 4 years.[130]

In a prospective cohort study of children with mild TBI (n = 402) or arm injury (n = 122), 43% of those with mild TBI and 26% of those with an arm injury had headache 3 months after injury.[131] Headache at both the 3-month and 12-month follow-up or persistent headache was present in 28% of mild TBI versus 19% with arm injuries, which did not have statistical significance. Persistent headache was associated with female gender, family history of headache, chronic pain prior to injury, lower quality of life, prior NSAID use, and low income but was not associated with injury characteristics.

In a 36-month follow-up study of adolescents after TBI (83% mild), persistent pain (an intensity of 3/10 at each assessment in months 3, 12, 24, and 36) of any site was present in 35 of 144 subjects, with headache reported by 86%, and infrequent pain in 109 of 144 subjects, with headache reported by 45.9%.[132] Female gender and increased symptoms of depression at 3 months after injury were risk factors for persistent pain.

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