

## Management of Chronic Posttraumatic Headache: A Multidisciplinary Approach

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**Although the definitions and possible treatments of posttraumatic headache have been discussed in the literature, treatment of this condition is not standardized. In addition, though various reviews and case studies have supported the use of spinal manipulation in the treatment of tension-type headaches and migraines, few describe the use of osteopathic manipulative treatment for patients with posttraumatic headache. In describing a 38-year-old woman with posttraumatic headache, we illustrate a multidisciplinary approach—including the use of osteopathic principles and practice—to treating patients with posttraumatic headache.**

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Headache is one of the most common complaints in patients with traumatic brain injury (TBI).<sup>1</sup> The overall incidence of TBI is 1.8 million cases per year, of which 30% to 90% have posttraumatic headache. Approximately 2% of the US population is disabled secondary to posttraumatic headache.<sup>2</sup> Head and neck injuries account for 15% of chronic daily headache cases.<sup>3</sup> In addition, nearly 45% of head and neck injuries are accompanied by chronic headache at 6 months. At 1 year, head pain in 20% of individuals becomes permanent.<sup>2</sup>

Despite the prevalence of TBI and its potential long-term effects, at least 25% of people with mild TBI seek no medical attention, compared with 14% who visit their primary care physician's office or a clinic.<sup>1</sup> The remaining patients are seen in the emergency room for mild to moderate head trauma, where they undergo some form of neuroimaging—typically a computed tomography (CT) scan. If the symptoms of TBI persist and the patient is evaluated later by another physician, then

magnetic resonance imaging (MRI) scans will likely be taken. However, neither of these studies can predict whether posttraumatic headache or postconcussional syndrome will develop in a patient.<sup>4</sup>

When results from imaging studies are normal, physicians may feel limited in their treatment options for patients with symptoms of TBI. However, if a multidisciplinary approach is considered, myriad therapy options are available. For example, medications—abortive and preventative—may improve headache symptoms.<sup>5</sup> Spinal manipulation, especially in conjunction with pharmacotherapy, may alleviate patient pain.<sup>6,7</sup> Although few studies reference the use of manipulation in posttraumatic headache,<sup>8</sup> an osteopathic structural examination can be used to evaluate patients for somatic dysfunction and, if appropriate, the need for subsequent osteopathic manipulative treatment (OMT).<sup>4</sup> Patients' emotional states should be assessed and treatment provided for those with depression, anxiety, or stress that may coincide with and exacerbate persistent pain.<sup>9</sup> Likewise, physicians should evaluate patients for food and stress triggers.<sup>9</sup> Depending on the distribution and timing of patients' pain, injections and nerve blocks should be considered as well.<sup>10</sup> Additionally, because muscular and emotional components may contribute to chronic headache, physical therapy with biofeedback and modalities should be considered as adjunctive treatments.<sup>4,10</sup>

We present the case of a 38-year-old woman with chronic headache who presented to the University Pain Care Center, a multidisciplinary center with specialists in headache treatment, physiatry, orthopedics, neuromuscular medicine/osteopathic manipulative medicine (NMM/OMM), and physical therapy at the University of Medicine and Dentistry of New Jersey-School of Osteopathic Medicine in Stratford. Patients such as the one described in the present report are often inter-referred for an integrated treatment plan, as is necessary for those with posttraumatic headache.

### Report of Case

A 38-year-old Ukrainian woman presented to the NMM/OMM specialist (M.K.C.) at the University Pain Center in Stratford, NJ, complaining of right-sided frontal headaches. The patient stated the headaches began 3 months ago after accidentally being hit in the face at the right frontal, maxillary, and zygomatic bones with a radiography machine at her dentist's office. She denied any loss of consciousness at the

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time of the incident and described the pain as a “constant achy” to “stabbing” pain primarily over the right eye and right zygomatic bone.

She complained of intermittent blurred vision when the pain became “stabbing.” She also complained of intermittent tinnitus. The pain typically became worse as the day went on but otherwise had no modifying factors. On a subjective rating scale of zero (no pain) to 10 (worst pain), the patient rated her pain as eight at best, nine on average, and 10 at worst.

Before presenting to the University Pain Center, the patient had seen an ophthalmologist and a neurologist. Results from a radiographic scan of the skull, CT scan of the brain, and MRI scan of the brain and cervical spine were normal. Likewise, results from slit lamp and neurologic testing were normal. The patient had been given a methylprednisolone acetate dose pack (tapered dose of 24 mg to 0 mg in 6 days) and acetaminophen (5 mg)/oxycodone hydrochloride (325 mg) for pain but had minimal relief.

