

# Posttraumatic Headache: Clinical Characterization and Management

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**Abstract** Headache is the most common symptom after traumatic brain injury (TBI). TBI has become a global health concern with an estimated 2.5 million reported TBIs per year in the USA alone. Recent longitudinal studies of posttraumatic headache (PTH) show a high cumulative incidence of 71 % after moderate or severe TBI and an even higher cumulative incidence of 91 % after mild TBI (mTBI) at 1 year after injury. Prevalence remains high at over 44 % throughout the year after moderate or severe TBI and over 54 % after mTBI. A prior history of headache is associated with a higher risk for PTH, whereas older age appears to be protective. Gender does not appear to be a risk factor for PTH. Most PTH has clinical diagnostic criteria meeting that of migraine or probable migraine when primary headache disorder classification criteria are used, followed by tension-type headache. There are no evidence-based treatment guidelines for PTH management; however, expert opinion has suggested treating the PTH using primary headache disorder treatment recommendations according to its type.

**Keywords** Posttraumatic headache · Concussion · Postconcussion syndrome · Tension-type headache · Sports concussion · Traumatic brain injury

## Introduction

As the most common symptom following traumatic brain injury (TBI) [1–3], headache is more frequently being encountered, assessed, and treated by a variety of care providers. The increase in posttraumatic headache (PTH) is primarily being driven by an increased rate of TBI diagnosis. Over the past decade, between 2001 and 2010, there was a sharp increase in total TBI-related hospitalizations, emergency department (ED) visits, and deaths to 2.5 million or from approximately 521/100,000 to 824/100,000 in the USA alone. This increase was largely driven by ED visits, with hospitalizations staying roughly the same and TBI-related deaths decreasing somewhat [4•]. A number of factors may be contributing to the increase in TBI-related ED visits including an increased awareness of brain injury through education (e.g., *Heads up: Concussion*) [5]; media interest and congressional attention to military service members fighting in the recent Iraq and Afghanistan wars and the veterans who have returned with TBI; and sports organization efforts to protect players and reduce TBI among athletes. A number of states have passed legislation to reduce TBI in youth sports by raising awareness of the importance of immediate attention and evaluation of head injuries during sports events and education among coaches, trainers, players, and their parent in recognizing symptoms related to head injuries. About 87 % of the total TBI cases (2,213,826) are treated and released from EDs, about 11 % are hospitalized and discharged, and 2 % died. Based on known cases, the CDC estimates that 75 % of all TBI is mild TBI (mTBI). There are no good follow-up data for where these

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TBI patients receive care after ED visits or hospitalization. Additionally, these numbers do not include people who did not receive medical care, had outpatient or office visits with a care provider, or from those receiving care at a federal facility, including persons serving in the military or at a veterans hospital [5].

There are gender differences in rates of TBI, except in the pediatric and elderly populations. Men are at higher risk of TBI. The Center for Disease Control (CDC) reports that for each year between 2001 and 2010, men have had higher rates of TBI-related ED visits compared to women. Rates among men grew from 492/100,000 in 2007 to 800/100,000 in 2010, a 63 % increase. Similarly, rates among women increased from 424/100,000 in 2007 to 634/100,000 in 2010, a 49 % increase. The M/F ratio is consistently between 1.3–1.5:1 [4•, 6].

Causes of injury associated with ED visits for TBI vary by age with falls being the primary cause of injury in the 0–4 years and 65 or older age groups, accounting for approximately 70–80 % of ED visits. For persons between 15 and 44 years, the proportion of ED visits due to assaults, falls, and motor vehicle traffic events are approximately equal within these ages. For more severe TBI-related injury resulting in death, TBI-related deaths in children 0–4 years are mainly due to assault (43 %) and motor vehicle traffic-related crashes (29 %). Motor vehicle events account for the majority of TBI-related deaths in youth (56 %) and are the cause of 47 % of deaths in young people 15–24 years old. Falls account for the majority of TBI-related deaths in those >65 years (54 %) [4•]. Despite high mortality rates from TBI after motor vehicle crashes, rates have declined since the 1970s, in large part to programs and policies that have resulted in the use of seat belts by vehicle occupants, improvement in vehicle design and air bag protection, and efforts to reduce risky behaviors such as speeding, alcohol-impaired driving, and texting or mobile phone use while driving.

A concussion is an mTBI; however, the term “concussion,” as commonly used, is not strictly interchangeable with mTBI but denotes the symptom complex seen after mTBI. The postconcussive syndrome (PCS) is a collection of symptoms that characterizes the underlying TBI. This requires a subjective reporting of most concussion symptoms, so underreporting by not recognizing symptoms is common. In sports-concussion statistics, there is the additional problem of underreporting because of wanting to “stay in the game.” Even so, the incidence of sports-related concussion has been reported at 3.8 million per year among the 44 million children and 170 million adults who play in organized sports events [7].

TBI statistics also fail to reflect TBI in military personnel. For both deployed and non-deployed members of all armed forces, the Army Office of the Surgeon General reports from the Defense Medical Surveillance System (DMSS) that

between 2000 and 2014 (Q2), TBI totaled 307,282 cases with 253,349 classified as mild (82 %) [8].

