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A 26-Year-Old Male with Post-Traumatic Cephalgia: Case Report

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Abstract

Post-Traumatic Headaches (PTH) is the most frequent complaint after Traumatic Brain Injury (TBI). According to ICHD-3 Post-Traumatic Headaches (PTH) is the most common secondary headache disorder, accounting for around 4% of all headache symptoms that begin in 7 days after head injury, craniotomy or post-traumatic awareness. The main risk factors for PTH include a history of migraines or headaches, female gender, younger age, more severe head injury, and concurrent psychological symptoms, such as anxiety and depression. The clinical profile of PTH varies based on headache onset, duration, and severity. Pharmacological treatment often consists of analgesics and nonsteroidal anti-inflammatory drugs, tricyclic antidepressants, or antiepileptic drugs. PTH treatment has two components: acute treatment and prophylaxis. Analgesics (nonsteroidal anti-inflammatory drugs [NSAIDs]) or certain anti-headache medications (triptans) are used for acute treatment.

Introduction

Post-Traumatic Headaches (PTH) are a common sequelae of traumatic brain injury and can develop into a chronic condition (Ashina et al., 2019). Post-Traumatic Headaches are classified by the International Classification of Headache Disorders (ICHD) as secondary headaches occurring seven days after injury or trauma, recovery of consciousness, and/or ability to report pain. The classification also divides PTH into acute headache associated with traumatic head injury, where the headache resolves within 3 months of onset of illness, and persistent headache associated with traumatic head injury, where the headache persists for more than 12 weeks (Guglielmetti et al., 2020; Metti et al., 2020; Tessler & Horn, 2025). Population-based data suggest that the lifetime prevalence of this disorder is estimated to be 4.7% in men and 2.4% in women (Ashina et al., 2021; Grangeon et al., 2020).

According to the type of pain and the severity of brain damage, the prevalence rate of chronic pain after brain injury ranges from 10% to 95%. Patients with brain injury may experience chronic pain due to various causes (Lambru et al., 2021). Suppose the post-traumatic headache lasts more than three months after the injury. In this case it is called chronic post-traumatic headache (CPTHA) (Tedyanto et al., 2023). The Centers for Disease Control (CDC) reports that TBI causes nearly 1.4 million emergency room visits, 275,000 hospitalizations, and 52,000 deaths per year. Mild TBI accounts for most non-lethal events, and motor vehicle accidents are the most common cause of injury (Mavroudis et al., 2023). Traumatic Brain Injury (TBI) is a leading cause of morbidity and disability. Approximately 80% of nonfatal TBIs are classified

as mild TBI. Mild TBI usually correlates with motor vehicle accidents (45%), falls (30%), workplace accidents (10%), recreational accidents (10%), and assaults (5%). Approximately 50% of individuals who experience mild TBI are aged 15-34 years (Russo et al., 2014; Susanti, 2020).

Acute post-traumatic headache has features of migraine or tension-type headache and usually resolves within 3 months. Within the first 72 hours, clinicians should assess for more serious complications including intracranial bleeding (Mavroudis et al., 2023). According to the Centers for Disease Control and Prevention, symptoms following traumatic injury (known as post-concussion symptoms) can be grouped into four categories physical (headache, dizziness, fatigue, imbalance, photophobia), cognitive (difficulty in focusing, concentration, and memory), and emotional (irritability, depression, and anxiety) (Piantino et al., 2019).

To diagnose PTH is based on history taking and physical examination (Dwyer & Zasler, 2020). Successful management of Post-Traumatic Headaches (PTH) involves a customized approach through a combination of pharmacological and non-pharmacological therapies (Kamins, 2021). Migraine-like or possibly migraine-like headaches are commonly found in Post-Traumatic Headaches (PTH) patients. Analgesics, nonsteroidal anti-inflammatory agents (NSAIDs), triptans, ditan, and gepant are prescribed for the treatment of acute migraine and can be used to treat migraine-like PTH (Blumenfeld et al., 2022). Mechanisms of migraine and TBI appear to involve impaired descending pain modulation, neurometabolic changes, neuroinflammation, cortical spreading depression, and calcitonin gene-related peptide (CGRP) release seen in both pathologies (D'Onofrio et al., 2014; Lambru et al., 2021; Kamins et al., 2024).

