TELE-REHABILITATION INTERVENTIONS GUIDELINE

Management of Headaches in the Patient with Traumatic Brain Injury

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1) Definition, Assessment, Diagnosis
   b) Definition
      (1) Post-Traumatic Headache (PTH): secondary headaches that can be ascribed to traumatic brain injury, whiplash injury, or craniotomy/craniectomy. Headaches occurs within 7 days of head and/or neck trauma following return to consciousness and/or the ability to report pain (ICHD-3, 2018).
      (2) Typical PTH subtypes include tension-type headaches, migraines, cervicogenic headaches, craniomandibular headaches, craniofacial neuralgias, and medication overuse headaches (Zasler, 2021) PTHs comprise up to 4% of all headache disorders (Ashina, 2019). Review of TBI Model Systems data has shown a 71% cumulative incidence of PTH following moderate to severe TBI with a 44% prevalence at 1 year (Hoffman, 2020).
      (3)
      (4) Pathogenesis remains unclear but likely involves overlapping mechanisms of both migraines and TBI including impaired descending pain modulation, neuroinflammation, trigeminal system activation, cortical spreading depression, CGRP-mediated pain signaling, vascular injury, and axonal shearing. (Mavroudis, 2023)
      (5) Risk factors: age ≤ 60, female sex, history of prior TBI, history of prior headaches, comorbid psychiatric conditions (Mavroudis, 2023)
         a) Risk factors for acute PTH:(Sergeyenko, 2025)
            (i) History of migraine
            (ii) Previous psychiatric history
            (iii) Positive CT scans
            (iv) Younger age
            (v) Female sex
         b) Risk factors for persistent PTH: (Sergeyenko, 2025)
            (i) History of migraine

Female sex

   c) Assessment (Zasler, 2021)
      (1) History of present illness:
         a) Details surrounding the head trauma:
            (i) Mechanism of injury
            (ii) Temporal relationship of headache and head trauma

b) Associated symptoms: dizziness, fatigue, reduced ability to concentrate, psychomotor slowing, mild memory problems, insomnia, anxiety, personality changes and irritability.
c) Alleviating and exacerbating factors
d) Functional impairments associated with headaches (i.e., missed work, issues performing ADLs, etc)

(2) Past Medical History
a) Previous history of headaches
b) Change in headache character (duration and frequency of attacks, headache location, type of pain, headache severity)
c) Associated symptoms such as nausea, vomiting, photophobia, phonophobia
d) Prior headache treatments
e) Mood disorders such as depression and/or anxiety

(3) Family history: headaches/migraines

(4) Physical Examination (Zasler, 2021)
a) Neurological examination:
(i) Mental status assessment
(ii) Cranial nerve examination
(iii) focused vestibular-ocular examination
(iv) Motor strength and coordination
(v) Gait assessment
(vi) Deep tendon reflexes
b) Head, neck, and shoulder exam:
(i) Inspection for obvious injuries and asymmetry; posture
(ii) Range of motion, including temporomandibular joint
(iii) Palpation

(5) Functional Assessment:
a) Discussion on level of activity including vocation and recreation; performance of ADLs and IADLs,
b) Migraine Disability Assessment questionnaire (MIDAS)- this is a 5-item survey that identifies headache impact on daily life.
c) Postconcussion Symptom Scale and Rivermeade Post Concussion Symptom Questionnaire can screen for common mTBI symptoms like headache. (Sergeyenko, 2025)

b) Diagnosis (ICHD-3):

(1) Acute PTH:
a) Head trauma acutely associated with:
(i) Transient confusion, disorientation or impaired consciousness
(ii) Loss of memory for events immediately before or after the head injury
(iii) Two or more other symptoms suggestive of traumatic brain injury: nausea, vomiting, visual disturbances, dizziness and/or vertigo, impaired memory and/or concentration.
b) Headache is reported to have developed within 7 days after one of the following:
(i) The injury to the head

(ii) Regaining consciousness following the injury to the head
1. Discontinuation of medication(s) that impair ability to sense or report headache after injury to the head

c) Either of the following:
(i) Headache has resolved within 3 months after the injury to the head
(ii) Headache has not yet resolved but 3 months have not yet passed since the injury to the head
1. Not better accounted for by another ICHD-3 diagnosis

d) Delayed onset PTH: Time of headache onset is uncertain or > 7 days.