## Epidemiology of PTH

Though headache is the dominant symptom following TBI, it has been difficult to ascertain its exact incidence and prevalence after head injury. Many with PTH never seek care and may not be aware that headache can be a symptom of concussion, more commonly seen in mild injuries such as sports-related concussions. In studies of symptoms after injury, variability is seen as to case ascertainment, whether the ED, specialty clinics, or general medicine clinics are sampled, whether TBI severity is restricted, and whether a strict 7-day latency requirement for the PTH definition is adhered to [9]. There has also been concern regarding lack of objective test results to determine headache, and symptoms may be discounted because of litigation concerns, though early recognition of post-traumatic symptoms preceded the beginning of financial compensation [10]. Some of these study design variables may explain the wide prevalence range in retrospective studies of 30–90 % [1, 11–13].

Selected epidemiologic studies from civilian, military, and veteran populations are listed in Table 1. There are few, large longitudinal studies following TBI; however, headache prevalence appears to be high in most studies whether the population is seen in inpatient or ED settings. In a large, rehabilitation inpatient group of 452 patients, admitted following moderate to severe TBI, the majority were men injured in vehicle crashes with an average age of 44. This cohort of patients was followed over 1 year after injury with 71 % reporting headache during the first year. Prevalence of headache was 46 % at inpatient evaluation and remained high with 44 % of patients endorsing headache at 1 year after TBI [14]. A study of mTBI using the same assessment tools found that 91 % of patients from a rehabilitation inpatient group of 212 patients following mTBI reported headache during the first year after injury with prevalence of new or worse headache at 54 % initially and 58 % 1 year after injury [15•]. A higher PTH prevalence after mTBI compared to moderate to severe TBI has also been noted in pediatric populations. Prevalence of PTH after mTBI at 1 year was 41 vs 35 % at 1 year after moderate to severe injury [16].

## Risk Factors for Development of PTH

In addition to mTBI being associated with a higher risk of PTH compared to moderate to severe TBI, a prior history of headache has been found to be a risk factor in both pediatric and adult populations for development of PTH as well [14, 15•, 17–19]. Older age appears to be protective for PTH. A

**Table 1** Epidemiologic studies of posttraumatic headache

Authors, year/location	Subject number	Study design	Study population	Key findings
Blume et al. [16] 2012/US	462	Pediatric; prospective cohort	Age 5–17; mTBI=402; mod-severe TBI=60; controls=122 with arm injury	PTH prev mTBI: after 3 months=43 %; after 1 yr=41 % PTH prev: after 3 months=37 %; after 1 yr=35 % Control 3 months=26 %; after 1 yr=34 %
Dikmen et al. [20] 2010/USA	732	Civilian, prospective case-control	Age>16; any severity TBI	PTH after 1 month=55 %; after 1 yr=26 % (within the previous week)
Erickson [32•] 2011/USA	100	US Army; retrospective cohort	Headache clinic; 100 consecutive soldiers with chronic PTH	77 % had blast-related PTH; >95 % met migraine criteria
Faux and Sheedy [64] 2008/ Australia	100	Civilian; prospective ED case-control	Age>16; mod-severe TBI	PTH prev at time of ED evaluation=100 % At 1 month=30 %; at 3 months=15 %
Finkel et al. [30] 2012/USA	25	Military; retrospective, observational by headache specialist	Soldiers after deployment with mTBI	Mean # concussions=3.2. Migraine type in 64 % (9 % started after 1 month)
Hoffman et al. [14] 2011/USA	452	Civilian; enrollment: prospective, in-person; phone interview 3, 6, and 12 months	Mod-severe TBI	PTH cumulative incidence at 1 yr=71 %; baseline prev=47 %; at 1 yr=44 %
Hoge et al. [65] 2008/USA	2525	Military; cross-sectional survey	Soldiers after 1 yr Iraq deployment	HA the only symptom associated with concussion adjusting for mood disorder
Kirk et al. [66] 2008/UK	117	Pediatric prospective longitudinal; initial in hospital with questionnaire at 2 months then 3/yr for 3 yr	Age 3–15; mTBI in 93; moderate to severe in 24	Chronic PTH in 9.4 % (new or worse). All resolved between 3 and 27 months
Kjeldgaard et al. [67] 2014/ Denmark	99	Civilians; tertiary headache center; case-control	Age 18–65; CPTH ( <i>n</i> =90) after mTBI vs chronic primary migraine ( <i>n</i> =45)	CPTH vs CM: mixed headache 36 vs 51 %; TTH 97 vs 82 %; Migraine w/wo 28 vs 43 %; MOH 13 vs 22 %
Kuczynski et al. [17] 2013/Canada	670	Pediatric; prospective ED cohort; retrospective chart review of treatment cohort from brain injury clinic; phone interview 7–10 days after injury and monthly until resolution	Age 0–18; mTBI	PTH prev at 16 days=11 %; at 3 months=8 % ED cohort migraine=54 % Clinic cohort migraine=39 % (mixed HA, MOH, mood disorders with HA excluded)
Lieba-Samal et al. [18] 2011/Austria	100	Civilian; prospective phone interview	Age 18–65; (exclusions: MOH, whiplash, prior chronic PTH)	Acute PTH prev 7–10 days=66% All resolved by 3 months; migraine/prob migraine=35 %
Lucas et al. [21] 2012/USA 2014/USA [15••]	452 212	Civilian; prospective in hospital enrollment; 3, 6, and 12 month telephone interview	Age>16; 2012: mod-severe TBI; 2014 mTBI	PTH mTBI cumulative incidence=92 % PTH type (mod-severe TBI) migraine/prob mig=52 %; at 1 yr=54 % PTH type (mTBI) mig/prob mig=49 %; at 1 yr=49 %
Patil et al. [68] 2011/USA	246	Veterans; retrospective cohort at Polytrauma Network site	Veterans with mTBI	Prev of PTH=74 % in preceding 30 days Persistent HA=33 % Migraine =45 % CDH=20 %
Rosenthal et al. [69] 2013/USA	270	US military retrospective neurology clinic cohort	Consecutive soldiers with PTH from concussion with/without PTSD	CDH=58.1 % (with PTSD) CDH=52.1 % (without PTSD) No difference in migraine characteristics
Stovner et al. [19] 2009/ Lithuania	217	Civilian; prospective ED cohort; case-control; questionnaire at 3 months, 1 yr	Age 18–60; LOC<15 min	PTH prev at 3 months=65 % migraine at 3 months=19 %; at 1 yr=21 %
	1033	US Army; cross-sectional, survey-based		HA in 98 % of soldiers (PTH criteria in 37 %).

