Synopses on Posttraumatic Headache (PTH), Facial Pain, Neck (Cervical Pain) and Related topics

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Revised 17 December 2009

Problems can be encountered clinically and in cases of litigation in the evaluation and appraisal of persistent headache, facial pain and other subjective complaints in patients following head injury however mild. Posttraumatic headache due to a head injury is characterised by a close time relationship with the accident or trauma and need to be differentiated from other neurological and psychiatric disorders.[1]

Epidemiology of Post-Traumatic Headache

Post-traumatic headache (PTH) is a well recognised occurrence following head trauma and is often one of several symptoms of the post-concussive syndrome, hence it may be accompanied by additional cognitive, behavioural, and somatic problems.[2] It is not related to the degree of trauma. After mild head injury, up to 50% of people develop a post concussion syndrome, with headache estimated to be present in 30% to 90% of such patients.[3, 4]

The PTH is considered chronic when it persists after the first two months of the injury. This is common and reported in 60% of patients when these headaches may become a daily occurrence.[2, 6] Chronic PTH is a common condition, often part of the post-concussion syndrome (PCS)[5] which is frequently accompanied by other manifestations of sequelae. These may include cranial nerve symptoms and signs, psychological disturbances, irritability and somatic complaints, and cognitive impairment either singly or in combination after mild head injury.[2-4, 15-16, 18] The symptoms can include dizziness, vertigo, perceptual changes, memory loss, paresthaesias and tinnitus in addition to psychological disturbances.[23]

In a UK study, PCS was accompanied by fatigue, headache, dizziness, irritability, sleep disturbances, poor concentration and poor memory in that order.[21]

Risk Factors in PTH

Risk factors in developing PTH which make people more susceptible include: pre-existing emotional problems,[18] gender (women), those injured by falls, and work based accidents, particularly in large impersonal organisations.[6] Precipitating factors in one study were the presence of sensory problems, lower socioeconomic status, lack of professional qualifications, psychiatric history and history of emotional shock.[23]

Severity of the Head Injury and PTH

The more severe the head or neck injury, the less the likelihood of developing PTH or persistent neck symptoms.

Patterns and Mechanism of PTH

The mechanism of PTH is poorly understood. Trauma-induced headaches are usually heterogeneous in nature, often including both non-specific headaches or that which mimics other forms of headache patterns such as tension-type pain or vasomotor headaches ie migraine-like and cluster headaches together with facial pain.[2,15,16,17, 27] Most headaches are, however, of the tension type, although vasomotor headaches (migraines and cluster headaches) can exacerbate or increase in frequency both acutely or chronically [2,-5, 7]

Rebound-headaches may develop from overuse of analgesic medications, and the occurrence of such may complicate significantly the diagnosis of post-traumatic headache. [2]

PTH in Moderate and Severe Brain Injury

Acute posttraumatic headache syndrome (PTHS) are experienced in 38% of patients with moderate to severe brain injuries. Frontal location was reported in 50% of cases and daily occurrence in 75%. There was no correlation between PTHS and the severity of the injury, emotional, or demographic variables. After the acute phase, PTH symptoms severity decreased in the cohort. Better individual improvement was associated with less anxiety and depression at 6-month follow-up.[29]

Posttraumatic Headaches in Children

Children after minor head injury appear particularly susceptible to severe but reversible neurological symptoms and/or signs. These are manifested in headache, confusion, drowsiness, vomiting, hemiparesis, cortical blindness, or seizures. These neurological episodes are not associated with any identifiable structural brain abnormality on neuro-imaging. It has been suggested that these episodes are due to reactive hyperaemia, a 'benign hyperaemic encephalopathy' mediated via activation of the trigemino-vascular system.[28]

Pathogenesis and Pathophysiology of PTH

The pathophysiology is not well understood but includes biological, psychological, and social factors[15]. Although a small minority are malingerers, frauds, or have compensation neurosis, most patients have genuine complaints.[3, 4] Both organic and neurotic causations are found to contribute to the pathogenesis of symptoms at six weeks.[6]

The organic basis of the syndrome has now become well recognised and abnormalities have been detected with neuropathological, neuropysiological, neuro-imaging, and neuropsychological studies.[15-16,19]

