POST-TRAUMATIC HEADACHES: A PAIN IN THE BRAIN?

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. Post-concussive headaches may be quite persistent; however, they cannot be positively correlated with severity of injury. Often, injured persons will seek medical care following concussion and/or cervical whiplash injury only to be diagnosed with "post-traumatic headache" (also termed PTHA). This article will provide the reader with an overview of issues related to diagnosis, treatment and outcome of post-traumatic headache.

Although the majority of headache conditions following mild brain injury are benign, there are, on occasion, complications that occur that may require surgical intervention. Specifically, certain more serious complications may occur after closed head injury that results in persistent headache including subdural and epidural hematomas (blood collecting between the brain and the skull), abscesses, and carotid cavernous fistulas (abnormal communication between the venous blood flow and arterial blood flow). Through appropriate clinical examination and potentially additional diagnostic tests, these types of conditions can be ruled out.

There are multiple sources of head and neck pain, both inside and outside of the head. The brain itself, interestingly, is not a source of pain. Inside the skull the major structures responsible for pain are the thin coating over the brain at its base (dura), the venous sinuses, blood vessels and certain cranial nerves (specifically II, III, V, XI and X). Outside of the skull the major structures that may produce pain after trauma include the skin, muscles, arteries, joint capsules, cavities within the head such as the sinuses, eyes, ears, nose and oral cavity, cervical nerves (1st through 3rd), and the thin layer of pain sensitive tissue coating bones in the head and neck (periosteum).

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 intracranial pressure

The reader should also be aware of the fact that PTHA is not a diagnosis per se but rather a symptom of an underlying disorder. It is all too often that patients are simply given a diagnosis of post-traumatic headache (PTHA) and no further elaboration is made relative to the problem causing the pain. Often PTHA is treated as vascular (or migraine headache), when, in fact, the great majority of these headaches are not due to migraine-like phenomena. It is therefore not surprising that persons treated in this manner often do not respond to the prescribed treatment regimen.

It is important for the examining clinician to keep the different mechanisms of PTHA in mind. Additionally, the mechanism of injury responsible for the initial insult should also be investigated. Specifically, inquiry regarding history pertaining to three main phenomena:

- brain injury
- cranial or cranial/adnexal trauma (damage to the head or structure in the head but outside the brain)
- cervical acceleration/deceleration (CAD) insult (also called whiplash injury).

One of the major clues for the examiner relative to the origin of the headache should come from establishing the symptom profile for that particular headache as well as the patient’s pre-injury history of headache. Just because an individual had headache pre-injury does not mean that he or she could not develop a different type of headache or a worsening of the pre-injury condition following trauma. The major questions relative to the headache profile that need to be asked are expressed in the pneumonic “COLDER”: ***C***haracter, ***O***nset, ***L***ocation, ***D***uration, ***E***xacerbation, and ***R***elief. Other descriptors including the frequency, severity, associated symptoms, and presence/absence of aura, degree of functional disability associated with headache episodes, as well as, the time of day that headaches come on are all important parameters to inquire about. With the aforementioned clues the clinician is then armed to conduct a clinical examination to allow a more specific conclusion as to the origin of the headache condition.

The major types of headaches seen following trauma include musculoskeletal headache, neuroma/neuralgic (nerve) headache, post-traumatic sympathetic nerve dysfunction, vascular (migraine) headache, and other rare causes of headache including seizure disorders, pneumocephalus (air in the head), cluster and paroxysmal hemicrania, as well as, the potential surgical conditions previously mentioned.

In this clinician’s experience, the most common cause of head pain (headache) after trauma is musculoskeletal relating to so-called myofascial pain syndromes secondary to cervical acceleration-deceleration insult with referred pain into the head. Another form of musculoskeletal pain that should be considered is temporomandibular joint dysfunction. Musculoskeletal headache typically presents with symptoms of pressure and tension, often with a cap-like distribution. The headache tends to get worse with stooping, bending or exertion and it may be associated with other symptoms such as dizziness or sensitivity to light (photophobia). Clinicians must have a good understanding of associated symptoms as well as factors which may perpetuate myofascial pain disorders following injury in order to appropriately diagnose and treat this condition.

Often seen in association with cervical myofascial dysfunction is somatic (vertebral) dysfunction. This condition is significantly under-appreciated by the vast majority of clinicians evaluating and treating this significant patient population. Subtle vertebral rotations, anterior as well as posterior, may cause pain, both local and referred into the head, through multiple mechanisms. Manual and/or manipulative therapy can be quite effective when used alone or in combination with other interventions to assist in “resetting” bony/osseous structures. Conservative

short-term and/or infrequent/episodic palliative treatment utilizing manual medicine therapy is recommended and ***not*** long term chronic treatment which does not address the underlying problem(s) at hand.

