Mild Head Injury and Posttraumatic Headache

Posttraumatic headache (PTH) is the most common symptom following mild head injury. These headaches are often very difficult to manage because of the problems in evaluating and treating a subjective symptom, the unclear clinical picture, and minimal evidence of organic abnormality. This document will provide a detailed review of PTH and the psychological / physiological factors contributing to its etiology and maintenance.

Background:

Symptoms following mild head injury (MHI) have been recognized for well over a hundred years, although the reasons hypothesized for the symptoms have shifted dramatically. The clinical illustration of head trauma with persistent postconcussion symptoms was first described by Maty in 1766 and later in the mid-nineteenth century by Boyer, Dupuytren, and Cooper (Trimble, 1981). Ericksen (1882) used these examples and numerous others in arguing that mild head injury or "concussion of the spine" could result in severe disability due to injuries of the central nervous system. The prevailing opinion in that era, however, was that a head injury did not occur in the absence of obvious external injury or damage. Many symptoms of MHI were considered to be malingering, "psychogenic," or "functional disorder notions" (Ericksen, 1882). Rigler (Trimble, 1981) raised the question of "compensation neurosis" in reports of an increase in postconcussion symptoms after the availability of financial compensation from accidental injuries occurring on the Prussian railways. Often overlooked was the observation that similar symptoms were reported prior to workmen's compensations and other liability laws.

With a dramatic increase in the number of head injuries due to accidents in the latter part of this century more credence was given to organic causes for PTH. Strauss and Savitsky (1934) interestingly pointed out that a subgroup of clinicians were certain that patients complaints after head injury were psychogenic until the clinicians themselves suffered MHI, whereupon they became certain that their own symptoms were due to organic changes in the brain. Controversy continues to exist about the legitimacy of PTH often because evidence of anatomic abnormalities is minimal. Only recently has technology provided means of assessing MHI (such as PET, SPECT MRI and evoked potential studies) that may indicate abnormalities in neurophysiology.

Incidence:

It is estimated that 2 million persons in the United States suffer closed head injuries each year (Brown, Fann, & Grant, 1994). Although approximately 500,000 of these injuries are serious enough to require hospitalization most head injures are rated as "mild" (Brown, et al., 1994). Surveys of the number of individuals who develop PTH as a result of MHI are variable ranging from 30% to 80% (Elkind, 1992). Paradoxically, the milder the head injury the more frequently PTH is noted as a symptom.

Most studies have been unable to delineate specific demographic factors related to the incidence of PTH. Guttman (1943) found no relationship of duration and severity of headache to age, gender,

occupation, circumstances of injury, or intellectual abilities. In a fairly recent study of the demographic characteristics of 117 individuals with PTH, Barnat (1986) found that 46% of the sample were male, with a mean age of 37.0 years, and 54% were female with a mean age of 38.4. An average of 12 years of education was reported by the subjects. The most highly represented occupational group was "skilled labor"; 85% claimed to have had good or excellent health before the injury; and 90% doubted or strongly doubted that they had problems with alcohol or drugs. It should be noted that alcohol consumption has been reported in greater than 40% of cases in MHI (Barnat, 1986).

Pathophysiology:

There is increasing evidence to support an organic basis in the pathophysiology of MHI. Organic changes may also play a role in the pathogenesis of PTH, although the specifics are still uncertain. After both mild and severe head injuries, damage to nerve fibers and nerve fiber degeneration are evident. Cerebral circulation is often abnormal after head injuries (Taylor, & Bell, 1966). In many patients, the cerebral circulation is slowed for months or even years after injury and this may accompany prolonged postconcussion symptoms.

In most instances, neurological dysfunction in head injuries is caused by acceleration or deceleration of the brain rather than by the impact itself (Ommaya, & Hersh, 1971). Rotational forces may cause the most significant injuries through shearing of axons. This may explain why injuries with the head free (such as automobile accidents) are more damaging than injuries with the head fixed (such as sports injuries). MRI examination may show areas of diffuse axonal injury at the gray-white matter margin (Gean, 1994).

Surprisingly, many of the symptoms following head injury are associated with mild rather than severe head injuries. In many cases, the incidence of headache is highest in those without loss of consciousness or posttraumatic amnesia (Yagamuchi, 1992). Little anatomical evidence exists to explain the phenomenon. However, impact forces are considerable even in low-speed auto accidents.

