TELE-REHABILITATION INTERVENTIONS GUIDELINE

Management of Headaches in the Patient with Post Traumatic Brain Injury

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I. Definition, Assessment, Diagnosis
  A. Definition
    1. Post-Traumatic Headache (PTH): headache that develops within 7 days of head trauma, after regaining consciousness and/or the ability to sense and report pain (International Classification of Headache Disorders, 3rd edition, ICHD-3).
      a. May occur after mild, moderate or severe traumatic brain injury (TBI). Headache severity tends to parallel injury severity (Brown, 2014).
      b. May have one or more subtypes of PTH, including tension or migraine (Lew, 2006).
      c. Prevalence of headache ranges anywhere from 30-90% of those with TBI and approximately 22% continuing to have headache beyond 1 year post trauma (Hoffman, 2011)
      d. Pathogenesis is often unclear, but numerous factors may contribute, including: axonal injury, alterations in cerebral metabolism, alterations in cerebral hemodynamics, genetic predisposition, psychopathology and the expectation of developing headache after head injury (ICHD-3).
      e. Head pain may be related to direct damage to the skull or brain tissue; muscular, tendinous and/or ligamentous injuries to peripheral nerves (Watanabe, 2012)
      f. Other nervous system injuries, such as visual and vestibular system damage, may also contribute to headache syndromes (Watanabe, 2012).
  B. Assessment (Kamins, 2018)
    1. History of present illness:
      a. Details surrounding the head trauma:
        1) Duration of LOC
        2) Duration of PTA
        3) Use of pain medications
        4) Temporal relationship of headache and head trauma
          a) Continuous vs episodic
        5) Associated symptoms: dizziness, fatigue, reduced ability to concentrate, psychomotor slowing, mild memory problems, insomnia, anxiety, personality changes and irritability.
      b. Past Medical History
        1) Previous history of headaches
          a) Change in character (duration and frequency of attacks, headache location, type of pain, headache severity)
          b) Associated symptoms such as nausea, vomiting, photophobia, phonophobia
          c) Prior treatments
        2) Family history of headaches
          a) Family history of migraines
          b) Family history of posttraumatic headache
        3) Mood disorders: depression and/or anxiety
    2. Physical Examination (Becker, 2015)
      a. Neurological examination:

1) Mental status assessment
2) Cranial nerve examination
3) Motor strength and coordination
4) Gait assessment
5) Deep tendon reflexes

b. Jaw and Neck exam: Range of motion, palpation

C. Diagnosis (ICHD-3):

D. Acute PTH

A. Any headache fulfilling criteria C and D

B. Injury to the head fulfilling both of the following:

a. Associated with none of the following:

1. Loss of consciousness for > 30 minutes
2. Glasgow Coma Scale (GCS) score < 13
3. Post-traumatic amnesia lasting > 24 hours
4. Altered level of awareness for > 24 hours
5. Imaging evidence of traumatic head injury such as intracranial hemorrhage and/or brain contusion

b. Associated immediately following the head injury with one or more of the following symptoms and/or signs:

1. Transient confusion, disorientation or impaired consciousness
2. Loss of memory for events immediately before or after the head injury
3. Two or more other symptoms suggestive of mild traumatic brain injury: nausea, vomiting, visual disturbances, dizziness and/or vertigo, impaired memory and/or concentration.

C. Headache is reported to have developed within 7 days after one of the following:

a. The injury to the head
b. Regaining consciousness following the injury to the head
c. Discontinuation of medication(s) that impair ability to sense or report headache following the injury to the head

D. Either of the following:

a. Headache has resolved within 3 months after the injury to the head
b. Headache has not yet resolved but 3 months have not yet passed since the injury to the head

c. Not better accounted for by another ICHD-3 diagnosis

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

c. Persistent PTH: Same as acute PTH, but headache persists ≥ 3 months

1) Chronic PTH attributed to moderate to severe head injury

a) Loss of consciousness for > 30 minutes
b) Glasgow Coma Scale < 13
c) Posttraumatic amnesia for > 48 hours
d) Imaging pathology such as intracranial hemorrhage and/or brain contusion