The development and persistence of PTH may be more related to neuroinflammation and activation of the trigeminal system involved in migraine and other primary headache disorders than to the underlying mechanisms of TBI (Wang et al., 2022; Belyaeva et al., 2022). TBI may cause direct effects of concussion, cerebral vascular damage, and axonal dismemberment, as well as a secondary cascade of metabolic and cellular excitotoxic and inflammatory changes that may drive the development of PTH. The similarity between PTH and migraine is supported by the observation that patients with PTH who have no previous history of migraine show hypersensitivity to CGRP. CGRP is a neuropeptide that can mediate trigemino-vascular pain transmission and trigger migraine attacks. CGRP antagonists are a class of drugs used in the acute and preventive treatment of migraine. A study of patients with PTH found that 28% experienced a 50% reduction in days with moderate or severe headache after open-label treatment with erenumab, a CGRP receptor antagonist (Mavroudis et al., 2023).

Management of Post-Traumatic Headaches depends on the severity of the disorder and its progression over time. Individuals who experience monthly headaches for several days with mild to moderate pain intensity may be managed with simple analgesics purchased over the counter (e.g., non-steroidal anti-inflammatory drugs and paracetamol). If self-medication is ineffective or headache episodes become increasingly frequent, clinical management is primarily performed by primary care providers, through whom preventive therapy can be initiated. What needs to be emphasized is the need for realistic goals to be agreed upon. Any therapeutic approach aims to effectively manage post-traumatic headache and is not considered a cure (Piantino et al., 2019). Patient education is key and necessary to achieve treatment compliance. Referral to specialist care should be considered if the patient is difficult to treat or if clinical management is complicated due to comorbidities (Ashina et al., 2021).

Methods

This study is a case report on Cephalgia Post Trauma Headaches. The implementation of the study has obtained informed consent from the patient and has been approved by the ethics committee.

Case Report

A 26-year-old man came with a complaint of headache. This headache was felt since the last 3 days. Complaints accompanied by dizziness that increases when the patient changes position from sitting to standing. was no nausea, no vomiting. Diarrhea is not present. Fever is not present. The patient denied any complaints of double or blurred vision, half weakness, tingling sensation, slurred speech, pursed lips, walking disorder, seizures, projectile vomiting, and weight loss. The patient denied any complaints of fainting, forgetfulness, and anxiety or depression. There was a history of trauma one day before admission, namely the patient fell from a tree and the back of his head hit the ground. History of stroke was denied. History of DM and HT was denied. History of treatment for 6 months was denied.

The patient's vital signs were within normal limits. No abnormalities were found on physical examination. Global or focal neurological deficits were not found on neurological examination. Laboratory examination and head imaging found no abnormalities. CT SCAN examination showed no hematoma or brain swelling and persistent cavum septum pallecidum.

Based on anamnesis, physical examination and supporting examination, the patient was diagnosed with Post Traumatic Cephalgia and Acute Vestibular Syndrome. And given therapy IVFD RL 20 tpm, Mecobalamin 500 mcg/12 hours / intravenously, Ketorolac 1 amp / 12 hours / IV, Flunarizin 5 mg / 12 hours / oral, Dimenhydrinate 50 mg / 12 hours / oral, Omeprasole 1amp / 12 hours / IV, Ondancentron 1amp / 12 hours /.