Recently, evidence has accumulated to support a neurochemical basis for migraine headache, and the possibility exists that PTH may operate under similar mechanisms (Haas, 1993). Neuropeptides have been found in perivascular nerve fibers and are thought to maintain homeostasis in the cerebral circulation. The neuropeptides manifested in perivascular nerve endings of cerebral vessels, which probably act as neurotransmitters, include neuropeptide Y, substance P, calcitonin-gene-related peptide, and vasoactive intestinal polypeptide. These are believed to be responsible for cerebral vasoconstriction and vasodilatation and the transmission of nociceptor stimuli to the central nervous system. There appears to be a cascade of neurochemical events occurring after experimental head injury, including calcium-mediated dysfunction, excitatory neurochemical release, altered neuromodulator transmission, and disrupted axoplasmic transport (Haas, 1993).

Postconcussion Syndrome:

A variety of postconcussion symptoms may follow MHI in addition to headache. Although these symptoms may differ somewhat in degree, they are surprisingly consistent from patient to patient. The classic postconcussion syndrome (PCS) consists of psychophysiologic, cognitive, and psychosocial symptoms typically observed after head injury. Although it is rare for a patient to present with all of the features of PCS, most patients who present with PTH also experience a few additional symptoms (Bennett, 1988).

The most common symptoms reported include dizziness, fatigue, nausea, weakness, depression, insomnia, attention/concentration disturbances, loss of memory, anxiety, hyperirritability, sensory disturbances, decrease in enjoyment of sex, alcohol intolerance, and temperature intolerance. Often, symptoms occurring later are associated with increased social morbidity as compared with early postconcussion symptoms, which may reflect progressive improvement. In a study of the patterns of symptom reporting, Alves, Colohan, O'Leary, et al. (1986) found that almost 50% of adult patients with MHI were at risk for developing 2 or more somatic or psychophysiologic symptoms.

Headache Types:

Individuals with PTH may develop one of several types of headache. Tension-type, migraine-like, cluster-like, and mixed posttraumatic headache are similar to their nontraumatic counterparts (Haas, 1993). PTH may also occur when the soft tissues of the head are injured or when there is scar formation. The site of injury is often extremely sensitive to finger pressure. Patients may have different types of headaches at different times or a variety of symptoms together that are characteristic of more than one type (Speed, 1986).

Tension-type posttraumatic headache is the most frequent type of headache following MHI. It has been estimated that 85% of patients with postconcussion syndrome have tension-type headaches (Mandel, 1989). They consist of a dull, aching sensation with variable degrees of intensity. Emotionally tense or stressful situations often accentuate headaches. Mixed PTH is also quite common; it usually consists of a combination of tension-type and vascular headaches.

Posttraumatic migraine headaches (PTM) are reported as rare (Mandel, 1989). Clinical features are almost identical to those of nontraumatic migraine. Patients with PTM may be classified as having either migraine with aura or migraine without aura. PTM patients may have a genetic predisposition to the migraine complex and may report migraine in family members. Trauma to the head or neck may trigger the migraine process in a susceptible individual who previously did not have migraine headaches (Speed, 1986). Also, head or neck injuries often increase the severity of headaches in preexisting migraine conditions.

Cluster-like headaches have also been reported following head trauma, with estimates of incidence from 6% to 10% (Duckro, Greenberg, Schultz, et al., 1992). The onset of pain is typically rapid, with a relatively brief duration. Cluster-like PTH, however, may be without the periods of remission that are expected in the episodic variety.

Whiplash injuries, typically caused by motor vehicle accidents, may also result in PTH (Evans, 1992). Whiplash refers to neck hyperextension followed by flexion, which occurs when an occupant of a motor vehicle is hit from behind by another vehicle. Headaches have been reported in 82% of individuals immediately following whiplash injuries (Balla, & Karnaghan, 1987). Most headaches following whiplash injury are tension-type headaches, often associated with cervical muscle injury, greater occipital neuritis, and possibly temporomandibular joint (TMJ) syndrome. Usually, the injury is a myofascial injury with damage to the muscle, ligaments, and connective tissue (Balla, & Karnaghan, 1987).

Other, less frequent types of headache pain following injury include TMJ syndrome and pericarotid syndrome (Friction, 1989). TMJ, a subtype of tension-type headache, usually results from stretching and tearing the ligamentous structures of the jaw joint. The mastoid muscles are usually tender, with pain, clicking, or popping in the involved joint and limitation of jaw opening. Vijayan (1977) described a rare type of PTH, dysautonomic cephalalgia, following injury to the anterior area of the carotid sheath. The headache was typically severe and unilateral, occurred in the frontotemporal area, and was associated with ipsilateral increased sweating of the face and dilation of the ipsilateral pupil.