e) Persistent PTH: Same as acute PTH, but headache persists ≥ 3 months

(2) Headache classification: PTH are commonly classified based on the primary headache they most resemble. Migraine type and tension type headaches are the most common subtypes of PTH (Zasler, 2021; Scwedt, 2021; ICHD-3, 2018;):

a) Migraine
(i) At least FIVE attacks
(ii) Headache pain lasts 4-72 hours (untreated or unsuccessfully treated)
(iii) At least TWO of the following:
1. Unilateral pain
2. Pulsating quality
3. Moderate or severe pain intensity
4. Worsened by or resulting in avoidance of routine physical activity
(iv) During headache at least one of the following:
1. Nausea and/or vomiting
2. Photophobia and phonophobia

b) Tension type headache
(i) Lasts from 30 minutes to 7 days
(ii) At least TWO of the following:
1. Bilateral pain
2. Pressing and/or tightening, non-pulsating quality
3. Mild or moderate intensity
4. Not aggravated by routine physical activity
(iii) Both of the following:
1. No nausea or vomiting
2. photophobia OR phonophobia

c) Cervicogenic headache
(i) Pain referred originating in neck and perceived in the head/face
(ii) Clinical, laboratory, and/or imaging evidence of pathology within the cervical spine or soft tissues of the neck
(iii) At least ONE of the following:
1. Clinical signs of pain source in the neck
2. Headache relief with diagnostic block of cervical structure or its nerve supply

d) Other causes of secondary headache that must be ruled out
(i) Intracranial disorder
(ii) Use of a substance or substance withdrawal
(iii) Infection

(iv) Disorder of cranium/neck/sinuses/teeth
(v) Psychiatric disorder
(vi) Medication overuse headache

2) Management and Treatment Recommendations (Zasler, 2021)
   a) Treatment Goals: Early treatment and patient education to avoid or decrease functional impairments and disability and prevent future headaches (Kamins, 2021)
   b) Current literature encourages clinicians to pursue treatment plans that are tailored to a patient's specific type and severity of symptoms (Heslot, 2022)
   c) Limited evidence regarding treatment in the TBI population; treatment follows the current evidence-based treatment guidelines for primary headache (ICHD-3, Brown, 2014)
      (1) Non-pharmacologic treatments
         a) Lifestyle medication including sleep regulation, addressing caffeine and medication overuse, changing diet consistencies for TMJ disorders.
         b)
         c) Early counseling, psycho-education, and/or CBT can be recommended to at-risk patients. (Heslot, 2022)
            (i) For best management, CBT alongside traditional therapies (drugs) should be considered to treat the psychological and physiological dimensions (Sanford, 2025).
         d) Learning to Cope with Triggers (LCT) is a CBT intervention that has shown potential benefits for managing mTBI, specific triggers including light, computer screens, and noise (Sanford, 2025). Acupuncture: Consists of one to two 30-minute sessions weekly for 2 or more months.
         e) Transcutaneous Electrical stimulation (TENs unit) (Schoenen, 2013) Supraorbital TENs is beneficial for patients with episodic headache. Treatment for 20 minutes daily for three months.
         f) Heat and cold modalities:
            (i) Heat modalities, either superficial or deep, can have an analgesic effect and help with muscle relaxation.
            (ii) Cold modalities help by slowing of blocking nerve conduction especially in the small pain nerve fibers.
         g)
         h) Sub-symptom threshold aerobic exercise can improve quality of life in patients with post-TBI headaches. (Mercier, 2025)
         i) Light therapy has proven to be beneficial for help with headaches and improving sleep which can contribute to symptom exacerbation (Heslot, 2025)
         j) rTMS is emerging as a treatment option for post-traumatic headaches after mTBI. (Heslot, 2022)
            (i) RTMS potentially reduces frequency and intensity of headaches in adults, but evidence supporting is currently deemed low to moderate quality (Moser, 2024).
            (ii) The dorsallateral prefrontal cortex (dlPFC) is the most targeted region, but M1 is also a possible target in reducing intensity and frequency of headaches (Galimberti, 2023)

k) Progressive Medicine Relaxation (PMR) is a behavioral relaxation technique which has shown possible benefits in reducing the number or headaches within a time period, but confidence in effectiveness is unknown (Moser, 2024).
l) Evidence suggests that multidisciplinary care (MDC) incorporating psychological interventions over 22 weeks reduces postconcussion headache intensity when compared with standard care (Price, 2023).
m) Studies do not support use of Hyperbaric Oxygen Therapy (HBOT) for persistent PCS after mTBI (Heslot, 2022). HBOT did not lead to a reduction in headache severity at the two- and three-year follow-up assessments (Price, 2023).