The patient’s medical history was clinically significant for infertility, for which an exploratory laparoscopy was conducted. She had chronic right shoulder pain after a motor vehicle collision, but that pain resolved before being hit with the radiography machine at the dentist’s office. The patient had no other clinically significant medical, surgical, or family history, and she was not on any medication other than that previously described.

On physical examination, the patient’s height was 5 ft, 6 in; weight, 155 lbs; blood pressure, 130/80 mm Hg; body temperature, 98.6°F; heart rate, 76 beats per minute; and respiratory rate, 16 breaths per minute. Spinal nerves C1 through C7 were grossly intact. She had 5/5 muscle strength with 2/4 deep-tendon reflexes of the upper extremities. Heart and lung sounds were normal.

An osteopathic structural examination revealed clinically significant findings: right cranial torsion, compressed right zygomatic bone, temporomandibular joint (TMJ) dysfunction, and tender points following the right orbicularis oculi muscle as well as across the greater wing of the sphenoid bone and the right temporal muscle. Vertebrae C3 through C5 were rotated right sidebent right. She had a hypertonic right levator scapulae muscle as well as having vertebrae T1 through T3 rotated left sidebent right and T5 through T8 rotated right sidebent left.

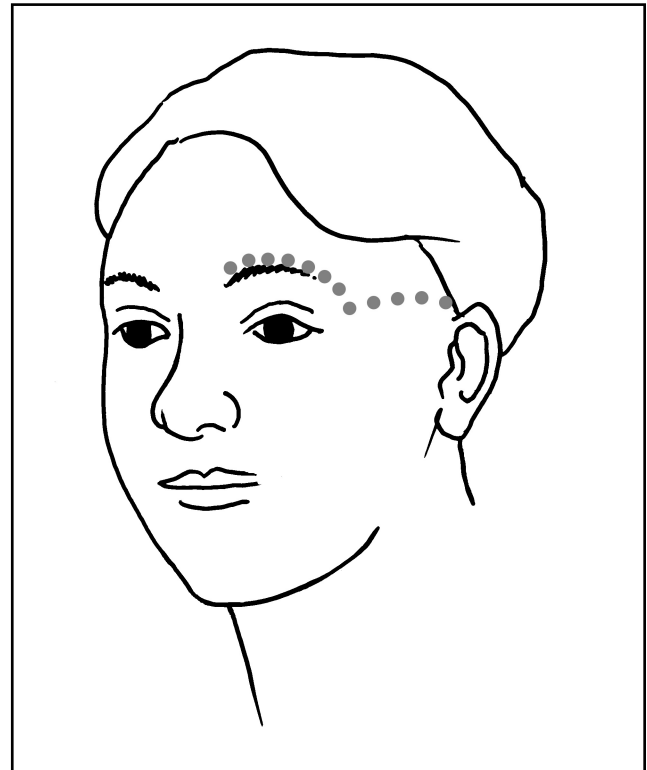
On initial evaluation, the patient’s somatic dysfunctions were treated using OMT techniques. Cervical and thoracic somatic dysfunctions were treated with muscle energy and high-velocity, low-amplitude (HVLA) techniques; cranial sacral techniques were used to manage cranial torsion and zygomatic bone compression; and TMJ dysfunction was managed using direct intra-oral and muscle energy techniques.

The patient’s reported pain decreased from a rating of 10 (pre-OMT) to eight (post-OMT). Celecoxib (200 mg/d) and

acetaminophen (325 mg)/butalbital (50 mg)/caffeine (40 mg) every 4 hours as needed were prescribed for the patient to manage pain. She was referred to the headache specialist for an appointment 6 weeks later.

The patient followed up with the NMM/OMM specialist (M.K.C.) 1 week after the initial OMT session and reported mild improvement. Using the same pain scale described earlier, she rated her headaches as seven at best, eight on average, and 10 at worst. Because of periorbital and temporal tenderness, the patient was again treated with the OMT techniques previously described and was additionally treated with another direct-pressure OMT technique known as *progressive inhibition of neuromuscular structures* (PINS).<sup>11</sup>

The PINS technique was applied over the right periorbital ridge, right greater wing of the sphenoid bone, and preauricular soft tissue (Figure 1) as it followed the distribution of the patient’s tenderness and mild “bogginess” of the soft tissues. The patient was treated with the course of OMT techniques, including the PINS technique, twice more over 4 weeks. She reported that her pain level reduced to an average and best of five on the pain scale, with pain worse (rated eight of 10) in the morning.



**Figure 1.** Distribution of tender points managed with progressive inhibition of neuromuscular structures technique. The patient was a 38-year-old woman who presented with complaint of headache. Printed with permission from the University of Medicine and Dentistry of New Jersey-School of Osteopathic Medicine.

