Chronic Post-Traumatic Headache (2003)

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Features

While most post-traumatic head and neck pain improves within days to weeks after injury, prolonged pain – known as chronic post-traumatic headache (CPTH) - afflicts millions. The IHS Classification system requires that CPTH begin within 2 weeks of head injury, but many patients have a delay in symptoms for longer than this. Often milder, rather than more severe, head injuries lead to significant CPTH. The majority of CPTH sufferers fit a chronic tension-type headache description, but many seem to have typical migrainous headaches, and some even develop cluster headache features. Most patients also have features of the Post-Concussive Syndrome (PCS), as listed below in Table 1.

Table 1: Symptoms of Post-Concussion Syndrome:

Headache, Neck pain
Vertigo, imbalance, dizziness
Mood disorders
- irritability, anger outbursts, depression, mania
Anxiety
Cognitive and attentional changes
Sleep dysfunction
Chronic fatigue
Vision disturbances (blurred, photophobia)
Sexual dysfunction

Many patients improve in the first several months but many have persistent recurring pain. Packard coined the term "Permanent Post-traumatic Headache" for those cases of pain lasting beyond 1 year or beyond 6 months with plateau over the last 3 months. Results of litigation seem not to influence CPTH and, contrary to common belief, the existence of premorbid headache disorders does not seem to be a risk factor for the development of CPTH.

Pathophysiology

The mechanisms of CPTH are not fully understood. It has been speculated that PTH may be due to "central sensitization" resulting from persistent peripheral input from painful injured tissues. Another postulated mechanism is "diffuse axonal injury", arising from acceleration-deceleration, and/or rotational forces, which might result in disruption of antinociceptive function. Axons traversing the upper brainstem seem to be particularly at risk for axonal injury in this setting. The area encompassing the periaqueductal gray/dorsal raphe nucleus is in this region, and has been implicated in headache (migraine) activity.

Assessment

Secondary ("symptomatic") causes of CPTH should be ruled out (see Table 2). Careful history and detailed head and neck exam are usually sufficient to exclude these, but MRI can sometimes be helpful. Despite much investigation, laboratory confirmation of CPTH is not available, so it remains a clinical diagnosis. This has led to much controversy over diagnosis. PET, fMRI and other approaches look hopeful.

Table 2: Possible Secondary Causes of CPTH

Whiplash/cervical spine injury
Upper cervical root entrapment
Temporomandibular joint injury
Vascular dissection (carotid, vertebral arteries)
Subdural hematoma (rarely, epidural hematoma)
Neuralgias, Eagle's syndrome, Neuromas
CSF hypotension (CSF leak)

Intracranial hypertension/hydrocephalus
Cerebral vein thrombosis
Post-traumatic seizures

History-taking must include medication use since CPTH may be perpetuated by overuse of analgesic medications, (analgesic rebound headache). Cranial examination should include inspection of the temporomandibular joint and palpation of the head and neck for the possible presence of painful scars and neuromas. A Tinel's sign over the occipital nerve may suggest occipital neuralgia. Tenderness in the submandibular region may suggest Eagle's syndrome (inflammation of the styloid process or stylohyoid ligament which can occur post-traumatically). MRI will exclude subdural hematomata, hydrocephalus and masses. Lumbar puncture may be performed if increased or decreased (CSF leak) intracranial pressure is being considered.

Treatment Strategies

The approach to the patient with PTH must be individualized. Co-morbid and co-existent conditions (such as associated PCS symptoms) impose therapeutic limitations but may also suggest therapeutic opportunities. We have had the best results with pharmacological and non- pharmacological treatment aimed at the primary headache type the CPTH most resembles. We generally combine non-pharmacologic measures such as physical therapy, cognitive behavioral therapy, and biofeedback with pharmacologic measures including acute medications for specific episodes, and prophylactic medication. Strict control of analgesics to prevent analgesic rebound seems to be essential. The use of headache calendars is essential to gauge progress.