(2) Pharmacologic treatment (Zasler, 2021; Silberstein, 2012; Tfelt, 2013)

a) Abortive treatment: Includes Nonsteroidal Anti-inflammatory Drugs (NSAIDs), ergot derivatives, triptans, calcitonin gene-related peptide (CGRP) antagonists, dopamine (D2) blockers and opioid medications.

(i) Acetaminophen and NSAIDs:
1. Acetaminophen dosing should not exceed 3 grams per day
2. NSAIDs such as ibuprofen, aspirin, and naproxen be used and have been shown to be more effective than placebo for acute migraines, but caution should be observed in those with or at risk for bleeding issues, gastritis/ulcers, and renal dysfunction.
3.

(ii) Ergot derivatives (dihydroergotamine (DHE)) (Mackenzie, 2023)
1. Repetitive IV DHE 0.5 mg given demonstrated good to excellent relief of headaches. Some side effects reported were mild nausea and a brief worsening of headache immediately post-treatment.
   a. Patients were given metoclopromide for nausea and discharged on TCA with either a beta-blocker or CCB for maintenance therapy

(iii) Triptans
1. Examples include: Sumatriptan, rizatriptan, zolmitriptan
2. Triptans bind and activate serotonin 1b/1d receptors thereby inhibiting the release of vasoactive peptides and promoting constriction of blood vessels as well as inhibiting dural nociception and pain (Hansen, 2000).
3. Found to be superior to other nonspecific treatments for patients with migraine headaches and can also relieve nausea, phonophobia, and photosensitivity. (Mackenzie, 2023)
4. Because of their vasoconstrictive properties, triptans should NOT be used in patients with cardiovascular disease and/or cerebrovascular disease or hemiplegic migraines.

(iv) Calcitonin gene-related peptide (CGRP) antagonists
1. Examples: Gepants, ubrogepant, rimegepant
2. This class of medications is reserved for those who cannot tolerate triptans due to side effects or cardiac/cerebrovascular disease.

(v) Dopamine (D2) blockers (Metoclopramide) (MacKenzie, 2023)
1. Administration of IV metoclopramide 20 mg and diphenhydramine 25 mg (for akathisia) demonstrated reduction in headache pain in patients

within one to two hours. Patients reported side effects of sleepiness 48 hours of post-treatment.

(vi) Opioids and combination analgesics containing opioids.

1. Routine use not recommended, but short-term use of opioids may be necessary when other medications are contraindicated or ineffective (ICHD-3, 2018).

b) Prophylactic treatment: is used to reduce headache frequency in those with significant disability despite optimal acute drug therapy, prevent medication overuse headaches, and utilized in those with contraindications to acute migraine medications. Patients may benefit from selection of a medication that also treats co-existing conditions.

(i) Beta-Blockers

1. Propranolol: Non-selectively binds beta1- and beta 2 adrenergic receptors preventing adrenergic stimulation.
   a. Max daily dose is 160-240 mg.
   b. Consider if co-morbid anxiety or agitation/restlessness.
2. Metoprolol: Selectively binds beta 2 adrenergic receptors preventing adrenergic stimulation.
   a. Max daily dose is 200 mg.
   b. Side effects include fatigue and hypotension.
3. Avoid or use with caution in patients with asthma, diabetes, bradycardia, and peripheral vascular disease.

(ii) Antidepressants

1. Amitriptyline: Tricyclic antidepressant that increases the concentration of serotonin and/or norepinephrine by inhibiting their reuptake.
2. Start 10 mg daily (at bedtime). Max daily dose is 150 mg.
3. Consider if co-morbid depression, insomnia, or anxiety.
4.
5. Possible side effects: oversedation/fatigue, irritability, nausea, abdominal discomfort, vivid dreams, heart palpitations, prolonged QT interval, dry mouth, decreased libido. (Mackenzie, 2023)Contraindicated in patients with angle-closure glaucoma.

(iii) Antiepileptics

1. Topiramate: Increases the availability of gamma-aminobutyric acid (GABA), an inhibitory neurotransmitter.
   a. 50 mg to 200 mg daily.
   b. *Avoid in patients with angle-closure glaucoma.*
2. Valproate: Increases the availability of GABA, an inhibitory neurotransmitter.
   a. Start at 250 mg BID. Maximum daily dose is 1000-1500 mg.
   b. Consider if comorbid depression or mood lability.
   c. Contraindicated in pregnancy and women of child-bearing age due to risk of teratogenicity. (AAN, 2013).
3. Gabapentin has not been found effective. (Silberstein, 2013).