At this point, approximately 5 months after headache onset, the patient was seen by the headache specialist (L.L.M.). Symptoms included daily right temporal, frontal, and nasal throbbing pain with associated nausea and, rarely, vomiting. During a headache she experienced photophobia, phonophobia, increased pain with physical exertion, and incapacitation requiring bedrest. They were not associated with neurologic prodromes (ie, aura) and lasted most of the day.

As a result of her headaches, the patient missed 27 days of work in the 3 months preceding her visit. Her migraine disability assessment score was 137. According to this measurement tool, a score of 21 or higher corresponds to grade IV, which is defined as "severe disability." Her headaches met ICHD-II criteria<sup>12</sup> for migraine without aura. However, because she first developed head pain 1 day after head trauma and denied headaches prior to this event, she was diagnosed as having chronic posttraumatic headache.

At the time the patient presented to the headache specialist, she had weaned herself from 10 tablets of acetaminophen/butalbital/caffeine daily 3 months ago to 1 to 2 tablets daily. Physical examination by the headache specialist (L.L.M.) was positive for right TMJ tenderness on palpation, right brow droop, change in sensation of the right temple, and pain on right eye abduction. Imaging studies, including MRI and magnetic resonance angiography (MRA) of the brain, were unremarkable.

At her initial visit with the headache specialist (L.L.M.), the patient was educated about headache treatment rationale, including rebound headaches with overuse (>2 d/wk) of nonpharmacologic, abortive, and preventive treatments. She was directed to stop all caffeine and butalbital medication and start nortriptyline hydrochloride (10 mg at bedtime), to be increased weekly. Diclofenac sodium was prescribed for mild pain, an isometheptene compound for severe pain, and ondansetron for nausea associated with headache. Serotonin (1B/1D) agonists (triptans) were not prescribed because the patient was undergoing workup for syncopal episodes.

Because of mental slowing caused by nortriptyline, the patient was transitioned to divalproex sodium ER (250 mg/d). A higher dose was not used because it would induce nausea in the patient. Her headaches improved, and during the next year, her headache frequency reduced to twice a month, and she was functional when she took her isometheptene compound abortively.

The patient was referred to the physiatrist (R.H.) for a sphenopalatine ganglion block to manage pain above her right eye and zygomatic bone. This treatment alleviated the patient's pain over the zygomatic bone completely.

Finally, the physiatrist (R.H.) recommended botulinum toxin injections at the patient's right temporalis muscle. However, the patient's insurance would not precertify this treatment and the patient was unable to afford the treatment directly.

The patient was sent to physical therapy where thera-

peutic ultrasound, heat, and range-of-motion exercises were executed with some therapeutic benefit. The patient was told to stop all caffeine intake, whether in medications or foods.

### Comment

Posttraumatic headache, sometimes under the topic of mild TBI, has been defined in several reports, including the International Classification of Headache Disorders, 2nd edition (ICHD-II),<sup>12</sup> the American Congress of Rehabilitation Medicine (ACRM),<sup>13</sup> and the *International Classification of Diseases*, 10th edition (ICD-10).<sup>14</sup> The variations in these definitions, which are outlined in *Figure 2*, reveal the lack of standardization in even defining posttraumatic headache. However, the commonalities between the institutional definitions are that mild posttraumatic headache, when acute, lasts less than 3 months and is a trauma-induced physiologic disruption of brain function, as manifested by at least one of several symptoms.<sup>12-14</sup>

The multiple biochemical effects resulting from physical trauma in patients with posttraumatic headache are thought to lead to the pain and cognitive effects of head trauma.<sup>1,15,16</sup> During the mechanical stretch of trauma, a deformation of the plasma membrane may induce depolarization and neuronal firing, which opens potassium channels and allows a substantial efflux of potassium from cells. Nonspecific depolarization of neurons leads to release of the excitatory neurotransmitter glutamate, which activates N-methyl D-aspartate (NMDA) and d-amino-3-hydroxy-5-methyl-4-isoxasolepropionic acid receptors. These receptors also lead to potassium efflux as well as calcium influx. This increase of intracellular calcium can be sequestered in the mitochondria, leading to multiple anomalous cascades:

- protease activation causing cell damage and death
- breakdown and dysfunction of neurofilaments and microtubules
- increased dependence on glycolysis-generated adenosine triphosphate (ATP) caused by dysfunction in production of ATP via oxidative metabolism<sup>16</sup>

Overall, decreased cerebral glucose metabolism may account for post-TBI impairments in consciousness, memory, and cognition. Increased intracellular sodium-potassium-adenosine triphosphatase works overtime in an attempt to restore the appropriate membrane potential. This activation of energy requires ionic pumps and leads to increased consumption of ATP and glucose.

