Evaluation of Headaches in Urgent Care Part 2: Non-Emergent Headaches

Urgent message: Headaches are challenging chief complaints and being able to identify and differentiate among the non-emergent types of headaches will help with medical decision making and patient care.

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In the conclusion of a two-part series, this article focuses on non-emergent headaches. For the purpose of this article, non-emergent headaches are classified as those that are not life-threatening and pose little to no risk of permanent functional disability.

Most non-emergent headaches discussed in this article have one root commonality: trigeminal nerve involvement. The trigeminal nerve (CN V) is the largest of the cranial nerves. The trigeminal nerve has 3 branches: the ophthalmic (V1), maxillary (V2) and the mandibular (V3). The trigeminal nerve is a direct link to the brain and as such, can cause the cascade of pain sensation that results in various headaches. Given that trigeminal nerve pain is present in many headache syndromes, the symptoms and signs of, and treatments for these headaches have overlying components.

Cluster Headache

Cluster Headaches affect less than 1% of the population. There is a significant male predominance, with a male:female ratio of 4:1.[1] The etiology is thought to be a stimulation of the trigeminal-autonomic reflex and new research suggests a familial component.[2] Cluster headache is arguably one of the most severe pain syndromes and suicide attempts have been reported among patients in whom the condition has gone undiagnosed or who have not been successfully treated for it .[3]

Patients with cluster headaches most often present with severe unilateral orbital pain. These headaches are often grouped attacks leading to the name origin, *cluster* headache. The headache may radiate around the orbit, including the frontal and temporal areas, and patients describe it as constant and stabbing and accompanied by autonomic phenomena. The autonomic signs are

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Table 1. NSAIDs Recommended as Treatment to Abort Migraine

- Ibuprofen 400 mg.
- Naproxen 250 mg to 500 mg.
- Diclofenac 50 mg to 100 mg.
- Diclofenac epolamine 65 mg.
- Tolfenamic acid 200 mg.
- Indomethacin 50 mg suppository (Most beneficial in patients with significant nausea or vomiting.)
- Ketorolac 7 mg to 15 mg IM injection.

IM = intramuscular; IV = intravenous; NSAID = nonsteroidal anti-inflammatory drug

ipsilateral to the pain and may include ptosis, meiosis, lacrimation, conjunctival injection, rhinorrhea, and nasal congestion.[4,5] Cluster headaches usually last 15 to 180 minutes and can reoccur up to 8 times per day and daily for several weeks, then remit.[5] Chronic cluster headaches, which are less common, lack sustained remission periods.

Cluster headaches are exclusively a clinical diagnosis, but if a patient has atypical symptoms or abnormal neurological findings, neuroimaging is recommended.

First-line treatment options for cluster headache include oxygen and triptans, with consideration for use of octreotide, intranasal lidocaine, and oral ergotamine in those who do not respond.

Pure oxygen administered at a flow rate of 10 to 15 L/min for 15 minutes via nonrebreathing facial mask with the patient in the upright position is considered safe and effective and was abortive in 78% of cases of cluster headache in one study.[6] Begin with 10 L/min and increase to a maximum of 15 L/min if the lower dose is ineffective. Caution with overuse of oxygen is recommended because it can increase the attack frequency when used repeatedly in some patients.[6] Caution should be taken when giving pure oxygen to patients with severe chronic obstructive pulmonary disease because it may cause severe hypercapnia and CO_2 narcosis.[6]

If oxygen therapy is ineffective, sumatriptan and zolmitriptan are effective for acute treatment of cluster headaches. Intramuscular (IM) injection is preferable to intranasal and oral administration because of the quicker onset. Unpleasant effects include non-ischemic chest pain and distal paresthesia. The triptans should be avoided in patients with known ischemic cardiovascular disease (CVD) and initial doses should be given under medical provider observation to patients with CVD risk factors without known disease.[7]

For patients whose cluster headaches do not respond to or who cannot tolerate the above measures, other therapies to consider are octreotide, intranasal lidocaine, and oral ergotamine.