Temporomandibular joint dysfunction is a controversial consequence of "whiplash" type injury. It is my experience that injury to the chewing muscles (muscles of mastication), is a much more common problem than injury to the joint itself, the latter being relatively rare. However, it should be noted that with significant muscle injury the joint may become secondarily involved. The condition is clearly ***over diagnosed*** as occurring from trauma as many persons have pre-existing TMJ disorders which may get worse following significant cervical "whiplash" injury.

Large nerves in the scalp may also be injured following trauma, either as a result of direct injury or entrapment from muscles which have been injured and are now in a state of "spasm" (more appropriately called myodystonia). The most common large nerves involved in headache pain are the greater and lesser occipital nerves; however, other nerves may also be involved. Sometimes one may see headache pain generated by local contusion to the scalp with underlying scar formation in the nerves. This type of pain tends to be a shooting, stabbing type pain. Particularly with greater occipital neuralgia there is a very classic finding of tenderness over the greater occipital nerve with referred pain into the fronto-temporal region of the head and sometimes with associated pain around or behind the eye. These condition should be treated in several ways including consideration of local nerve blocks, treating associated muscle spasm, counter-irritation techniques, possible use of topical medication such as Zostrix (Capsaicin) for pain mediated by small nerves in the scalp, and lastly and most aggressively, surgical intervention.

Certain nerve fibres in the neck, both in the front or anterior section as well as the back or posterior section, may also be damaged from excessive flexion or extension of the neck associated with cervical acceleration/ deceleration insult. Anterior injury may produce a variety of clinical conditions including so-called dysautonomic cephalalgia. There may be partial or total insult to these nerves which impacts on how the condition is treated relative to medication choices. Involvement of posterior cervical sympathetic dysfunction (also known as Barre-Lieou syndrome) may produce symptoms of pain in the back of the head, tinnitus (buzzing in the ears), blurry vision, and vertigo.

Post-traumatic migraine accounts for up to 20% of chronic post-traumatic headache. It is generally treated similarly to non-traumatic migraine. There are some atypical variants of post-traumatic migraine, such as basilar artery migraine, that are known to occur more frequently in young females, particularly following "whiplash" injury. The exact reason for this is still unclear. This type of vascular headache also known as BAM can also be treated with atypical migraine medications such as carbamazepine (Tegretol) or valproic acid (Depakote or Depakene) if unresponsive to more traditional migraine treatment. Another atypical migraine "variant" following trauma is transient cortical blindness which is occasionally seen in the pediatric population.

Vascular headache treatment should include looking at all associated factors that may influence the headache picture including reduction of so-called trigger factors (this may include certain food groups as well as external and internal stressors). Treatment should be directed at minimizing the functional disability associated with the headache through other interventions including appropriate medication prescription which may be abortive, symptomatic, and/or prophylactic. A small percentage of women who take birth control pills may be exacerbating their migraines and this should be considered in the overall holistic treatment of patients with post-traumatic migraine. Other intervention such as relaxation training and biofeedback may also be utilized.

There are multiple rare causes of headache that should also be considered in the post-trauma population including tension pneumocephalus, carotid cavernous fistulas, late extra-axial collections including subdural and epidural hematomas, as well as, subdural hygromas, cluster headache, "over-shunting" and communicating hydrocephalus. Appropriate neurodiagnostic tests such as CT or MRI scanning of the brain, plain x-rays, angiography or magnetic

resonance angiography (MRA), and other vascular studies should be conducted as deemed appropriate by the treating clinician to rule out such disorders.

Multiple studies, some completed only in the last 3-5 years, demonstrate that ongoing litigation has little to no effect on the persistence of headache complaints. Specifically, studies have shown that patients still continue to report significant symptoms even after litigation has ended. A small number of patients will develop intractable post-traumatic headache. In this practitioner's experience, when properly treated, most PTHA is not permanent and/or disabling over the long term.

With the appropriate time taken in acquiring an adequate pre-injury and post-injury history as well as conducting a careful clinical evaluation and as necessary ordering appropriate further diagnostic testing, the experienced clinician should be able to determine the underlying cause for the post-traumatic headache condition. Once the appropriate diagnosis is made treatment should be instituted in a holistic fashion with a sensitivity to 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. Do not accept a "diagnosis" of "post-traumatic headache" ...it simply suggests the clinician does not know what is causing the headache.

FOR FURTHER INFORMATION: SUGGESTED READING

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