2) Chronic PTH attributed to mild head injury

a) Either no loss of consciousness or loss of consciousness < 30 minutes
b) Glasgow Coma Scale of 13 or greater
c) Signs or symptoms of concussion

i. Headache classification (Watanabe, 2012)

1. Migraine without aura

a. At least FIVE attacks that fulfill criteria 2-4
b. Headache pain lasts 4-72 hours

c. At least TWO of the following:
    a) Unilateral pain
    b) Pulsating quality
    c) Moderate or severe pain intensity
    d) Aggravation by or causing avoidance of routine physical activity (eg walking or climbing stairs)
d. During headache at least one of the following:
    a) Nausea and/or vomiting
    b) Photophobia and phonophobia

2. Migraine with aura
    a. At least TWO attacks that fulfill criteria 2-4
    b. Aura consisting of at least ONE of the following:
        a) Fully reversible visual symptoms (flickering lights, spots or lines and/or loss of vision)
        b) Fully reversible sensory symptoms (pins and needles and/or numbness)
        c) Fully reversible dysphasic speech disturbance
    c. At least TWO of the following:
        a) Homonymous visual symptoms and/or unilateral sensory symptoms
        b) At least ONE aura symptom
        c) Each symptom lasts 5-60 minutes
        d) Migraine headache without aura beings during the aura or follows the aura within 60 minutes
3. Probable migraine (with/without aura): fulfills all but one of the criteria for either type of migraine headache.
4. Tension type headache
    a. Lasts 30 minutes to 7 days
    b. At least TWO of the following:
        a) Bilateral pain
        b) Pressing and/or tightening (non-pulsating) quality
        c) Mild or moderate intensity
        d) Not aggravated by routine physical activity
    c. Both of the following:
        a) No nausea or vomiting
        b) photophobia OR phonophobia
5. Cervicogenic headache
    a. Pain referred originating in neck and perceived in the head/face
    b. Clinical, laboratory, and/or imaging evidence of pathology within the cervical spine or soft tissues of the neck
    c. At least ONE of the following:
        a) Clinical signs of pain source in the neck
        b) Headache stops after diagnostic block of a cervical structure or its nerve supply
        c) Resolves within 3 months after successful treatment of the causative disorder

ii. Other causes of secondary headache (ICHD-3) that must be ruled out
1. Head/neck trauma
2. Intracranial disorder
3. Use of a substance or substance withdrawal
4. Infection
5. Disorder of cranium/neck/sinuses/teeth
6. Psychiatric disorder
7. Medication overuse headache

F. Management and Treatment Recommendations

A. Treatment Goals: Early treatment and patient education to avoid or decrease functional impairments and disability and prevent future headaches. (Lucas, 2011 & 2012; Manzoni, 2014).

B. General Approach

1. Limited evidence regarding treatment in the TBI population; treatment follows the current evidence-based treatment guidelines for primary headache (ICHD-3, Brown, 2014).

C. Non-pharmacologic treatments (Barbanti, 2014)

1. Migraine prophylaxis
   a. Relaxation Training, Biofeedback, and Cognitive Behavioral Therapy (CBT) are considered to be effective
      1) These modalities are more effective when used in conjunction
   b. Acupuncture: Consists of one to two 30 minute sessions weekly for 2 or more months.
   a. Transcutaneous Electrical stimulation (TENs unit) (Schoenen, 2013). Supraorbital TENs is beneficial for patients with episodic headache. Treatment for 20 minutes daily for three months.
2. Acute tension type headaches
   a. Relaxation Training, Biofeedback, and Cognitive Behavioral Therapy (CBT) are considered to be effective components of stress management training.
   b. Physical therapy (most commonly prescribed) and/or therapeutic exercise program.