Octreotide is a somatostatin analog that has been shown to be superior to placebo in achieving symptom improvement to pain-free status when given in a single 100-mcg dose. The most common side effect is minor gastrointestinal upset.[8]

The effective dose of intranasal lidocaine is 20 to 60 mg given with the patient's head in 45-degree extension and rotated toward the symptomatic side by 30 to 40 degrees. Headache improvement can be achieved within 10 minutes, but complete relief is rare.[2] Intranasal use of lidocaine generally lacks systemic side effects.[2]

Ergotamine has been used to treat cluster headaches since the 1940s, but it lacks efficacy in modern studies and must be initiated very early in the attack.[9] The dose is 2 mg sublingual, which can be repeated every 30 minutes to a maximum of 6 mg per day and 10 mg per week.[2] The most common side effects include gastrointestinal upset, weakness in the legs, and numbness in finger and toes.

Measures for prevention of cluster headache include the use of verapamil, which should be initiated at the onset of a cluster episode, given that recurrence over weeks to months is common. The starting dose is 240 mg daily divided in 3 doses. It may be necessary to increase the dose to a maximum of 960 mg daily.[10] When cluster headache periods last less than 2 months, administration of glucocorticoid medication is recommended as a preventative therapy alone. The dosage is 60 mg to 100 mg once daily for 5 days, tapered to a dose of 10 mg daily during the cluster period.[10]

Surgical interventions for cluster headache, including occipital nerve stimulation, hypothalamic deep brain stimulation and nerve sectioning, are still investigational and should be considered with caution.[11,12]

Migraine Headaches

Migraine headaches are estimated to affect about 12% of the general population with a threefold female predominance.[13] The pathophysiology was once believed to relate to vasodilatation of vessels, but that theory has not stood the test of time and science has brought an alternative explanation. Migraine headaches are now understood to be caused by neuronal and glial depolar-

ization spreading across the cerebral cortex, which is believed to trigger aura, stimulate the trigeminal nerve, and alter the blood-brain permeability. The result is inflammatory changes to the pain-sensitive meninges.[14,15]

Migraine headaches are recurrent and typically follow a series of events over hours to days. The typical series of events starts with a prodrome, followed by aura, headache, and the postdrome.

Up to 60% of patients with migraine headaches report experiencing prodromal symptoms 24 to 48 hours before the migraine. These symptoms may include euphoria, depression, irritability, food cravings, constipation, neck stiffness, and increased yawning.[16]

Approximately 25% of migraineurs experience a focal neurological symptom, often before the headache, but sometimes at the same time as the headache. Auras can include visual, sensory, verbal, or motor disturbances.[17] Visual auras are most common and often include vision loss (scotoma) lateral to the fixation point, which expands within 60 minutes to involve a quadrant or hemifield of vision. At the edges of the expanding visual disturbance will be zigzagging lines with a shimmering quality.[18] The second most common aura is sensory disturbance. Patients often describe tingling that progresses to numbness and is unilateral, affecting the face (sometimes including the tongue) and/or limbs. The gradual progression from tingling to numbness is characteristic of migraine aura and not typical of ischemic events.[18] Visual disturbances differ between migraines and simple or complex partial seizures. In seizures the visual disturbance has a rapid time course of 1 – 3 minutes, are small colored circular patterns mostly at the temporal hemifield – flashing lights, zigzag and non-circular patterns are rare in seizures.[19]

The migraine headache itself is typically unilateral and has a throbbing quality, often worsened with movement or Valsalva maneuvers such as sneezing, coughing and straining. Nausea with or without emesis is common and many migraineurs complain about photophobia and/or phonophobia. Some sufferers experience vertigo, cutaneous allodynia (increased skin sensitivity) or osmophobia (increased sensitivity to smells).[20]

Once the throbbing pain ceases, many migraineurs report a postdrome that includes brief mild pain with sudden head movements and many will feel exhausted and drained.

Diagnostics studies are rarely indicated of migraine headaches and the diagnosis is clinical. New onset of migraines is rare after age 60 years and an alternate etiology should be considered. Neuroimaging is recommended for patients who have focal neurological findings or who do not meet the diagnostic criteria for migraine headache.