**Table 1** (continued)

Authors, year/location	Subject number	Study design	Study population	Key findings
Theeler et al. 2010/USA [33]			Soldiers with concussion in postdeployment evaluation over 5 months	Migraine type=89 %; CDH=20 % (PTH criteria in 55 %)
2012/USA [52]				

*CDH* chronic daily headache, *ED* emergency department, *HA* headache, *MOH* medication overuse headache, *PTH* posttraumatic headache, *mTBI* mild traumatic brain injury, *mod-severe TBI* moderate to severe TBI, *prev* prevalence, *CPTH* chronic posttraumatic headache, *CM* chronic migraine, *yr* year, *PTSD* posttraumatic stress disorder, *mig/prob mig* migraine or probable migraine

higher risk for PTH was seen in subjects younger than 60, when age was examined as a risk factor [15•, 20]. Females do not appear to be at increased risk of PTH over males, when prior history of headache is noted in many pediatric and adult studies of both mild and moderate to severe TBI [14, 17, 21, 22]. However, one study of concussion or suspected concussion found an adjusted odds ratio of 2.6 when women were compared with men (1.2–5.8) for PTH whereas odds ratio for other concussion symptoms was 1.0 [23].

### Typology of Posttraumatic Headache

There is an implied causation of an underlying disorder or injury when the secondary headaches are classified, with a requirement of a close temporal relationship to an underlying event but little clinical description of the associated headache [9]. Also, the secondary headache diagnosis should not be better accounted for by another ICHD-3 diagnosis. As with other secondary headaches, PTH has no defining clinical features, and the diagnosis may be problematic in those who may have pre-injury headache. A PTH definition has a requirement for the headache to have developed within 7 days of injury to the head or within 7 days of regaining consciousness after injury or of discontinuation of any medication which could impair perception or experience of the headache. Many TBI studies have adhered to this definition which may account for some variance in prevalence of PTH. In a study of returning war veterans, only 27 % of headaches developed within 7 days after injury [24], similar to findings of a prospective civilian study of primarily moderate to severe injury, in which 28 % of new headaches were reported after 3 months following the injury [14]. Thirty-two percent of hospitalized pediatric patients reported headache 2–3 weeks after injury [25] and in a pediatric ED cohort of mTBI patients; 11 % reported acute PTH at approximately 16 days (mean), and 8 % had increased headache at 3 months after their injury [17]. Because of this variable latency in time of onset of the headache, clinical

judgment on the association of headache after trauma has been used in most recent studies of PTH. To differentiate the headache after trauma to possible pre-existing primary headache, a differentiation of “new” or “worse” has been used in some study methods.

There is no single defining, clinical type of PTH. As with most other secondary headaches, the majority have characteristics that are either indistinguishable from primary headache disorders, or they are not classifiable using primary headache disorder symptom grouping. However, the importance of examining the clinical characteristics of the PTH is of epidemiological interest and may have a useful clinical benefit in guiding treatment, especially with a stratified care approach to headache severity or disabling consequences of PTH.