D. Pharmacologic treatment (Silberstein, 2012; Tfelt, 2013)

1. Migraine/ Probably Migraine Headache
   a. Pharmacologic treatment of Acute Attacks Treat attack rapidly and consistently, minimize adverse events, restore the patient's ability to function.
      a. Simple Analgesics:
         i. Nonsteroidal anti-inflammatory drugs (NSAIDs):
            1. First line agents.
            2. Acetylsalicylic acid 1000 mg, Ibuprofen 400 mg, and Naproxen Sodium 500 to 550 mg.
            3. *NSAIDs can cause gastric irritation, bleeding, and renal dysfunction.*
         ii. Acetaminophen (Tylenol ®):
            a) First line agent.
            b) Acetaminophen 1000 mg for mild to moderate severity
            c) Daily dosage should not exceed 3 grams per day
            d) If NSAIDs and/or acetaminophen are ineffective, a triptan should be tried.
      b. Triptans (Sumatriptan (Imitrex®) , Rizatriptan (Maxalt ®), Zolmitriptan (Zomig ®): Bind to and activate Serotonin 1b/1d receptors in the brainstem, which inhibits the release of vasoactive peptides, promotes constriction of blood vessels, and inhibits dural nociception and pain (Hansen, 2000; Bartsch, 2004).
         a) Oral triptans are 1st line agents for acute attacks of all severities when NSAIDs and Acetaminophen are ineffective
         b) If not relieved with one triptan, a different triptan should be offered
         c) If the migraine recurs after initial relief, patient should take a second dose (within recommended dosage limits)
         d) Subcutaneous Sumatriptan 6 mg should be considered for severe migraine or where vomiting precludes effective use of the oral route.

e) Triptans are vasoconstrictors and should be avoided in patients with cardiovascular disease and cerebrovascular disease, as well as those with moderate to severe liver impairment.

f) Concomitant use of Rizatriptan and Propranolol is cautioned, decrease the dose of Rizatriptan when using both since propranolol increases rizatriptan levels by 70 %.

c. Anti-emetics : Bind to Dopamine D2 receptors and block the action, decreasing nausea and vomiting. In addition, they are effective for reducing migraine headache pain.
   a) Oral agents, Metoclopramide (Reglan ®) 10 mg up to 4 times per day orally and Domperidone 10 mg up to 3 times per day) are recommended to treat nausea and potential emesis in migraine.
   b) Intravenous Metoclopramide 10 mg can be used as monotherapy in the acute treatment of patients with migraine.
   c) These drugs may improve the absorption of analgesics.
   d) Domperidone has fewer side effects than Metoclopramide.
   e) In contrast to intravenous or intramuscular preparations, oral antiemetics should not be considered as monotherapy in acute migraine.
   f) *Possible adverse side effects include akathisia, dystonia, QT prolongation and Torsades des pointes*.

d. Opioids and combination analgesics containing opioids.
   a) Routine use not recommended, but short term use of opioids may be necessary when other medications are contraindicated or ineffective (ICHD-2).

b. Pharmacologic Migraine Prevention: Prophylactic treatment is used to reduce migraine frequency in those with significant disability despite optimal acute drug therapy, those at high risk for medication overuse headache, and those with contraindications to acute migraine medications. Patients may benefit from selection of a medication that also treats co-existing conditions.
   1) Medications
      a) Beta-Blockers
         1. Propranolol (Inderal ®): Non-selectively binds beta1- and beta 2 adrenergic receptors, preventing adrenergic stimulation.
            a. First-line agent.
            b. Max daily dose is 160-240 mg.
            c. Consider if co-morbid anxiety.
         2. Metoprolol (Lopressor ®): Selectively binds beta 2 adrenergic receptors, preventing adrenergic stimulation.
            a. Max daily dose is 200 mg.
         3. *Side effects include fatigue and hypotension.*
         4. *Avoid or use with caution in patients with asthma, diabetes, bradycardia, and peripheral vascular disease.*
      b) Antidepressants
         1. Amitriptyline (Elavil ®): Tricyclic antidepressant that increases the concentration of serotonin and/or norepinephrine by inhibiting their reuptake.
            a. First-line agent.
            b. Start 10 mg daily (at bedtime). Max daily dose is 100 mg.
            c. Consider if co-morbid depression, insomnia, or anxiety.

d. Side effects include dry mouth, constipation and sedation, arrhythmia.
e. Contraindicated in patients with angle-closure glaucoma.

