

Post-traumatic headache: clinical caveats

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Key words. Cerebral concussion. Cerebral traumatism/complications. Headache/etiology. Neck pain/etiology.

INTRODUCTION

Headache and neck pain are the most common physical complaints following concussion (mild brain injury) and are experienced early after injury by up to 70 % of persons with these types of injuries. Headache also occurs after more severe brain injury. However, for some reason, as yet unidentified, it tends to be a much less common phenomenon in this group of patients when compared with the incidence following mild traumatic brain injury. This suggests that the traumatic brain injury (TBI) itself is probably not the primary cause of the headache because it would logically follow that if it was one would expect more headache problems with more severe TBI, which is not the case in practice. Headache is also a common problem after cranial trauma, as well as spinal acceleration-deceleration (i.e. whiplash) injuries (1,2).

Often, injured persons will seek medical care following traumatic injuries only to be diagnosed with "post-traumatic headache" (PTHA). Such a non-specific diagnosis without elaboration as to the specific pain generators leaves the treating clinician, fellow clinicians, as well as others (including patients and their families) with no real information regarding the true etiology of the headache disorder, its prognosis, or the appropriate treatment regimen that should be administered to modulate or ideally cure the headache (1).

Although the majority of headache following mild brain injury is most likely a non-surgical problem, there are, on occasion, complications that occur (i.e. after more severe TBI) that may require surgical

intervention. Subdural and epidural hematomas (blood collecting between the brain and the skull), carotid cavernous fistulas (abnormal communication between the venous blood flow and arterial blood flow), traumatic carotid artery dissection, cavernous sinus thrombosis, as well as post-traumatic intracranial pressure (ICP) abnormalities (high versus low ICP), among other conditions can all be responsible for PTHA and bring with them a potential need for surgical intervention (2). Mechanisms of injury must also be considered in the context of PTHA assessment as recent experience with blast concussion and polytrauma have demonstrated (3-5).

The experienced clinician should be able to determine the underlying cause for the PTHA with appropriate time taken to acquire an adequate pre-injury, injury and post-injury history, as well as conduct a careful physical evaluation and as clinically indicated appropriate diagnostic testing. Treatment should be instituted in a holistic fashion with a goal of maximizing the benefit/risk ratio of any particular intervention, prescribing treatment that can be optimally complied with and educating the patient and family regarding the condition, its treatment and prognosis. Late onset headaches (i.e. greater than 6 months post-trauma) should cue the treating clinician to think of less common injury related conditions such as seizures as a cause for the headache disorder or, just as likely, a non-injury related cause such a space occupying lesion (i.e. brain tumor), among other conditions. Recent research provides insights into the prevalence, natural history and characterization of PTHA that all clinicians involved with treatment should be familiar (6-8).

CATEGORIZATION

The major types of headaches seen following trauma include: musculoskeletal headache (including direct cranial trauma, cervicogenic

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headache and temporomandibular joint disorders), neuralgic (nerve) headache, tension type headache, migraine, as well as more uncommon causes of headache including dysautonomic headaches, seizures, pneumocephalus (air in the head), cluster, paroxysmal hemicrania, post-traumatic sinus infections, drug induced headache, medication overuse headache (previously called rebound headache) and the surgical conditions previously mentioned (1,2).

The most common cause of PTHA in this clinician's extensive experience is cervicogenic headache which may have several different possible causes. Referred cervical myofascial pain as a consequence of cervical whiplash is a particularly common cause for this condition, particularly when there is involvement of proximal aspects of cervical or associated musculature... such as trigger points in the sub-occipital musculature, proximal portions of the sternocleidomastoid and/or upper trapezius muscles.

ETIOLOGY

There are multiple sources of head and neck pain, both inside and outside of the head (2). The brain itself, interestingly, is not a source of pain. Headache typically results from six major physiologic phenomena:

- Displacement of intracranial (within the skull) structures.
- Inflammation.
- Ischemia (decreased blood flow) and/or metabolic changes.
- Myodystonia (increased muscle tone).
- Meningeal irritation (inflammation/irritation of the thin layers of tissue "coating" the brain).
- Increased or decreased intracranial pressure.