Table 2. Common Triptans and Routes of Administration

- Sumatriptan: 25 mg, 50 mg, 100 mg oral; 5 mg, 20 mg intranasal; 4 mg, 6 mg subcutaneous
- Rizatriptan: 5 mg or 10 mg oral tablet or an oral dissolving tablet
- Zolmitriptan: 2.5-mg or 5-mg oral tablet or oral dissolving tablets and 5-mg intranasal spray
- Eletriptan: 20-mg or 40-mg oral tablet
- Naratriptan: 1-mg and 2.5-mg oral tablet
- Almotriptan 6.25-mg and 12.5-mg oral tablet

Avoiding migraine triggers is the first step in migraine treatment and patients should be encouraged to record a headache diary to pinpoint triggers and to recognize the early symptoms of their headache. Despite diligent efforts to avoid triggers, however, many patients require medication intervention when acute migraine occurs.

It is important to recognize that oral agents may be ineffective during a migraine because of migraine-associated gastric stasis and should be avoided if a patient has significant nausea or vomiting.[21]

All the drugs recommended for migraine are most effective when taken early in the headache cycle and clinicians need to educate patients to take their medication at the first sign of a headache.

Non-steroidal anti-inflammatory drugs (NSAIDs) are the treatment of choice for aborting a migraine headache already underway. Some patients will respond to acetaminophen alone. The recommended dose of 1000 mg is highly effective for treatment of pain and will reduce pain levels in patients with mild-to-moderate symptoms 20% of the time.[9] Acetaminophen can be combined with NSAIDs. NSAIDs studied for migraine headache and their recommended dosage are listed in **Table 1**.[17,22-25]

Numerous articles exist in the literature documenting each NSAID's efficacy; however, it is the class of medication rather than the specific brand that is effective. Caution is advised when prescribing NSAIDs because of the many adverse effects associated with these drugs that are dose-, frequency- and duration-dependent. Evidence exists to indicate that ibuprofen dosed at 400 mg is as effective in pain control as higher doses (600 mg and 800 mg) with fewer side effects and complications.[25] it is important that patients consume a snack with

NSAID use to decrease gastric irritation. In patients with a history of gastrointestinal bleed or at higher risk for GI bleeds, consider using a proton pump inhibitor with NSAIDs. While H2 blockers with NSAIDS reduce dyspepsia, they do not prevent adverse GI complications.[26]

Appropriate patient selection for NSAID use is necessary. Clinicians need to balance risk versus benefit, particularly in patients with a history of bleeding disorders, gastrointestinal bleeds/peptic ulcer disease, anticoagulant therapy, gastric bypass surgery, and NSAID-related rebound headaches.

All triptans work by inhibiting the release of vasoactive peptides, promoting vasoconstriction, and blocking pain pathways to the brainstem. The commonly available triptans and routes of administration are listed in **Table 2**.

The highest likelihood of consistent success is found with rizatriptan 10 mg, eletriptan 80 mg, and almotriptan 12.5 mg.[27]

One report suggests that IM injection of sumatriptan is more effective, followed by intranasal spray compared to pills. Adverse events, however, follow the same curve, with the highest incidence of side effects associated with injections and lowest incidence of side effects associated with oral routes.[28] The same likely is true of all triptans because they all have a similar mechanism of action.

The side effects most common with triptans are flushing/aresthesias/warm sensation of skin, dizziness, weakness, chest pressure or heaviness and injection site pain. Most of the side effects are temporary and resolve within 30 minutes.

Caution is advised when administering triptans to patients with cardiac problems. Acute myocardial infarction and sudden cardiac death have been reported with triptans, likely due to coronary artery constriction.[7]

To address nausea and vomiting in patients with migraine headaches, prokinetic/antiemetic medications can be prescribed. Metoclopramide (prokinetic; 10mg IV) helps with gastric emptying, thus reducing nausea and vomiting. Prochlorperazine (antiemetic; 10mg IV or IM) can be effective as monotherapy in some patients with migraine.[29] Diphenhydramine (12.5 – 20 mg IV) with metoclopramide and prochlorperazine are recommended to prevent akathisia and other dystonic reactions.[30]

While there is no evidence-based literature studying the effectiveness of ondansetron to treat nausea and vomiting associated with migraine headache, clinical experience supports it as a viable option.[31] Given that gastroparesis is common with migraine headaches, using the oral-dissolving-tablet (ODT) formula is the best option. The benefit of ondansetron is that it does not cause sedation. Caution is advised in patients with known or suspected long QT syndrome because this medication has been shown to cause QT prolongation. Another disadvantage to this medication is that a common reaction is headache.