Following injury, intracellular magnesium decreases substantially for up to 4 days. It is hypothesized that decreased magnesium impairs ATP production in both glycolysis and oxidative phosphorylation.<sup>16</sup> Magnesium is also necessary for initiation of protein synthesis and maintenance of cell membrane potentials. In addition, the voltage-gate of NMDA receptor ionosphere is dependent on the blocking presence

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### ■ ICD-10

- History of “head trauma with loss of consciousness” preceding the onset of symptoms by up to 4 weeks
- At least three of six symptoms
  - headaches, dizziness, general malaise, excessive fatigue, or noise intolerance
  - irritability, emotional lability, depression, or anxiety
  - subjective complaints of concentration or memory difficulty
  - insomnia
  - reduced tolerance to alcohol
  - preoccupation with these symptoms and fear of permanent brain damage

### ■ ACRM

- Trauma-induced physiologic disruption of brain function, as manifested by at least one symptom
  - any decrease in level of consciousness ( $\leq 30$  min or less)
  - any loss of memory for events immediately before or after the accident
  - any alteration in mental state at the time of the accident (feeling dazed, disoriented, or confused)
  - focal neurologic deficits that may or may not be transient

### ■ ICHD-II

- Starts within 7 days of trauma
- When acute, lasts  $< 30$  min
- Has at least one of three symptoms
  - loss of consciousness ( $< 30$  min)
  - symptoms and signs of concussion
  - Glasgow Coma Scale score of at least 13

**Figure 2.** Criteria for mild posttraumatic headache and traumatic brain injury by classification system. **Abbreviations:** ACRM, American Congress of Rehabilitation Medicine; ICD-10, International Statistical Classification of Diseases and Related Health Problems, 10th revision; ICHD-II, International Classification of Headache Disorders, 2nd edition. **Sources:** International Classification of Diseases, 10th rev (ICD-10). Geneva, Switzerland: World Health Organization; 1992. The International Classification of Headache Disorders, 2nd ed (ICD-II). Cephalalgia. 2004;24(suppl 1):1-160. Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine (ACRM). Definition of mild traumatic brain injury. J Head Trauma Rehabil. 1993;8:86-87.

of magnesium. When magnesium ions are low, this voltage block may be overcome more easily, leading to higher levels of calcium influx.<sup>16</sup>

Finally, trauma also leads to lactate accumulation—caused by decreased metabolism and increased production—in the brain. Elevated lactate has been implicated in neuronal dysfunction by inducing acidosis, membrane damage, altered blood-brain barrier permeability, and cerebral edema.<sup>16</sup>

For more information, Giza and Hovda<sup>16</sup> provide an excellent description of the pathophysiology of posttraumatic headache.

### **Incorporation of Osteopathic Principles and Practice**

There is moderate evidence that for patients with chronic headache, the efficacy of OMT—specifically cranial, muscle energy, and HVLA techniques—is comparable to prophylactic medications.<sup>6,17</sup> However, the percentage of osteopathic physicians incorporating OMT into their practices is low.<sup>18</sup> This lack of OMT use leaves not only a void in research but also a void in available practitioners for patients who might benefit from this therapy.

However, in our experience, the incorporation of other osteopathic principles into practice does not seem to be diminishing. As suggested by several osteopathic medical professionals,<sup>19</sup> illness is thought to “represent the body’s inadequate, self-regulatory responses to challenges from the internal and external environment.” Therefore, a whole-patient approach is needed to facilitate patient recovery from disease. As osteopathic physicians, we should include the patient’s emotional state and lifestyle—including their daily activities, physical environments, emotional stressors, and food triggers that may exacerbate the patient’s pain—in every patient evaluation.

### **Conclusion**

Treatment of patients with chronic posttraumatic headache is multifaceted but not standardized. Comprehensive plans should include symptomatic pain relief, pharmacoprophylaxis, and complementary therapy.<sup>4,20</sup>

Given the prevalence and incidence of post-traumatic headache, attention should be given to developing more standardized treatment protocols for this patient population. We believe that a standardized, multi-disciplinary approach should include the following:

- pharmacologic intervention
- osteopathic manipulation
- psychological evaluation for stress, anxiety, and depression
- evaluation for headache triggers, including food and environment
- localized injections (eg, botulinum toxin, anesthetic/steroid combination)
- physical therapy, including biofeedback, modalities, and muscle re-education

As osteopathic physicians, it is incumbent on us to use all available resources—including osteopathic principles and practice—in treating our patients.

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