NATURAL HISTORY, PROGNOSTIC FACTORS AND OUTCOME

There are inadequate evidence based studies to stipulate dogmatically the natural history, prognostic factors and long term outcomes of PTHA, in part, because PTHA is not one single pathophysiological disorder but rather a symptom descriptor that may involve multiple pain generators. Additionally, there are major methodological flaws including an absence of validity measures of any kind and a relative over-reliance on using IHS (International Headache Society) headache classification systems as the only method to categorize headache subtypes.

This relative lack of prospective, controlled and blinded studies only further challenges our ability to accurately diagnose and treat this group of patients. There are also multiple methodological challenges in studying an impairment that is predominantly based on subjective patient report including issues of misattribution bias, recall bias, nocebo effect (i.e. a negative psychic reaction experienced by a patient who is given an inaccurate diagnosis or prognosis), or potential response bias relative to symptom amplification regarding pain reporting and/or associated pain related disability, among other issues. Conversely, secondary gain may also apply to patients with PTHA underreporting their symptoms (e.g. the football player who wants to go back in the game or the soldier who want to return to his unit) (9).

Any study of chronic PTHA must also address the inherent co-morbidities of the psychological and medical effects of chronic pain (and stress) on not only the patient's reporting of their pain but also on myriad other aspects of function including cognition, behaviour and sleep (2,9). The majority of the studies to date have not based conclusions on comprehensive physical assessments that integrate neurological and musculoskeletal assessment and/or additionally link specific exam findings with current headache classification systems (the latter which have been criticized relative to their lack of applicability and relevance to this particular population) (2,10).

Studies have demonstrated that ongoing litigation has little effect on the persistence of headache complaints. Specifically, studies have shown that patients still continue to report significant symptoms even after litigation has ended (Note: the work in this area is limited and further research to confirm the findings of prior studies is recommended). A small number of patients will develop intractable, severe PTHA; however, this group of patients has been poorly studied and the influence of non-organic and/or psychogenic factors in such patients remains unclear.

When properly diagnosed and treated, most PTHA is able to be cured, and if not cured, modulated, and will not likely be disabling over the long term. There is now a substantive literature discussing PTHA treatment options; however, it is lacking in an adequate foundation of prospective, randomized and controlled research (1,2,10,11).

PTHA prognosis must be based on an exact understanding of headache etiology (based on history and focused examination, overlay as relevant of psychogenic (including patient pre-injury characterological issues) and secondary gain factors, response to appropriate historical treatment

and consideration of whether the correct treatment for the pain generator was ever instituted at all (2,9,10).

CONCLUSIONS

PTHA is ultimately a symptom and not a diagnosis. This complex disorder has multiple potential causes and as a result has multiple potential ways to address the pain that is associated with the underlying pain generators. Assessing and treating PTHA is a process that requires adequate time commitment and knowledge by the treating clinician... some will consider this “a pain” and if that is the case, then those clinicians should defer treatment to others who make it their business to assess and treat these types of patients.

Pejorative and potentially self-prophesizing labels such as “chronic PTHA” are often a misnomer due to the fact that the actual pain generators were never diagnosed correctly in the first place. Ideally, such labels should be avoided. There is in fact hope for those with PTHA regardless of how long they have suffered from pain. The challenge is finding clinicians who understand the disorder and have experience in holistic assessment and treatment of post-trauma patients including those with TBI, cranial trauma and whiplash injuries.

Conflicts of interest

The author declares no conflict of interests.

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Palabras clave. Cefalea/etiología. Conmoción encefálica. Dolor cervical/etiología. Traumatismo cerebral/complicaciones.

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WEBSITES OF INTEREST

- American Council for Headache Education. www.achenet.org
- National Headache Foundation. www.headaches.org
- The American Council for Headache Education provides a listing of on-line and local support groups. www.achenet.org
- An on-line headache diary is available at: www.achenet.org/your/diary1.php

Article available in English and Spanish

Received: 27.05.2013. Accepted: 6.06.2014.

How to cite this article: Zasler N. Post-traumatic headache: clinical caveats [Cefalea postraumática: advertencias clínicas]. *Rev Cubana Neurol Neurocir*. [Internet] 2014 [cited year, month and day];4(2):105–8. Available from: <http://www.revneuro.sld.cu> Spanish, English.

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ISSN 2225–4676

Editor: P. L. Rodríguez García M.D.

Editor recommendation: another articles of N. Zasler

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