Non-Pharmacological Treatment

Lifestyle adjustment including sleep regulation, avoidance of trigger activities, discontinuation of nicotine and alcohol, and regular appropriate exercise should be stressed. Relaxation techniques, including thermal and myographic biofeedback, imagery, and hypnotherapy have proven helpful for many patients. Cognitive-behavioral programs can also be highly effective, but are clearly limited in patients with significant post-traumatic cognitive impairments. Individual (as well as family or group) psychotherapy can address associated post-traumatic mood and behavioral changes, but can also provide effective pain-coping strategies. Massage, mobilization techniques, and myofascial release can be effective in management of PTH, particularly in cases where cervicogenic headache seems significant. TENS and acupuncture have been helpful in many cases as well.

Pharmacological Treatment - Acute

Acute symptomatic treatment of PTH pain is best treated with non-addictive medication. Specific choices, including non-steroidal antiinflammatory medications, muscle relaxants, and others, are discussed below. Acute therapy of migraine has been revolutionized by the advent of the "triptans". Currently, almotriptan, eletriptan, frovatriptan, naratriptan, rizatriptan, sumatriptan, and zolmitriptan are available. Non-steroidal anti-inflammatory drugs may be useful if given early in the attack and at high enough doses. A gastric motility-enhancing drug such as metoclopramide may improve absorption and increase efficacy. We have found hydroxyzine a useful adjunct for headache pain and associated nausea. Intranasal or subcutaneous/intramuscular dihydroergotamine remains useful although less convenient to utilize than the oral triptans. Selecting the correct route of drug administration is very important. It is important to consider non-oral routes for medication if there is prominent nausea and/or vomiting. Injections, nasal sprays, and suppositories may be appropriate (Ward 1998).

Pharmacological Treatment - Prophylactic

Prophylactic pharmacological therapy for PTH should be considered when acute medications are ineffective, required frequently, or are not well tolerated. Doses should be low initially and advanced as necessary and as tolerated. Adverse effect profiles should be tailored to the individual, and carefully explained. Multiple symptoms should be targeted with the minimum of medications (e.g. – the choice of cyclic antidepressants for patients with concomitant depression and pain). Daily preventive medications should be challenged for effectiveness and discontinued when possible. Migraine prophylactic meds with strong support in the literature include propranolol, valproic acid and amitriptyline, and these are used extensively in patients with CPTH. Other antidepressants such as bupropion, venlafaxine and clomiprimine have been used for CPTH and can alleviate some of the other symptoms of PCS (eg insomnia, depression, anxiety) as well. Newer anticonvulsants such as gabapentin, topiramate, felbamate and zonisamide are being proposed by some for CPTH.

Anesthetic Blocks

Neuralgic syndromes can frequently co-occur with other headache types in patients with PTH. Local nerve infiltration

with lidocaine and/or bupivicaine can be both diagnostic as well as palliative in patients with occipital neuralgia, supraorbital neuralgia, and Eagle's syndrome. Trigger point injection, particularly in patients with cervicalgia, can be very effective in selected cases.

Inpatient Treatment

Refractory daily or frequent severe headaches may require hospitalization. Repetitive intravenous dihydroergotamine as described by Raskin (1986) can be dramatically effective. Other intravenous protocols include chlorpromazine and valproic acid (Mathew et al. 1999). Appropriate selection and performance of these regimens often requires a high level of experience and knowledge. Referral of the patient to a knowledgeable headache expert or headache center may be the most efficient way to manage the patient, especially if more straightforward and simpler measures have failed to provide sufficient benefit. Such referrals are usually appropriate for those patients with unusual features, unclear diagnoses, poor response to therapies, or failure to improve over time.

Conclusion

The evaluation and management of patients with CPTH must be individualized and comprehensive. The majority of patients will spontaneously improve within 6 months. The remainder can still be helped by a symptom-based approach that is both competently applied and compassionate.