Most recent, prospective studies of PTH have found that migraine alone or migraine and probable migraine together were the most common type of PTH in studies of adults and children. In a longitudinal study of civilians following moderate to severe TBI, these headache types were found in 52 % of those with PTH at baseline and in 54 % at 1 year after injury. When subjects who had never had a pre-injury history of headache were evaluated, migraine or probable migraine was the most common type, found in 62 % at initial evaluation and in 53 % at 1 year after injury [14, 21]. The same research group evaluated headache type in a cohort of mTBI subjects, finding that migraine or probable migraine was the most common headache type as well, occurring in 49 % of subjects with PTH at 1 week and at 1 year [26]. Though migraine and probable migraine were the most common headache types in both the moderate to severe and mTBI groups, interestingly, tension-type headache (TTH) was found to be the next most common headache type in the mTBI group (37 %) whereas 10 % of PTH were unclassifiable [26]. In the moderate to severe TBI study, unclassifiable PTH was more common (23 %) than TTH (11 %) [21]. This finding may illustrate one difficulty in using a classification system for primary headache disorders for PTH; the rigidity of adhering to a definition may defy classification of type when some classification features of the primary

headache disorders such as nausea, photophobia, physical activity worsening a headache can independently be associated with the head injury itself, and not a headache. Other smaller patient series found migraine type to be the most common PTH as well [18, 27]. In a large study of children following mTBI, an ED cohort reported migraine as the most common PTH type occurring in 55 % of those with headache after injury [17].

Prior studies found that TTH was the most common type of PTH; however, the methodology differences make comparison between these studies difficult. The time between injury and evaluation, retrospective reviews, and subject selection are some potential differences that could account for the difference in findings [19, 28, 29]. Some study designs documented a single PTH type; others, most commonly clinic-based and retrospective, would assess all headache types within a single patient [24, 26, 30].

In active duty military studies, the prevalence of migraine-type PTH is high, with one study of USA Army soldiers in a single combat brigade showing 36 % of the PTH with migraine features [31]. Various other studies using different military populations and methodologies have shown migraine-type PTH ranging from 60 to 97 % [24, 32, 33, 34].

The frequency of headache days is higher in those who have more severe PTH, as indicated by headache type. In the prospective study of PTH following moderate to severe TBI, those with migraine-type PTH were more likely to report headache occurring several days a week or daily [21]. Twenty-three percent reported headache in >15 days per month at all evaluation time points over 1 year. In the study of mTBI, 62 % of those having headache several times a week or daily had migraine 1 year after injury [26]. In a composite model of headache frequency, duration, and intensity (headache density) after moderate to severe TBI, density did not change significantly over time even after 1 year following injury [35]. Studies of chronic daily headache (CDH) find that head and neck injury account for about 15 % of all CDH cases [36]. Unfortunately, findings of CDH are similar in military populations. Twenty percent of a cohort of 978 US Army soldiers with concussion reported headache 15 or more days per month [37]. Migraine headache features are noted in at least 70 % of chronic PTH in military studies [32, 38].

No significant correlation has been shown between presence or absence of acute neuroimaging abnormalities after moderate to severe TBI and PTH at 1 year [39], though the odds of more severe headache density was 2.84 times larger for those with penetrating skull injuries than for those with closed injuries [35].

Though migraine-type PTH are most common in civilian and military populations, other less common cases are described as similar to cluster headache [30, 40], hemicranias continua [30, 41], chronic paroxysmal hemicranias [30, 42], short-lasting unilateral neuralgiform headache attacks with

conjunctival injection and tearing (SUNCT) [43], and nummular headache and primary stabbing headache [30].

## Posttraumatic Headache Management

After a concussion or TBI, attention is likely to be focused on the most severe injury. In the case of moderate to severe TBI, there may be other co-existing severe physical injuries or brain trauma requiring surgical attention, and PTH is not likely to be addressed at this time. Those with PTH after the initial injuries are stabilized or resolve, may not be asked about this symptom, may think that it “goes with the territory” of a concussion and not bring it up to their health care provider, unless it causes significant pain or disability. In a study of high school athletes who were diagnosed with concussion or thought they may have had a concussion, initial symptoms were not recognized because of lack of knowledge of the symptoms associated with concussion [44]. Even, however, with knowledge of signs and symptoms of concussion, one study reported that 43 % of 262 athletes with a concussion history, responding to an anonymous survey hid their symptoms to stay in the game [45]. Occasionally, other symptoms of PCS such as dizziness, memory problems, and fatigue may resolve, and headache is left as an isolated symptom. Fear of the pain or that the PTH reflects ongoing brain injury may bring those with PTH to a practitioner. Typically, the first provider seen is associated with the most significant injury, and many layers of providers may be consulted before a headache specialist is seen.

The management of PTH is empiric with no strong evidence from clinical trials directing treatment of PTH. The design of clinical trials for a treatment intervention of PTH presents hurdles not seen in those without a TBI. It is not known whether cognitive difficulties would preclude the use of a headache diary or other tracking device during a trial, and even remembering to take study medication is difficult in any trial even without the potential additional burden of TBI-related issues.

There were no class I studies and only one class II study of 23 subjects with PTH in which manual spine therapy vs cold packs were used, in a review of interventions for PTH [46]. Though initially headache intensity decreased 5 weeks after treatment with spine therapy, the treatment effect was lost after 2 months [47]. A retrospective pediatric case series of 28 subjects demonstrated good therapeutic effect with peripheral nerve blocks in 93 %, with 71 % reported immediate relief from PTH, with decrease in intensity of 94 % [48].