High-flow oxygen (10–15 L/min with nonrebreathing mask for 15 min) has been shown to be an effective treatment for migraine, tension and cluster headaches alike.[32] As detailed in the cluster headache section, caution should be employed when using it in patients with a history of COPD.

Because migraine headaches, like many non-emergent headaches, have nerve-related etiologies (e.g., trigeminal or occipital) regional scalp anesthesia (occipital or ophthalmic nerve block) can be used to provide relief. Common anesthetics used include lidocaine (short-acting) and/or bupivacaine (long-acting).[33]

Clinical trials are being conducted on use of propofol for acute treatment of migraine headache.[24] One comparison of propofol to dexamethasone for acute treatment of migraine headache found that propofol was more effective with quicker headache resolution and no significant side effects.[34]

The FDA has approved the first medical device to treat migraine headache, which is available by prescription only. Called the Cerena Transcranial Magnetic Stimulator, it is indicated for migraine with aura headache. Using both hands, the patient holds the device to the back of his or her head and presses a button, which releases a pulse of magnetic energy that stimulates the occipital cortex to stop or reduce the pain.[35] The FDA reports that this device was effective in treating migraine pain in 38% (compared 17% in the control group) of people in a study of 113 participants. This devise does not help with associated symptoms of migraine (photophobia, phonophobia or nausea/ vomiting). The most common adverse reaction is dizziness and should not be used in patients with seizure disorder and is contraindicated in patients with metal in their head, neck or upper body that are attracted by a magnet.

Tension-type headache

Tension-type headache (TTH) is one of the most common reasons the general population uses over-the-counter (OTC) analgesics. Given the mild and temporary nature of this type of headache, few patients seek medical care and, as such, self-diagnose and treat with OTC medication.

The pathophysiology of TTH is not well established; however, is thought to be caused by activation of myofascial nociceptors. Reported precipitating factors include stress and mental tension.[36]

The symptoms of TTH are described as a headache of mild-to-moderate intensity that is bilateral, non-throbbing, and without other features. Patients often use terms such as "dull," "pressure." and "band-like" to describe their symptoms. Tenderness at the pericranial muscles and other myofascial trigger points of the head, neck, and shoulders is common. The neurological exam with TTH is normal.

The 3 main subtypes of TTH are infrequent episodic (<1 per month), frequent episodic TTH (1–14 days per month) and chronic TTH (>15 days per month). TTH is slightly more prevalent in women than in men and incidence peaks in the fourth decade of life.[37]

No diagnostic tests are necessary or recommended for TTH. The diagnosis is clinically based.

Treatment for TTH is with OTC NSAIDs. For patients with mild to moderate symptoms, a single dose of ibuprofen (200 mg to 400 mg), naproxen sodium (220 mg to 550 mg) or aspirin (650 mg to 1000 mg) can be given. For adults, the maximum dose in 24 hours is ibuprofen 2400 mg, naproxen sodium 1375 mg, and aspirin 4 g. If NSAIDs are contraindicated, acetaminophen 1000 mg is recommended. It can also be used as an adjunct to an NSAID. The maximum dose of acetaminophen in 24 hours for adults is 3250 mg.[39]

For patients whose headache fails to respond to simple NSAID/acetaminophen therapy, adding caffeine (65 mg) may provide relief.[39] A single IM injection of ketorolac (7.5 mg to 15 mg) should be considered for patients who present to an urgent care clinic with acute TTH and have moderate to severe pain.[40]

Patients should be counseled to avoid frequent use of OTC analgesics because of the risk of overuse headache (discussed below) and of gastrointestinal complications.

