Tension-type Headache and Migraine as Manifestations of Chronic Post-Traumatic Headache

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ABSTRACT

Introduction: A subsequent headache within seven days of a head injury (or after regaining consciousness after the head trauma)is referred to as a post-traumatic headache (PTHA); it is referred to as chronic or chronic post-traumatic headache (CPTHA) if it lasts longer than three months after the injury. **Case :** A 17-year-old male with headache since 3 months ago, 3 days after suffered a blow to his left head from falling from a chair. At that time, the patient fainted for about 15 minutes but had no complaints after regained consciousness. Pain is felt on the left side of the head, throbbing, mild-moderate intensity, and feels heavier with a loud sound or a too-bright light. **Discussion:** Post-traumatic headache is clinically diagnosed. Laboratory and routine diagnostic imaging studies are unnecessary and have minimal clinical utility. **Conclusion:** Chronic post-traumatic headaches often occur, especially after minimally traumatic brain injury. The clinical picture is variable and may be similar to tension-type headaches and/or migraines.

Keywords: Migraine, post-traumatic headache, tension-type headache.

ABSTRAK

Pendahuluan: Nyeri kepala dalam tujuh hari setelah cedera kepala atau setelah sadar kembali dari trauma kepala disebut nyeri kepala pasca-trauma (*post-traumatic headache*/PTHA); disebut sakit kepala pasca-trauma kronis atau kronis (CPTHA) jika berlangsung lebih dari tiga bulan setelah cedera. **Kasus:** Seorang laki-laki berusia 17 tahun dengan keluhan nyeri kepala sejak 3 bulan, 3 hari setelah kepala kiri terbentur karena jatuh dari kursi. Saat itu, pasien pingsan sekitar 15 menit, tidak ada keluhan setelah sadar. Nyeri dirasakan di sisi kiri kepala, berdenyut, intensitas ringan-sedang, terasa lebih berat jika ada suara keras atau cahaya terlalu terang. **Diskusi**: Nyeri kepala pasca-trauma didiagnosis secara klinis. Laboratorium dan studi pencitraan diagnostik rutin tidak diperlukan dan memiliki utilitas klinis minimal. **Simpulan**: Nyeri kepala pasca-trauma kronis sering terjadi, terutama setelah cedera otak traumatis minimal. Gambaran klinisnya bervariasi dan dapat mirip nyeri kepala tipe tegang dan/atau migrain. **Eric Hartono Tedyanto, I Made Oka Adnyana, I Putu Eka Widyadharma. Sakit Kepala Tipe Tegang dan Migrain sebagai Manifestasi dari Sakit Kepala Kronis Pasca-Trauma**.

Kata kunci: Migrain, sakit kepala pasca-trauma, sakit kepala tipe tegang.

INTRODUCTION

Patients with brain injuries may experience chronic pain due to various causes; its prevalence rates range from 10 to 95%.[1] A subsequent headache within seven days of a head injury is referred to as a post-traumatic headache (PTHA) (or after regaining consciousness after the head trauma). If a post-traumatic headache lasts longer than three months after the injury, it is referred to as a chronic post-traumatic headache (CPTHA).[2] CPTHA is probably the most prevalent type of pain following mild brain damage, but its cause and course of treatment are yet unknown.[3] Clinical characteristics and neurological examination are the keys to diagnosing CPTHA.[3]

CASE

A 17-year-old male with headaches for three months, three days after a traumatic injury to his left head from falling from a chair, fainted for about 15 minutes; he regained consciousness with no complaints of a headache. Headache is on the left side of the head, throbbing, with mild-moderate intensity, and aggravated by loud sound or too-bright light; it is felt almost every day, approximately 10 times daily, and lasts about 5-10 minutes and interferes with his daily activities.

The headache increases with his daily activity with numeric pain rating scale (NPRS 3-4/10) and decreases after rests with NPRS 1/10. The patient also reported nausea and vomiting. A flash of white light was once reported before the headache. Persistent tension and pulling pain are also felt in the neck and back of the head. Paracetamol did not give any improvement. The patient denied any symptoms of double or blurred vision, half-body weakness, tingling sensation, slurred speech, impaired walking, convulsions, projectile vomiting, and weight loss. The patient denied fainting, forgetfulness, anxiety, or depression.

The patient's vital signs were within normal

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limits. No abnormalities were found on physical examination; global or focal neurological deficits were not found. EEG showed no abnormalities. Laboratory examinations and head imaging were not indicated. A combination of paracetamol and ibuprofen, and amitriptyline gave an improvement on day 3 (NPRS 2-3/10). The patient fits post-traumatic headache according to ICHD-3 criteria. A secondary headache related to the trauma or injury (post-traumatic headache) is coded when a new headache develops for the first time in close temporal proximity to trauma or injury to the head and/or neck.