Neuropathological studies

Neuropathological studies suggest the presence of neuronal injury without gross pathology in concussion. Closed head injury of seemingly minor degrees may lead to chronic symptoms, often stereotypic, similar to those following concussion, and they have been described by the term post head trauma syndrome or post-concussional syndrome.[15] Head trauma can, therefore, damage extra- or intracranial peripheral or central nervous structures, hence predisposing to the future development of cluster headaches (CH - see later). In the latter condition (CH) there also is the possibility that previous head injuries may be more frequent amongst these patients because of their lifestyle, which may leave them more exposed to the risk of traumatic events.[10] Lesions of intra- and extra-cranial structures sensitive to pain, are prone to precipitate subjective complaints in the form of headache. Severe craniocerebral injuries with persisting headache may be suggestive of chronic disturbances in cerebrospinal fluid circulation.[14]

Pathophysiology

The physiological mechanisms predisposing to the headache and other symptoms in the PTS are believed to relate to vascular and neuronal disturbances.

Diagnostic Techniques

Neuro-imaging

The use of neuro-imaging has greatly enhanced diagnosis in head-injured patients by providing objective evidence of changes in the brain, however, it has not satisfactorily explained post-traumatic symptoms in the less severely traumatised.[15,16]

Electrophysiological Studies

Electodiagnostic investigations of patients with minor head trauma in the form of brainstem trigeminal and auditory evoked potentials (BTEP, BAEP) and middle-latency auditory evoked potentials (MLAEP) within the first 48 hours following their admission and at 3 months after the injury have showed significantly increased latencies at the initial assessment, thus suggesting disseminated axonal damage. These findings point to an organic diencephalic-paraventricular primary damage that may account for the occurrence of PCS.[20]

Neuropsychological Assessment od PTH

Neuropsychological assessment can be helpful in demonstrating deficiencies in mildly impaired individuals and explain the poor response to headache therapy in some patients suggesting more widespread injury.[15]

Effect on Work by PTH

In a study in the UK, the persistence of symptoms in a significant number of patients with minor head injury complaining of post concussive symptoms contribute to their delay in returning to work.[21] In mild to moderate head injury most patients return to work despite having complaints.[22]

Treatment

The literature on the treatment for post-traumatic headache is scarce.[2] A thorough assessment is required to eliminate structural lesions together with assessment of the psychological status before considering treatment.[15] The principle of management consists of diagnosing the type of headache followed by selecting the appropriate therapy[15] both as in preventive and therapeutic medications. The treatment is within the lines used for treating primary headaches, despite the different aetiologies.[2, 5] Adequate treatment obviously requires both "peripheral" and "central" measures. Although a variety of medication and psychological treatments are currently available, ongoing basic and clinical research of all aspects of mild head injury are crucial to provide more efficacious treatment in the future.[3]

A comprehensive neuropsychological rehabilitation programme may be required.[15] In terms of drug therapy, simple analgesics such as NSAI agents for short-term treatment and tricyclic antidepressants for chronic pain are considered to be effective in patients without structural damage. Other medication regimens used are beta blockers and MAOI.[15,16]

Prognosis in Posttraumatic Headaches

Prognostic studies clearly substantiate the existence of a post-concussion syndrome. The resolution of PTH varied but most often the prognosis is favourable for resolution of symptoms.[1,15,16, 24,25] In most patients, manifestations of the post-concussion syndrome resolve in 3 to 6 months after the injury. In a small minority, however, cognitive deficits persist additional months or years.[23] In minor head and neck injuries from motor vehicle accidents, 70% of patients settled within a few weeks and 30% continued to complain of headaches and/or neck pain.[25] In another study of patients who had PTH that persisted for more than 1 year, approximately 20% did not resolve after the settlement of any pending litigation,[3, 4] and in a small percentage of patients symptoms persisted after 3 years.[15,16]

Recovery usually encompasses the headache, psychological symptoms, and cognitive impairment.[15] Long-term prognosis in general is considered good with the majority of patients recovering after 1 year.[16]

In PTH following moderate to severe brain injuries, nearly all patients with PTH symptoms that persisted into the 6-month follow-up period remained symptomatic at 12-month follow-up. The pattern of improvement in this group levels off by 6 months from the date of initial hospital admission.[29]