2. Venlafaxine (Effexor ®): Increases the concentration of serotonin and/or norepinephrine by inhibiting their reuptake.
   a. Start at 37.5 mg per day. Maximum daily dose is 150 mg.
   b. Consider if co-morbid depression, insomnia, or anxiety.

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

2) Injections
a) Onabotulinum toxin A (Botox ®): Chemical denervation occurs when the intramuscular injection of the toxin prevents fusion of the acetylcholine-filled vesicles with the synaptic cleft, preventing the release of acetylcholine and therefore muscle activity (Nance, 2011; Govindarajan 2016). Evidence suggests
1. A total of 155 units are injected intramuscularly over 31 total sites per protocol every 3 months by clinicians experienced in its use for headache.
2. Recommended for migraine prophylaxis (headache ≥15 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.

2. Tension Type Headache (TTH): Usually mild to moderate in severity. Patients with chronic TTH are more likely to require medications including prophylactic therapy. (Guidelines, 2012).

a. Pharmacologic Treatment of acute headache
1) Simple Analgesics are the drugs of first choice .
   a) Ibuprofen 200 mg to 400 mg.
   b) Acetylsalicylic acid 500 mg to 1000 mg.
   c) Naproxen sodium 275 mg to 500 or 550 mg.
   d) Acetaminophen 500 mg to 1000 mg oral.
2) Combination analgesics containing caffeine are drugs of second choice.
3) The following are not recommended for routine use: Muscle relaxants, opioids, triptans.

b. Injections (Barbanti, 2014; Dach, 2015)

1) Lidocaine : Local anesthesia occurs due to sodium channel blockade in a frequency-dependent and voltage-dependent manner which suppresses cellular excitability.
   a) Recommended for both acute treatment and prevention of craniotomy induced TTH with trigger points, as well as frequent episodic TTH
   b) 0.5% lidocaine injected into each painful point along the scar or each pericranial 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.
2) Botox: botulinum neurotoxin is probably ineffective for treating chronic tension-type headaches as per American Academy of Neurology Practice Guidelines 2016

c. Pharmacologic Prevention of TTH: Used when TTH attacks are frequent, but the efficacy of the preventive drugs is limited.
   1) Amitriptyline 10 mg to 100 mg daily is a first line agent.
   2) 2nd line agents
      a) Mirtazipine (Remeron ®): Is a tetracyclic antidepressant that works by its central presynaptic alpha2-adrenergic antagonist effects, which increases the release of norepinephrine and serotonin. It is also a potent antagonist of 5-HT2 and 5-HT3 serotonin receptors and H1 histamine. Start at 7.5mg daily. Max dose is 30 mg daily. Side effects include drowsiness and weight gain.
      b) Venlafaxine 150 mg daily. Side effects include vomiting, nausea, dizziness, and loss of libido.
   3) Muscle relaxants
      a) Flexeril (Cyclobenzaprine): 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.
      b) Methocarbamol (Robaxin ®): Causes skeletal muscle relaxation by general CNS depression.
      c) Baclofen: Inhibits transmission of both monosynaptic and polysynaptic reflexes at the spinal cord level with resultant relief of muscle spasticity. Avoid abrupt withdrawal
      d) Tizanidine (Zanaflex ®) : 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.

E. Restrictions
   1. Avoid all medications except Tylenol 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-2).

G. Prevention and Education

a. Inadequate or inaccurate treatment may result in transformation to chronic daily headache or medication overuse headache. (Lew 2006, Watanabee 2012).
b. Persistent headache can incite or worsen mood disorders, insomnia and cognitive impairment, all of which can affect functional outcome. (Watanabee 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 the dosage gradually to minimize side effects.
e. Ensure the patient has realistic explanations with migraine prophylaxis:
    1. Headache attacks will likely not be abolished completely.
    2. Migraine prophylaxis treatment is considered successful if migraine attack frequency is reduced by 50%.
    3. It may take 4 to 8 weeks for significant benefit to occur.

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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