There have been class III studies. A retrospective study of CDH in 100 subjects after mTBI measured headache frequency and intensity after at least 30-day treatment with divalproex

sodium. Other treatments with analgesics, NSAIDs, chiropractic, or physical therapy were allowed during this time; 44 % of subjects had a 24–50 % improvement, and an additional 16 % had greater than 50 % improvement with these interventions [49]. In a study of amitriptyline for depression, a subgroup analysis of patients after trauma with or without mTBI who had depression and headache found that those without mTBI ( $n=12$ ) did better on headache severity and depression scores than those with mTBI ( $n=10$ ) [50]. Onabotulinum toxin treatment was evaluated in treatment of PTH for service members with mTBI in a retrospective case series of 64 subjects. Other treatments were allowed during this study, and PTH headache types were carefully documented, with the most common type being a mixed continuous headache. Though injection protocols were variable, 63 % of study patients received onabotulinum toxin injections and 64 % reported their headaches as “better” and 28 % reported that headaches were unchanged, with 8 % as worse or unknown [51]. In another retrospective analysis of 100 active duty service members seen in a single neurology clinic with chronic PTH with a migraine type after mTBI, topiramate ( $n=29$ ) was an effective preventive therapy, associated with a significant reduction in headache frequency. Amitriptyline ( $n=48$ ) did not show a significant decrease in the same study and a high non-responder rate (35 %) to preventive medication was found [32•]. The Defense and Brain Injury Center (DVBIC) has provided PTH treatment guidelines for deployed and non-deployed service members based on class IV study evidence [52–54].

Acute treatment for persistent PTH presents similar difficulties to those seen in acute treatment of chronic migraine. The recognition of worsening or escalation of a continuous headache or daily headache may be challenging. In one retrospective analysis of service members with PTH after mTBI, triptans were effective as acute therapy when treating a predominantly migraine-type PTH [32•]. Anecdotal reports of successful treatment of acute PTH with sumatriptan or dihydroergotamine after concussion or mTBI support the use of migraine-specific therapies [55, 56].

Expert opinion has suggested treating PTH according to its clinical characteristics using the ICHD criteria for the primary headache disorders [57, 58], though migraine-specific therapies for successful treatment of headache after trauma have been recognized for over 50 years [59, 60].

Despite this, PTH is not well managed. Self-treatment with over-the-counter (OTC) medication is very common. Management of PTH was studied in a prospective natural history study of mTBI in 212 subjects [26]. In this study, prevalence of PTH was at least 58 % at any time point over 1 year with migraine as the most common headache type. Despite a high prevalence of a headache defined as moderate to severe, more than 70 % of subjects used

acetaminophen or an NSAID to treat their headache. Less than 5 % used triptans, and in the group with migraine headache type, only 19 % reported complete relief after using medication to treat PTH [61•].

Typologic similarity of PTH to the primary headache disorders does not necessarily mean there is a single underlying common physiologic mechanism of pain. Without definitive, blinded, and controlled studies of migraine-specific therapies, we rely on anecdotal evidence of success in treatment of headache symptoms. Typically, characterization studies of PTH rely on questions used to type primary headache disorders, such as severity, lateralization, worsening with physical activity, pulsatile quality, nausea or vomiting, and photophobia or phonophobia. However, the underlying TBI can also produce similar symptoms due to the concussion, such as nausea, photophobia, worsening symptoms with physical activity, or concentration with or without a PTH. So, typing the PTH is problematic in determining which symptoms are due to the PTH and which due to the TBI. Since symptoms may resolve at different times, it may take time to determine the headache type. One approach would be to identify the symptom associated with the most functional loss, such as the severity of pain and target therapy to a severe headache, which is a “migraine” feature. If the PTH is episodic, or at least, the severe PTH is identifiable, then effective acute treatment is crucial. The same treatment principles used in primary headache treatment may apply to PTH. Treating early with effective therapy may be the most important principle, since it is known that 80 % of those who self-treat have incomplete relief and the vast majority are using OTC products. Migraine-specific therapy could be effective, though if PTH is continuous or daily, it may be difficult to avoid medication overuse with any product, even the triptans. A suboptimal response to acute therapy or daily headache may warrant preventive therapy along with acute therapy for worsening or spiking headache pain [62], as overuse of acute or abortive medication could contribute to persistence of PTH after injury through development of medication overuse headache (MOH) or rebound headache. The extent of MOH associated with treatment of PTH is not known, but even without a history of pre-injury headache, it is important to recognize the possibility of MOH, especially given the extensive use of OTC products by those with PTH (60). Education regarding medication overuse is important, as medication may be used to treat pain other than PTH after an injury which may involve more than TBI. The use of a headache diary as well as a medication log may be helpful; however, the possibility that cognitive limitations secondary to the TBI or the additional responsibility of keeping daily records may limit this useful source of information. Assistance from caregivers or

family members may be needed when management, especially medication use, is discussed.

## Conclusions

PTH is the most common sequela reported after any severity TBI. High-profile professional athletes, children playing organized sports, the large numbers of returning service members and veterans of war who have TBI and PTH have driven the interest and research efforts in this area. Recent data from civilian adult and pediatric, as well as military populations, have shown that PTH is frequent and more chronic and persistent than previously thought [63]. Though the majority of PTH may be classified using primary headache disorder criteria, the usefulness of this approach in targeting treatment remains to be seen. The most helpful result of this approach may be in finding that migraine or probable migraine was the most frequent match when using primary headache disorder criteria, so that the migraine-specific medications may be used instead of OTC products.