For patients with refractory TTH, tricyclic antidepressants (amitriptyline) and anticonvulsants (topiramate) can be considered in refractory cases, although data are sparse and caution is warranted, given the side-effect profile of these classes of medications.[41,42]

As detailed in the migraine section, high-flow oxygen therapy may be beneficial for TTH and has little risk in patients with no history of pulmonary disease. Scalp anesthesia should be considered in patients who present to an urgent care center with acute pain that has failed to respond to OTC analgesia and a nervous (trigeminal and/or occipital) or muscular component is suspected.[33]

In general, use of narcotic analgesia and muscle relaxers is not recommended. These medications have not been proven effective for treatment of TTH and their use poses concerns for habituation and adverse side effects.

Nonpharmacologic treatment options that may be helpful for some patients with TTH include heat, ice, massage, rest, EMG biofeedback, and stress management. Data are limited on these methods and one modality cannot be recommended over another.

Often a combination of these therapies is needed to maximize benefit for TTH.

Medication Overuse Headache

Estimates indicate that approximately 1% of the population suffer from medication overuse headache (MOH), also known as analgesic rebound headache, drug-induced headache. MOH can be challenging because often a patient has been self-treating an underlying headache disorder, frequently migraine or tension-type.[43,44]

The pathophysiology of MOH is likely facilitated by trigeminal pain. Chronic exposure to triptans or other analgesics is thought to cause a downregulation of serotonin receptors, inhibiting central pathways and translating to permanent head pain because of impairment of antinociceptive activity.[45]

The clinical features of MOH vary among patients. Because the underlying headache disorder is often migraine or TTH, patients will often describe features of these specific headaches. The key point is eliciting a history of frequent and excessive use of acute symptomatic medication.

MOH is more predominant in women than in men (as with migraine and TTH) and is often associated with substance dependency, anxiety, and psychological drug dependency.'[43,46]

Medications associated with the highest risk of MOH are opioids, butalbital-containing combination analgesics and aspirin/acetaminophen/caffeine combinations.[47,48] Triptans and ergotamine represent a modest risk of MOH, whereas NSAIDs are the lowest risk.[49]

The treatment for MOH is discontinuation of use of the causative medication. Withdrawal symptoms may include increased headache, nausea, vomiting, anxiety, nervousness, and sleep disturbances.[50] The withdrawal period usually lasts 2 to 10 days.[50]

Strategies for discontinuation of the medication fall into 2 categories.[45] With barbiturates, opioids, or benzodiazepines, the pace of withdrawal depends on the amount and frequency of use. For patients who use barbiturates or benzodiazepams in high doses or frequently, tapering the dose over a 2- to 4-week period is recommended. When discontinuing opioid use, consider using a once-weekly transdermal clonidine patch (0.1 to 0.2 mg/24) for 1 to 2 weeks to reduce withdrawal symptoms. For patients discontinuing butalbital, a phenobarbital taper is recommended for seizure precaution at 30 mg twice daily for 2 weeks followed by 15 mg twice daily for 2 weeks. Consider bridging therapy with NSAID treatment, and address the underlying cause of MOH, and explore preventative medication and lifestyle modalities.

For medications other than those previously discussed, abrupt discontinuation should be bridged with NSAIDs or prednisone.[50] The underlying etiology of the MOH should be established and preventative measures explored.

Conclusion

Headache is a common chief complaint encountered in urgent care medicine. Evaluation of patients with this complaint can be overwhelming for even the most experienced practitioner.

The more concerning constellation of symptoms are: sudden onset (thunderclap) of severe intensity, new and different headache, papilledema and any abnormal neurological signs and warrant an emergent workup. Less concerning features include headaches that are of more than 10 weeks duration, are recurrent without change, without focal neurological findings and follow patterns consistent with cluster, migraine, tension-type and medication overuse headaches.