Delayed Recovery

If aggressive treatment is initiated early, PTH is less likely to become a permanent problem, once fully established the cycle of ongoing headaches is more difficult to interrupt. [2]

Delayed recovery from PTH may be a result of inadequately aggressive or ineffective treatment, overuse of analgesic medications resulting in analgesia rebound phenomena, or co-morbid psychiatric disorders (eg, post-traumatic stress disorder, insomnia, substance abuse, depression, or anxiety). [2] It has also been found that the prolonged management, extensive physiotherapy and slow court settlement can lead to excessive introspection and prolongation of symptoms. [25] Poor response to headache treatment in some patients suggest a wide spread injury. [15]

Risk Factors in the Prognosis of PTH

Risk factors for persisting sequelae include age over 40 years; lower educational with lack of professional qualifications, intellectual, and socioeconomic level; female gender; alcohol abuse; prior head injury; multiple trauma.[3, 4, 23]

Symptoms and Court Settlement

The notion that litigation prolongs the duration of the illness has been proven to be invalid.[25] Most patients with litigation or compensation claims are not cured by a verdict.[3, 4] In a study based on telephone interviews conducted on 50 patients after their court settlement in the US, all patients interviewed continued to report persistent headache symptoms one year or more following legal settlement. Improvement in headache pattern after legal settlement was only reported by four patients.[26]

Other Headache, Facial Pain Types

Cluster headache

This is a form of neurovascular headache. Attacks usually are severe and unilateral and typically are located at the temple and periorbital region. The pain is usually associated with ipsilateral lacrimation (eye watering), nasal congestion, conjunctival injection, miosis, ptosis, and lid oedema. Each headache is brief in duration, typically lasting a few moments to 2 hours.

Cluster refers to a grouping of headaches, usually over a period of several weeks. There are two existing forms of cluster headache; (1) episodic clusters with attack phases lasting 4-16 weeks followed by a cluster-free interval of 6 months to years, and (2) chronic form, in which the cluster-free interval is less than 1 week in a 12-month period. The condition can be distinguished clinically from other forms of migraine in that:

Cluster headaches occurs predominantly in the fourth to sixth decade of life and are more common in men than women in a ratio of 5:1. The condition is characterised by (1) unilaterality in the oculotemporal region (between the eye and temple); (2) sudden onset as a severe burning sharp pain; (3) nocturnal occurrence; (4) brief duration usually 2 to 4 hours; (4) clustered occurrences several times in 24 hours often daily; (5) seasonal occurrence usually autumn and spring; (6) a tendency of the patient to pace about in anguish in contrast to migraine; (7) isolated ipsilateral autonomic phenomenon such as lacrimation, conjunctival injection (red eye), sweating, rhinorrhea (running nose) and Horner syndrome (ptosis - droopy lid; small pupil; anhydrosis - reduction in sweating on the affected side of the face; and others).[8]

Cluster Headache and Trauma

A relation between head trauma and cluster headache is frequently described in the literature.[12] However, it has been thought that these associations are casual and that post-traumatic cluster headache is a very rare occurrence. [9]

There are also a variety of causes other than trauma that mimic cluster headache. These secondary causes include infections, tumours, vascular abnormalities. In

addition, other trigeminal autonomic cephalgias (facial pain and headache resulting from the third trigeminal nerve) can occasionally be difficult to distinguish from primary cluster headache[10] and the possible influences from cervical and masticatory structures in the development of cluster or cluster-like headache.[11, 12] Other causations have also been described [13]

Face / Mid Face Trauma

Persistent headache and/or concentration difficulties are described in isolated upper midface area (mainly the naso-orbito-ethmoidal region) with / without associated central midface injury.[24]

Cervical causation of PTH

Cervical pain is a frequent accompaniment of post-traumatic headache. [2, 27] Possible connections between head or neck injuries and headache have been addressed and neck injury should be excluded in all cases of PTH.[15,17] In a rare cases of PTH following minor head trauma causing exertional headaches, Chiari type-1 mal-formation was found at the cranio-cervical junction without bony occipito-cervical dysplasia.[15]

Tempro-mandibular joint (TMJ) pathology

Pathology of the tempro-mandibular joint can produce facial pain. Inflammatory and degenerative changes of TMJ can develop after facial trauma.[18]

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