The similarity has also helped in driving research studies into finding brain pathophysiologic mechanisms occurring after TBI. Animal models of head injury will address injury-related brain changes, but the common ground between the injury and occurrence or mechanism of PTH cannot be answered completely with these models.

Expert opinion now suggests treating PTH based on a clinical similarity to the primary headaches. Retrospective data has already shown a high non-responder rate, but controlled, clinical trials are needed to determine true efficacy in this setting. Additionally, the extra burden of difficulty in enrolling TBI patients is the dynamic state of concussion. Resolution of some symptoms of concussion or the concussion itself may leave PTH as a long-term if not permanent sequela. It will be extremely important to design clinically meaningful subgroups of those with PTH. It is highly likely that besides headache type, immediate onset or delayed onset, accompanying features, and disability, a marker of injury evolution will be necessary. Structural and functional imaging, genetic susceptibility to injury and possibly PTH, and biomarkers reflecting the character of the inflammatory response may be important in determining the most effective treatment for PTH.

## Compliance with Ethics Guidelines

**Conflict of Interest** Sylvia Lucas is a section editor for *Current Pain and Headache Reports*.

**Human and Animal Rights and Informed Consent** This article does not contain any studies with human or animal subjects performed by any of the authors.

## References

Papers of particular interest, published recently, have been highlighted as:

- Of importance
  - Of major importance
1. Lew HL, Lin PH, Fuh JL, Wang SJ, Clark DJ, Walker WC. Characteristics and treatment of headache after traumatic brain injury: a focused review. *Am J Phys Med Rehabil*. 2006;85(7):619–27.
  2. Nampiaparampil D. Prevalence of chronic pain after traumatic brain injury: a systematic review. *JAMA*. 2008;300(6):711–9.
  3. Uomoto JM, Esselman PC. Traumatic brain injury and chronic pain: differential types and rates by head injury severity. *Arch Phys Med Rehabil*. 1993;74(1):61–4.
  4. Centres for Disease Control and Prevention (CDC). Traumatic brain injury statistics 2012. [www.cdc.gov/traumaticbraininjury/statistics](http://www.cdc.gov/traumaticbraininjury/statistics). **This is an excellent website for up-to-date information on concussion, but also for physician and patient access to relevant literature on statistics of occurrence, symptoms, diagnosis and what to expect.**
  5. Faul M, Xu L, Wald MM, Coronado VG. Traumatic brain injury in the United States: emergency department visits, hospitalizations, and deaths 2002–2006. Atlanta, GA: Centers for Disease Control and Prevention, National Center for Injury Prevention and Control; 2010.
  6. Annegers JF, Grabow JD, Kurland LT, Laws ER. The incidence, causes, and secular trends of head trauma in Olmsted County, Minnesota, 1935–1974. *Neurology*. 1980;30(9):912–9.
  7. Daneshvar DH, Nowinski CJ, McKee AC, Cantu RC. The epidemiology of sport-related concussion. *Clin Sports Med*. 2011;30(1):1–17:vii.
  8. Congressional Research Service communication with Dr. Michael Carino, Army Office of the Surgeon General, September 11th, 2014. Defense Medical Surveillance System (DMSS), Defense and Veterans Brain Injury Center, <http://www.dvbc.org/dod-worldwide-numbers-tbi>.
  9. The International Classification of Headache Disorders: Third Edition-beta. 2013. *Cephalalgia*. 33:629–808.
  10. Taylor AR. Post-concussional sequelae. *Br Med J*. 1967;3:67–71.
  11. Keidel M, Diener HC. Post-traumatic headache. *Nervenarzt*. 1997;68(10):769–77.
  12. Evans RW. Post-traumatic headaches. *Neurol Clin*. 2004;22(1):237–249:viii.
  13. Linder S. Post-traumatic headache. *Curr Pain Headache Rep*. 2007;11(5):396–400.
  14. Hoffman J, Lucas S, Dikmen S, Braden CA, Brown AW, Brunner R. Natural history of headache following traumatic brain injury. *J Neurotrauma*. 2011;28:1–8.
  15. Lucas S, Hoffman JM, Bell KR, Dikmen S. A prospective study of prevalence and characterization of headache following mild traumatic brain injury. *Cephalalgia*. 2014;34(2):93–102. **The importance of this large prospective study was to show that prevalence is higher for PTH after mTBI than after moderate to severe TBI, and also to characterize the PTH with ICHD criteria supporting migraine/probable migraine as the most common PTH type.**
  16. Blume HK, Vavilala MS, Jaffe KM, Koepsell TD, Wang J, Temkin N, et al. Headache after pediatric traumatic brain injury: a cohort study. *Pediatrics*. 2012;129:e31–1439.
  17. Kuczynski A, Crawford S, Bodell L, Dewey D, Barlow KM. Characteristics of post-traumatic headaches in children following mild traumatic brain injury and their response to treatment: a prospective cohort. *Dev Med Child Neurol*. 2013;55:636–41.