Understanding the key features that are specific to each type of headache disorder will, it is hoped, help urgent care providers make appropriate diagnostic and treatment decisions. ■

REFERENCES

1. Russell MB, Anderson PG, Thomsen LL. Familial occurrence of cluster headache. J Neurol Neurosurg Psychiatry 1995; 58:341-343.
2. May A. Cluster headache: pathogenesis, diagnosis, and management. Lancet 2005; 366-843.
3. Bahra A, May A, Goadsby PJ. Cluster headache: a prospective clinical study with diagnostic implications. Neurology 2002; 58:354-361.
4. Drummond PD. Dysfunction of the sympathetic nervous system in cluster headache. Cephalgia 1988; 8:181-186.
5. Headache Classification Subcommittee of the International Headache Society. The International Classification of Headache Disorders: 2nd edition. Cephalgia 2004; 24 Suppl 1:9-160.
6. Francis GJ, Becker WJ, Pringsheim TM. Acute and preventive pharmacologic treatment of cluster headache, neurology 2010; 75:463-473.
7. MassenVanDenBrink A, Reekers M, Bax WA, et al. Coronary side-effect potential of current and prospective antimigraine drugs. Circulation 1998; 98:25-30.
8. Matharu MS, Levy MJ, Meeran K, Goadsby PJ. Subcutaneous octreotide in cluster headache: randomized placebo-controlled double-blind crossover study. Ann Neurol 2004; 56:488-494.
9. Lipton RB, Baggish JS, Stewart WF, et al. Efficacy and safety of acetaminophen in the treatment of migraine: results of a randomized, double-blind, placebo-controlled, population-based study. Arch Intern Med 2000; 160:3486-3492.
10. Leone M, D'Amico D, Frediani F, et al. verapamil in the prophylaxis of episodic cluster headache: a double-blind study versus placebo. Neurology 2000; 54:1382-1385.
11. Jarrar RG, Black DF, Dodick DW, Davis DH. Outcome of trigeminal nerve section in the treatment of chronic cluster headache. Neurology 2003; 60:1360-1362.
12. 12. Matharu MS, Goadsby PJ. Persistence of attacks of cluster headache after trigeminal nerve root section. Brain 2002; 125:976-984.
13. Primary care medicine: office evaluation and management of the adult patient. 6th edition. 2009. Chapter 165
14. Moskowitz MA, Nozaki K, Kraig RP. Neocortical spreading depression provokes the expression of c-fos protein like immunoreactivity within trigeminal nucleus caudalis via trigeminovascular mechanisms. J Neurosci 1993; 13:1167-1177.
15. Karatas H, Erdener SE, Gursoy-Ozdemir Y, et al. Spreading depression triggers headache by activating neuronal Panx1 channels. Science 2013; 339:1092-1095.