DISCUSSION

Post-traumatic headaches are defined by the most recent International Classification of Headache Disorders (ICHD-3) as a secondary headache with onset within seven days following trauma or injury, seven days after regaining consciousness, or seven days after regaining the ability to sense and report pain.[15] However, this criteria has recently been contested, as some individuals may report symptoms between three months and one year following a shock or injury. The ICHD-3 temporal criteria lack specific phenotypic diagnoses that would enable clinicians to treat and manage the various forms of PTHA.[4] Post-traumatic headaches are secondary headaches that develop after a traumatic brain injury. Mild to moderate symptoms like a tension-type headache may accompany a post-traumatic headache. Post-traumatic headaches may be intermittent or recurrent. If headaches remain for more than three months following a concussion, it is referred to as chronic post-traumatic headache.[13]

Persistent post-traumatic headaches and migraines are related to brain anatomy changes, possibly indicating distinct underlying pathophysiology. Compared to migraine patients, persons with persistent post-traumatic headaches have differences in regional volumes, cortical thickness, brain surface area, and brain curvature. The structures of the right lateral orbitofrontal lobe, left caudal middle frontal lobe, left superior frontal lobe, left precuneus, and right supramarginal gyrus varied between groups.[12]

People with tension-type CPTHA experience mild to moderate bilateral pain with pressing, tugging, and dull quality, worse by exertion, stress, and emotional strain. People with the migraine-type of CPTHA experience severe pounding, throbbing, and stabbing unilateral pain and are made worse by physical activity. Patients may also express sensitivity to loud noises or bright lights (photophobia and phonophobia). Less than one-third of CPTHA patients experience "mixed headaches," which are symptoms of the various forms of headaches that overlap.[4,5] It is coded as a secondary headache related to the trauma or injury (post-traumatic headache) when a new headache develops for the first time in close temporal proximity to trauma or injury to the head and/or neck. This still applies whether the newly developed headache resembles any of the primary headache diseases listed in Section 1 of ICHD-3.[15] In this case report, the patient complains of mixed headaches for the first time related to head trauma. The patient complains of tension-type headaches and headaches resembling migraines. The patient fits the diagnosis criteria of post-traumatic headache.

Most people get daily or weekly headaches, whereas a smaller percentage experience them only once a month or less frequently. Headaches were said to steadily worsen during the episodes, reach very high intensities (VAS or NPRS = 8–10), and be painful enough to prevent activities. People with CPTHA may also experience neck pain, described as a feeling of muscular spasm and tightness in the neck, particularly in the posterior area.[2]

There is still a lack of knowledge on the pathogenesis of post-traumatic headache (PTHA); several hypotheses include altered neurometabolic processes, poor descending regulation, and trigeminal sensory system activation with the probability of several overlapping pathways.[6] Schwedt et al. showed that patients with PTHA had structural variations in cortical thickness and brain volume.[14] Diffuse axonal damage after a brain injury may lead to structural remodeling of cortical and subcortical areas in the somatosensory and insular cortex, downregulating the neuromodulation of pain-modulating pathways.[2]

The hypothesis for the origin of PTHA centres on abnormalities in the neurometabolic system. Damage to cells caused by physical stress results in the uncontrolled release of ion-exchange neurotransmitters.[3] Neuronal activity-induced metabolic stress (lactate and free radicals) causes axonal damage (secondary axotomy). Cortical spreading depression (CSD) is an electrical process during the migraine aura.[7] In CSD, excessive glutamate and potassium release caused by cellular depolarization increases nerve excitability and activation of the trigeminal sensory system. CSD may contribute to PTHA by causing additional brain damage after brain injury.[7] Due to neuroinflammation, the central nervous system (CNS) can become more excitable, causing CSD and activating the trigeminal sensory system. Due to overlapping communication pathways, nociceptive signals from upper cervical afferents can activate the trigeminal system. The convergence of cervical afferent and trigeminal nerve pathways supports the discovery that therapies for cervical neck pain generators can contribute to PTHA relief.[4]

Similar to other primary headaches, post-traumatic headache is clinically diagnosed. Laboratory and routine imaging diagnostics are unnecessary and have limited clinical utility. A head CT scan without contrast may be performed to rule out acute cerebral hemorrhage, particularly in the elderly and patient with neurological deficit.[8] Because this case is young and has no focal or generalized neurologic deficits, imaging tests are unnecessary.

Post-traumatic headaches are managed and treated using a multimodal approach, including oral medications, musculoskeletal manipulation and treatment, interventional techniques, and behavioral therapy; a multidisciplinary approach was the most effective in treating PTHA.[9] Cognitive-behavioral therapy (CBT), biofeedback, progressive muscle relaxation therapy, acupuncture, and physical therapy were investigated.[9] Nonsteroidal anti-inflammatory drugs (NSAIDs), triptans, and intravenous antiemetics are used as acute therapeutic options.[10] In preventive regimens, tricyclic antidepressants (TCAs), anticonvulsants, and gabapentin are all included. Most patients benefited from amitriptyline.[16] Cushman et al. discovered that patients taking either gabapentin or amitriptyline improved compared to those who did not receive treatment.[10]

Patients who took acetaminophen/paracetamol, ibuprofen, or both had fewer headache days, episodes, and intensities than those who did not receive analgesic drugs. Patients taking ibuprofen and acetaminophen (79%) or ibuprofen alone (61%) were more likely to return to school one week after the accident than those taking acetaminophen (33.3%) or aspirin (21.1%).[11] Regularly administering ibuprofen and acetaminophen together or separately to treat headaches during the acute concussion period appears to reduce the number of headache days, the number of headache episodes per day, the intensity and depth of the headache, and the difficulty of returning to school one week after the injury.[11]

CONCLUSION

Chronic post-traumatic headaches are common, especially after mild brain injury. The clinical presentation may resemble tension-type headaches and migraines. The mechanism of CPTHA is poorly understood; it can develop due to intracranial/peri-cranial tissue injury that induces chronic local sensitization. Routine administration of ibuprofen and acetaminophen or ibuprofen alone effectively reduces the symptoms of post-traumatic headache during the acute concussion period.

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