18. Lieba-Samal D, Platzer P, Seidel S, Klaschterka P, Knopf A, Wober C. Characteristics of acute post-traumatic headache following mild head injury. *Cephalalgia*. 2011;31(16):1618–26.
19. Stovner LJ, Schrader H, Mickeviciene D, Surkiene D, Sand T. Headache after concussion. *Eur J Neurol*. 2009;16(1):112–20.
20. Dikmen S, Machamer J, Fann JR, Temkin NR. Rates of symptom reporting following traumatic brain injury. *J Int Neuropsychol Soc*. 2010;16(3):401–11.
21. Lucas S, Hoffman JM, Bell KR, Walker W, Dikmen S. Characterization of headache after traumatic brain injury. *Cephalalgia*. 2012;32(8):600–6.
22. Carlson KF, Taylor BC, Hagel EM, Cutting A, Kerns R, Sayer NA. Headache diagnoses among Iraq and Afghanistan war Veterans enrolled in VA: a gender comparison. *Headache*. 2013;53(10):1573–82.
23. Jensen OK, Thulstrup AM. Gender differences of post-traumatic headache and other post-concussion symptoms. A follow-up study after a period of 9–12 months. *Ugeskr Laeger*. 2001;163(37):5029–33.
24. Theeler BJ, Erickson JC. Mild head trauma and chronic headaches in returning US soldiers. *Headache*. 2009;49(4):529–34.
25. Blinman TA, Houseknecht E, Snyder C, Wiebe DJ, Nance ML. Postconcussive symptoms in hospitalized pediatric patients after mild traumatic brain injury. *J Pediatr Surg*. 2009;44:1223–8.
26. Lucas S, Hoffman JM, Bell KR, Dikmen S. A prospective study of prevalence and characterization of headache following mild traumatic brain injury. *Cephalalgia*. 2014;34(2):93–102.
27. Martins H, Ribas V, Martins B, Ribas R, Valenca M. Posttraumatic headache. *Arq Neuropsiquiatr*. 2009;2009:43–5.
28. Haas DC. Chronic post-traumatic headaches classified and compared with natural headaches. *Cephalalgia*. 1996;16(7):486–93.
29. Baandrup L, Jensen R. Chronic post-traumatic headache—a clinical analysis in relation to the International Headache Classification 2nd Edition. *Cephalalgia*. 2004;25(2):132–8.
30. Finkel AG, Yerry J, Scher A, Choi YS. Headaches in soldiers with mild traumatic brain injury: findings and phenomenologic descriptions. *Headache*. 2012;52(6):957–65.
31. Theeler B, Mercer R, Erickson J. Prevalence and impact of migraine among US Army soldiers deployed in support of Operation Iraqi Freedom. *Headache*. 2008;48(6):876–82.
32. Erickson JC. Treatment outcomes of chronic post-traumatic headaches after mild head trauma in US soldiers: an observational study. *Headache*. 2011;51(6):932–44. **This is an, open label retrospective study in a military population showcasing the difficulty of finding effective treatment for PTH and a high non-responder rate to preventive medication.**
33. Theeler BJ, Flynn FG, Erickson JC. Headaches after concussion in US soldiers returning from Iraq or Afghanistan. *Headache*. 2010;50(8):1262–72.
34. Ruff RL, Ruff SS, Wang XF. Headaches among Operation Iraqi Freedom/Operation Enduring Freedom veterans with mild traumatic brain injury associated with exposures to explosions. *J Rehabil Res Dev*. 2008;45(7):941–52.
35. Walker WC, Marwitz JH, Wilk AR, Ketchum JM, Hoffman JM, Brown AW, et al. Prediction of headache severity (density and functional impact) after traumatic brain injury: a longitudinal multicenter study. *Cephalalgia*. 2013;33(12):998–1008.
36. Couch J, Lipton R, Stewart W, Scher A. Head or neck injury increases the risk of chronic daily headache: a population-based study. *Neurology*. 2007;69(11):1169–77.
37. Theeler BJ, Erickson JC. Post-traumatic headaches: time for a revised classification? *Cephalalgia*. 2012;32(8):589–91.
38. Theeler B, Lucas S, Riechers RG, Ruff RL. Post-traumatic headaches in civilians and military personnel: a comparative, clinical review. *Headache*. 2013;53(6):881–900. **Good comparison of prevalence and characteristics of PTH in the civilian and military populations with remarkable similarity of findings despite etiology of the injury.**
39. Lucas S, Devine J, Bell K, Hoffman J, Dickmen S. Acute neuroimaging abnormalities associated with post-traumatic headache following traumatic brain injury (P04.020). *Neurology*. 2013;80(American Academy of Neurology meeting abstracts).
40. Clark ME, Bair MJ, Buckenmaier CC, Gironde RJ, Walker RL. Pain and combat injuries in soldiers returning from Operations Enduring Freedom and Iraqi Freedom: implications for research and practice. *J Rehabil Res Dev*. 2007;44(2):179–94.
41. Lay CL, Newman LC. Post-traumatic hemispheric headache. *Headache*. 1999;39(4):275–9.
42. Matharu MJ, Goadsby PJ. Post-traumatic chronic paroxysmal hemicrania (CPH) with aura. *Neurology*. 2001;56(2):273–5.
43. Piovesan EJ, Kowacs PA, Werneck LC. S.U.N.C.T. syndrome-report of a case preceded by ocular trauma. *Arq Neuropsiquiatr*. 1996;54(3):494–7.
44. Trivedi N. International School; Youth sports concussion. Presentation to TBI Model Systems group at University of Washington from original research. 2015; personal communication.
45. Torres DM, Galetta KM, Phillips HW, et al. Sports-related concussion: anonymous survey of a collegiate cohort. *Neurol Clin Pract* 2013;3:279–87.
46. Watanabe T, Bell K, Walker W, Schomer K. Systematic review of interventions for post-traumatic headache. *PMR*. 2012;4:129–40.
47. Jensen OK, Nielsen FF, Vosmar L. An open study comparing manual therapy with the use of cold packs in the treatment of post-traumatic headache. *Cephalalgia*. 1990;10(5):241–50.
48. Dubrovsky AS, Friedman D, Kocilowicz H. Pediatric post-traumatic headaches and peripheral nerve blocks of the scalp: a case series and patient satisfaction survey. *Headache*. 2014;54(5):878–87.
49. Packard RC. Treatment of chronic daily post-traumatic headache with divalproex sodium. *Headache*. 2000;40(9):736–9.
50. Saran A. Antidepressants not effective in headache associated with minor closed head injury. *Int J Psychiatry Med*. 1988;18(1):75–83.
51. Yerry JA, Kuehn D, Finkel AG. Onabotulinum toxin for the treatment of headache in service members with a history of mild traumatic brain injury: a cohort study. *Headache*. 2015;55(3):395–406.
52. Theeler BJ, Erickson JC. Post-traumatic headache in military personnel and veterans of the Iraq and Afghanistan conflicts. *Curr Treat Options Neurol*. 2012;14(1):36–49.
53. Defense and Veterans Brain Injury Study Group. Clinical guidance for evaluation and management of concussion/ mTBI-acute/sub-acute. Deployed and CONUS Setting Versions. 2012. [www.dvbic.org/](http://www.dvbic.org/). Accessed 06.21.15.
54. Schultz BA, Cifu DX, McNamee S, Nichols M, Came W. Assessment and treatment of common persistent sequelae following blast-induced mild traumatic brain injury. *Neuro Rehab*. 2011;28(4):309–20.
55. Gawel MJ, Rothbart P, Jacobs H. Subcutaneous sumatriptan in the treatment of acute episodes of post-traumatic headache. *Headache*. 1993;33(2):96–7.
56. McBeath JG, Nanda A. Use of dihydroergotamine in patients with post-concussion syndrome. *Headache*. 1994;34(3):148–51.
57. Seifert TD, Evans RW. Post-traumatic headache: a review. *Curr Pain Headache Rep*. 2010;14(4):292–8.
58. Lucas S. Headache management in concussion and mild traumatic brain injury. *PMR*. 2011;3(10 suppl 2):S406–412.
59. Raskin NH. Post-traumatic headache: The postconcussion syndrome. In: *Headache*. 2<sup>nd</sup> edition. N. Raskin, ed. Churchill Livingstone, New York, 1988. pp. 269–281.
60. Simons DJ, Wolff HG. Studies on headache: mechanisms of chronic post-traumatic headache. *Psychosom Med*. 1946;8:227–42.
60. DiTommaso C, Hoffman JM, Lucas S, Dikmen S, Temkin N, Bell KR. Medication usage patterns for headache treatment after mild traumatic brain injury. *Headache*. 2014;54(3):511–9. **This paper reports a lack of effective therapy for PTH and a very high rate of self-treatment with OTC products, reflecting the need for management strategies and more research in the area of treatment.**