16. Kelman L. the premonitory symptoms (prodrome): a tertiary care study of 893 migraineurs. Headache 2004; 44:865-872.
17. Rabbie R, Derry S, Moore RA. Ibuprofen with or without an antiemetic for acute migraine headaches in adults. Cochrane Database Syst Rev 2013; 4:CD008041.
18. Cutrer FM, Huerter K. Migraine aura. Neurologist 2007; 13:118.
Charles A. The evolution of a migraine attack – a review of recent evidence. Headache 2013; 53:413-419.
19. Panayiotopoulos CP. Visual phenomena and headache in occipital epilepsy: a review, a systematic study and differentiation from migraine. Epileptic Discord 1999 Dec; 1 (40; 205-216.
21. Silberstein SD. Practice parametere: evidence-based guidelines for migraine headache (an evidence-based review): report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology 2000; 55:754-762.
22. Rabbie R, Derry S, Moore RA. Naproxen with or without an antiemetic for acute migraine headaches in adults. Cochrane Database Syst Rev 2013; 10:CD009455.
23. Rabbie R, Derry S, Moore RA. Diclofenac with or without an antiemetic for acute migraine headaches in adults. Cochrane Database Syst Rev 2013; 4:CD008783.
24. Taggart E, Doran S, Kokotillo, et al. Ketorolac in the treatment of acute migraine: a systematic review. Headache 2013; 53:277-287.
25. Rabbie R, Derry S, Moore RA. Ibuprofen with or without an antiemetic for acute migraine headaches in adults. Cochrane Database Syst Rev 2013; 4:CD008039
26. Ong CKS, Lirk p, Seymour RA. An evidence-based update on nonsteriodal anti-inflamatory drugs. Clin Med Res. Mar 2007; 5(1): 19-34.
27. Ferrari MD, Roon KI, Lipton RB, Goadsby PJ. Oral triptans (serotonin 5-HT (1B/1D) agonists) in acute migraine treatment: a meta-analysis of 53 trials. Lancet 2001; 358:1668-1675
28. Tfelt-Hanson P. Efficacy and adverse events of subcutaneous, oral, and intranasal sumatriptan used for migraine treatment: a systematic review based on number needed to treat. Cephalgia 1998; 18:532-538.
29. Kelly NE, Tepper DE. Rescue therapy for acute migraine. Headache. 2012; 52:467-482.
30. Vinson DR, Drotts DL. Diphenhydramine for the prevention of akathisia induced by prochlorperazine: a ramdomized, controlled trial. Ann Emerg Med. Feb 1001; 37(2):125-131.
31. Dulli D, Johnson S, Ochowski M, Rifkin A. Migraine assessment and treatment in a primary care setting – adult clinical practice guideline. Unity health insurance affiliated with UW Health. Reapproved and released 5/2013.
32. Ozkurt B, Cinar O, Erdem, C, et al. Efficacy of high-flow oxygen therapy in all types of headache: a prospective, randomized, placebo-controlled trial. American J of Emergency Medicine 2012 V30 issue 9; 1760:1764.
33. Cloyd J. Scalp Anesthesia. Medscape. Feb 21, 2013.
34. Soleimanpour H, Ghafouri R, Taheraghdam A, et al. Effectiveness of intravenous dexamethasone versus propofol for pain relief in the migraine headache. BMC Neurology. 2012; 12 (114).
35. Jeffery S. FDA approves first device to treat migraine pain. Medscape. Dec 13, 2013.
36. Spierings EL, Ranke AH, Honkoop PC. Precipitating and aggravating factors of migraine versus tension-type headache. Headache 2001; 41:554-558.
37. Rasmusssen BK, Jensen R, Schroll M, Olesen J. Epidemiology of headache in a general population – a prevalence study. J Clin Epidemiol 1991; 44:1147-1157.
38. Krenzelok EP, Royal MA. Confusion: acetaminophen dosing changes based on NO evidence in adults. Drugs R D 2012. June 1; 12 (2): 45-48
39. Motov S. Why ED physicians over prescribe ketorolac. Medscape sep 24, 2013.
40. Diamond S, Freitag FG. The use of ibuprofen and caffeine to treat tension-type headache. Curr Pain Headache Rep 2001; 5:472-478.
41. Jackson JL, Shimeall W, Sessums L, et al. Tricyclic antidepressants and headaches: systematic review and meta-analysis. BMJ 2010; 341:c5222.
42. Lampl C, Marecek S, May A, Bendtsen L. A prospective, open-label, long-term study of the efficacy and tolerability of topiramate in the prophylaxis of chronic tension-type headache. Cephalgia 2006; 26:1203-1208.
43. Pascual J, Colas R, Castillo J. Epidemiology of chronic daily headache. Curr Pain Headache Rep 2001; 5:529-536.
44. Mathew NT, Kurman R, Perez F. Drug induced refractory headache – clinical features and management. Headache 1990; 30:634-638.
45. Boes CJ, Black DF, Dodick DW. Pathophysiology and management of transformed migraine and medication overuse headache. Semin Neurol 2006; 26:232-241.
46. Saper JR, Hamel RL, Lake AE 3rd. Medication overuse headache (MOH) is a biobehavioural disorder. Cephalgia 2005; 25:545-546.
47. Dodick D, Freitag F. Evidence-based understanding of medication-overuse headache: clinical implications. Headache 2006; 46 Suppl 4:S202-211-.
48. Johnson JL, Hutchinson MR, Williams DB, Rolan P. Medication-overuse headache and opioid-induced hyperalgesia: a review of mechanism, a neuroimmune hypothesis and a novel approach to treatment. Cephalalgia 2013; 33:52-64.
49. Bigal ME, Rapoport AM, Sheftell FD, et al. Transformed migraine and medication overuse in a tertiary headache centre – clinical characteristics and treatment outcomes. Cephalalgia 2004; 24:483-490.
50. Diener HC, Limmroth V. Medication-overuse headache: a worldwide problem. Lancet Neurol 2004; 3:475-483.