Psychological Aspects:

After MHI, victims are often faced with a number of new and distressing concerns. Therefore, it is reasonable to assume that psychological disturbances are often a factor in head trauma. Family complaints and tensions are high up to 2 years after injury, primarily because of personality and behavior changes rather than the physical disability (Goethe, & Levin, 1983). As many patients become more aware of their cognitive, social, and occupational difficulties, they be come more distressed.

Patients with PTH may meet DSM-IV diagnostic criteria for posttraumatic stress disorder and require additional treatment. In a study comparing individuals with chronic PTH to other chronic pain groups and a non-pain control groups, results indicated that, in general, individuals with PTH exhibited more psychopathology than individuals with a type of idiopathic headache or control subjects (Hickling, Blanchard, Schwartz, et al., 1992). A wide variety of coping styles appeared to exist within each chronic pain group. Ham, Andrasik, Packard, et al. (1994) hypothesized that the injury itself and possible presence of posttraumatic stress disorder accounted for this difference.

Personality factors usually do not in themselves cause a headache, but they certainly can be involved in symptom development and response to injury. Stable individuals with good coping abilities should tend to adjust better to mild head injury and subsequent treatment than anxious, depressed, or insecure individuals (Ericksen, 1882). Fordyce, Roueche, & Prigatano (1982) suggested that premorbid personality traits appear related to increased psychopathology in some chronic patients with head trauma. It is also probable that individuals with significant psychopathology before the injury would be less motivated to change their condition than individuals who do not evidence psychopathology and who are motivated to "get back to normal."

Treatment:

Headaches that follow MHI are often difficult to treat, but a variety of methods have been useful in managing PTH. Although most medical professionals recognize the importance of medication, psychology has "made in-roads" in the form of biofeedback and psychotherapies, that are being increasingly recognized as helpful for many patients.

The type of medication prescribed would depend on the mechanism responsible for the headache. For patients with PTM, tricyclic antidepressants, sometimes combined with a beta-blocker, have been effective. Patients with daily or chronic tension-type PTH may respond to tricyclic compounds, particularly amitriptyline, imipramine, and doxepin (Haas, 1993). Patients with mixed-type PTH are typically treated with a tricyclic antidepressant; the addition of a beta-blocker, usually propranolol, may be helpful, as well. Headaches resulting from scar formation or associated myofacial trigger points often respond to lidocaine or lidocaine combined with dexamethasone injected into the tender area (Haas, 1993).

Biofeedback. in combination with medication has been beneficial for a number of patients (Adler, Adler, & Packard, 1987). Simply stated the concept of biofeedback is to enable the patient by using relaxation techniques to recognize muscle tension and bring it under voluntary control. Electromyograph biofeedback for specific muscles and thermal biofeedback for vascular components are often used in teaching relaxation. These skills if properly mastered, will often transfer to other areas of the patient's life and will equip the patient to have more control over his or her treatment.

CBT or behavior modification may be necessary for some PTH patients. Many patients simply need support, education, and someone to help rearrange some of the external contingencies in their lives. Others require limited intervention, whereas a few may need long-term psychotherapy (Adler, & Adler, 1987). Some of the issues involved in therapy with PTH patients include depression, anxiety, frustration, excessive expectations, anger, and unresolved grief and loss. Psychotherapy is often very effective in combination with medication and is advantageous in that it allows the patient to exhibit some control over his or her condition.

Many patients with head injuries may be less concerned with obtaining pain relief than with receiving an explanation, support, and reassurance from the clinician about the injury. In a study by Packard (1979) examining what headache patients want, it was found that an explanation of what was wrong ranked higher in importance than pain relief. One of the most frustrating experiences for patients may be to be told by the clinician, "Your neuropsyc. testing, MRI, etc. are normal; there is nothing wrong with you." It is important for clinicians to aid patients in establishing realistic goals (total relief of pain is generally unrealistic) (Haas, 1993). The clinician and patient should work together however, to find the best means for managing the pain. Finally, patients can often be helped to achieve less frequent and/or less severe headaches and better overall management with pacing, coping, and relaxation skills and education about the entire problem.

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