62. Silberstein SD. Preventive treatment of migraine. *Rev Neurol Dis.* 2005;2(4):167–75.
63. Scher AI, Monteith TS. Epidemiology and classification of post-traumatic headache: what do we know and how do we move forward? Comment on Lucas et al., “Prevalence and characterization of headache following mild TBI”. *Cephalalgia.* 2014;34(2):83–5.
64. Faux S, Sheedy J. A prospective controlled study in the prevalence of post-traumatic headache following mild traumatic brain injury. *Pain Med.* 2008;9:1001–11.
65. Hoge C, McGurk D, Thomas J, Cox A, Engel C, Castro C. Mild traumatic brain injury in US soldiers returning from Iraq. *N Engl J Med.* 2008;358(5):453–63.
66. Kirk C, Nagiub G, Abu-Arafeh I. Chronic post-traumatic headache after head injury in children and adolescents. *Dev Med Child Neurol.* 2008;50:422–5.
67. Kjeldgaard D, Forchhammer H, Teasdale T, Jensen RH. Chronic post-traumatic headache after mild head injury: a descriptive study. *Cephalalgia.* 2014;34(3):191–200.
68. Patil VK, St. Andre JR, Crisan E, Smith BM, Evans CT, Steiner ML, et al. Prevalence and treatment of headaches in Veterans with mild traumatic brain injury. *Headache.* 2011;51:1112–21.
69. Rosenthal JF, Erickson JC. Post-traumatic stress disorder in U.S. soldiers with post-traumatic headache. *Headache.* 2013;53:1564–72.