(iv) Muscle relaxants

1. Flexeril: Centrally acting skeletal muscle relaxant pharmacologically related to tricyclic antidepressants; reduces tonic somatic motor activity influencing both alpha and gamma motor neuron. Use with caution in patients with mild hepatic impairment.
2. Methocarbamol: Causes skeletal muscle relaxation by general CNS depression.
3. Baclofen: Inhibits transmission of both monosynaptic and polysynaptic reflexes at the spinal cord level with resultant relief of muscle spasticity. Avoid abrupt withdrawal
4. Tizanidine: An alpha2-adrenergic agonist agent which decreases spasticity by increasing presynaptic inhibition; overall effect is to reduce facilitation of spinal motor neurons. Avoid use in hepatic impairment.

(i) Calcitonin gene-related peptide (CGRP) inhibitors

1. Erenumab: Peripherally acting large CGRP monoclonal antibody.
2. Moderate quality evidence suggests that erenumab reduces headache intensity at 12 weeks following initiation of treatment (Price, 2023).
3. Treatment with erenumab 70 mg to 140 mg administered subcutaneously, once monthly, showed a greatly reduced number of monthly headache days after 8 weeks of treatment. Side effects included constipation, occurring in approximately 20% of patients (MacKenzie, 2023).

d) Restrictions

(1) Avoid all medications exceptTylenol if pregnant.
(2) Triptan use should be limited to fewer than 10 days a month to avoid medication overuse headache.
(3) Medication Overuse Headache (Rebound Headache) - Taking headache relief drugs ≥ 3 times a week may lead to overuse headache, in which the initial headache is relieved temporarily but reappears as the drug wears off. Taking more medication to treat the recurrent headache leads to progressively shorter periods of pain relief and results in a pattern of recurrent chronic headache. It may take weeks for these headaches to end once the drug is stopped (ICHD-3, 2018).

(3) Injections:

a) Onabotulinum toxin A: Chemical denervation via intramuscular injection of botulinum toxin which prevents fusion of the acetylcholine-filled vesicles with the synaptic cleft and prevents the release of acetylcholine (Nance, 2011; Govindarajan 2016).

(1) Recommended dosing follows chronic migraine protocol (PREEMPT) : A total of 155 units are injected intramuscularly over 31 head and neck sites per protocol every 3 months
(2) Recommended for migraine prevention for those with ≥15 headache days per month for at least three months with at least eight of the 15 headaches fitting migraine criteria)

b) Occipital nerve (ON) injections with corticosteroids and/or local anesthetics have been employed for the acute and preventive treatment of migraine but there is little evidence to support this, so it is not recommended.

c) Other injections (Barbanti, 2014; Dach, 2015)
   a) Lidocaine: Local anesthesia occurs due to sodium channel blockade in a frequency-dependent and voltage-dependent manner which suppresses cellular excitability.
      (1) Recommended for both acute treatment and prevention of craniotomy induced TTH with trigger points, as well as frequent episodic TTH
      (2) 0.5% lidocaine injected into each painful point along the scar or each peri-cranial myofascial trigger point. For those not responding to one treatment, repeat injection once a week for 3 consecutive weeks or one treatment of 2% lidocaine plus dexamethasone 1mg/mL in 7:3 proportion.

(4) Prevention and Education
   a) Inadequate or inaccurate treatment may result in transformation to chronic daily headache or medication overuse headache. (Lew, 2006; Watanabe, 2012).
   b) Persistent headache can incite or worsen mood disorders, insomnia and cognitive impairment, all of which can affect functional outcome. (Watanabe, 2012).
   c) For severe attacks, consider an additional rescue medication if acute medication does not consistently work.
   d) Initiate therapy with a low dose and increase gradually to minimize side effects.
   e) Ensure the patient has realistic explanations with migraine prophylaxis:
      a) Headache attacks will likely not be abolished completely.
      b) Migraine prophylaxis treatment is considered successful if migraine attack frequency is reduced by 50%.
      c) It may take 4 to 8 weeks for significant benefit to occur.
   f) Treatment of post-traumatic headache should be individualized, with consideration given to prior treatment response, medication tolerability, the patient's specific constellation of post-traumatic symptoms and the functional impact of headache on daily activities. Clinical decision making should also account for the possibility and rates of adverse events with limited available evidence regarding comparative effectiveness of treatment options (MacKenzie, 2023).

This guideline was developed to improve health care access in Arkansas and to aid health care providers in making decisions about appropriate patient care. The needs of the individual patient, resources available, and limitations unique to the institution or type of practice may warrant variations.

Guideline Developers
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