Result and Discussion

Post-Traumatic Headaches are non-degenerative, non-congenital disorders of the brain resulting from external mechanical forces, which cause permanent or temporary impairment cognitive, physical, and/or psychosocial functioning, accompanied by decreased or altered consciousness. Approximately 1.7 million people suffer TBI each year in the United States mainly due to falls, motor vehicle traffic accidents, and direct blows to the head. The most common areas of pain are the temples (82% of individuals), followed by the forehead (76.5%), neck (76%), back of the head (53%), eyes (47%), and vertex (29%) (Defrin, 2014).

The diagnostic criteria for PTH are outlined in the International Classification of Headache Disorders, 3rd edition (ICHD-3) (Ashina et al., 2019). In section 2 of the ICHD-3, which categorizes secondary headaches, 3 types of PTH are defined: 1) headache after traumatic injury to the head, 2) whiplash, or 3) craniotomy. These types are each subdivided into subcategories of acute (lasting <3 months) or persistent (lasting ≥3 months) (Ashina et al., 2019). The ICHD-3 diagnostic criteria for PTH states that the headache should be reported as occurring within 7 days of injury or after regaining consciousness or the ability to perceive or report headache (Schwedt, 2024) . In this case report, the patient complained of a headache that was felt 3 days after the trauma.

Most people experience headaches daily or weekly, while a smaller percentage only experience them once a month or less frequently. Headaches are said to worsen during persistent episodes, reach a very high intensity (VAS or NPRS= 8-10, where a value of 0 means no headache, 1 is mild headache, and 10 is very severe headache), and are painful enough to prevent activity. People with CPTHA may also experience neck pain, which is described as a feeling of muscle spasm and tightness in the neck, especially in the posterior area. Knowledge regarding the pathogenesis of post-traumatic headache is lacking. However, the origin of PTHA is explained by several ideas. These hypotheses include altered neurometabolic processes, poor descending regulation and activation of the trigeminal sensory system. There may be several overlapping pathways involved in PTHA. PTHA may result from chronic sensitization of nociceptors in intra and/or extracranial peripheral tissues, from cranial nerve damage, or both. PTHA may result from damage to the spinothalamic/thalamocortical pathway and its consequences, as in

central pain, from altered central modulatory control of nociceptive input, or both (Defrin, 2014; Octina & Kurniawan, 2023).

Similar to other primary headaches, post-traumatic headaches are diagnosed clinically. Patients with a history of head injury and headache symptoms require additional imaging. CT of the head without contrast can be performed to rule out the possibility of acute brain hemorrhage, especially in people. The routine laboratory and imaging diagnostics in this case report revealed no abnormalities. Brain MRI showed no structural changes in the patient (Roper et al., 2017).

Post-traumatic headache is managed and treated using a multimodal approach, including oral medication, musculoskeletal manipulation and treatment, interventional techniques, and behavioral therapy. A number of studies have examined oral and intravenous medications for acute and preventive care. NSAIDs as first-line acute treatment for tension-type PTH and migraine phenotypes, followed by aspirin-paracetamol-caffeine (for both phenotypes) or triptans for migraine phenotypes. For individuals requiring prophylactic treatment, amitriptyline, mirtazapine, or venlafaxine is recommended for phenotypes, and atenolol, bisoprolol, candesartan, metoprolol, propranolol, or amitriptyline for migraine phenotypes (Roper et al., 2017).

Conclusion

Post-Traumatic Headaches (PTH) continue to be a complex and frequently encountered challenge. They encompass a variety of presentations that share a common etiology yet vary in their clinical basis, pathophysiology and clinical course. The current classification using the ICHD-3 has significant limitations that require a reassessment of the classification methodology. The importance of an adequate trauma and headache history and relevant headache physical examination cannot be overstated in the context of obtaining an accurate diagnostic picture and improving the efficacy of recommended treatment. The varied clinical picture may resemble tension-type headache and migraine. The mechanism of CPTHA is still poorly understood; however, there is evidence that the disease may develop due to intracranial/peri-cranial tissue injury leading to chronic local sensitization. Routine administration of ibuprofen and acetaminophen or ibuprofen alone is effective in reducing post-traumatic headache symptoms during periods of pain.

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