Primary Headache

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KEYWORDS

• Primary headache • Migraine • Tension-type headache • Cluster headache
• Trigeminal autonomic cephalalgias • Pediatric headache • Headache in pregnancy
• Elderly headache

KEY POINTS

- Primary headaches account for the majority of ED headache presentations, with migraine being the most commonly diagnosed type.
- Red flag symptoms (SNOOP2 mnemonic) should always be assessed to rule out dangerous secondary headaches.
- Routine neuroimaging is unnecessary for primary headaches unless red flags are present.
- Opioids should be avoided due to inadequate sustained relief, dependence, and an increased risk of medication overuse headaches.
- The aim of the emergency physician is to rule out secondary causes of headaches, alleviate distress caused by these headaches, and arrange close follow-up.

PRIMARY HEADACHE

Introduction

Headache is one of the most common emergency department (ED) presentations, accounting for approximately 4 million visits annually (~2.6% of all visits).[1] While most cases are ultimately diagnosed as benign or primary headache processes, it is critical to consider dangerous diagnoses, such as subarachnoid hemorrhage, cervical artery dissection, meningitis, and encephalitis. Although roughly 5.5% of patients presenting with headaches to the ED end up with a pathologic diagnosis, neuroimaging is obtained in 14% of cases.[2] This burden of volume and resources underscores the importance of proper evaluation and risk stratification.

Regardless of the underlying cause, the primary goals in headache management include ruling out dangerous conditions, providing effective pain control, and ensuring follow-up. Primary headaches are typically grouped into 4 categories: migraine

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Abbreviations	
ED	emergency department
NSAID	nonsteroidal anti-inflammatory drug
TAC	trigeminal autonomic cephalalgia
TTH	tension-type headache

headaches, tension-type headaches (TTHs), trigeminal autonomic cephalgias, and other primary headaches. Despite the presence of a standard classification schema (https://ichd-3.org/), providing an exact diagnosis can be challenging, with up to one-third of patients going undiagnosed despite thorough evaluation.[3,4]

Evaluation

The initial evaluation of patients with headache should focus on differentiating primary from secondary etiologies. The SNNOOP10 mnemonic is useful in identifying "red flag" signs and symptoms that prompt further evaluation.[5,6] Evaluation and management of dangerous headaches are covered elsewhere.

*S*ystemic symptoms or signs including fever, rash, or weight loss.
*N*eurologic symptoms or signs including focal weakness, numbness, or confusion.
*N*eoplasm in history.
*O*nset that is sudden, maximal, and/or severe at onset.
*O*lder patients (>50 y old) with new onset or change in headache features.
*P*attern change or recent onset of headache.
*P*ositional headache.
*P*recipitated by sneezing, coughing or exercise.
*P*apilledema.
*P*rogressive headache or atypical symptoms.
*P*regnant or postpartum.
*P*ainful eye with autonomic features.
*P*ost-traumatic onset of headache.
*P*athology of the immune system, such as human immunodeficiency virus.
*P*ainkiller overuse or new drug at onset of headache.

After careful consideration of secondary headaches, the history should pivot to headache features that can differentiate primary headache causes. This includes asking about prodromal symptoms, associated symptoms, onset, duration, frequency, and features that differentiate the headache from prior episodes.

Patients with primary headache processes should have an unremarkable physical examination (with the exception of cluster headaches). Abnormalities found on examination or with vital signs should prompt the clinician to reconsider whether the patient actually has a secondary headache. A thorough neurologic assessment should be performed, including assessment of cranial nerves, strength, sensation, cerebellar function, and gait. Often, this can be challenging during the initial evaluation if the patient is suffering from significant pain. In these situations, treatment to alleviate symptoms and a complete neurologic assessment should be undertaken after improvement. A complete head and neck examination should also be performed as it may reveal causes of secondary headache including otitis media, odontogenic infections, sinusitis, and meningitis. Patients with visual complaints and headache should have visual acuity checked, intraocular pressure assessment (tonometry), and, depending on the scenario, fundoscopic evaluation looking for papilledema (or ocular ultrasound).

Diagnostic testing plays a limited role in evaluating and managing patients with primary headache processes and serves to risk stratify dangerous diagnoses. The

American College of Emergency Physicians (ACEP) and the American College of Radiology have created guidelines to support clinicians in determining the utility of advanced imaging.[7,8] As previously detailed, imaging is not necessary in the absence of "red flag" features. Laboratories are similarly rarely needed in evaluation and management but can be useful under certain circumstances. For instance, a positive pregnancy test may alter the likelihood of certain secondary headache processes (e.g. pre-eclampsia and cerebral venous thrombosis). Finally, an electrocardiogram (ECG) should be considered to check the QTc interval as many of the medications administered to abort headaches have QT-prolonging effects. This becomes more of an issue with repeated doses or when multiple QT-prolonging meds are administered.

Refer to **Table 1** for general headache pain management medications/dosages. Further details regarding specific treatment strategies for each major class of primary headache (e.g. migraine, tension, and cluster) are discussed under their appropriate header. Opioid-containing analgesics are strongly discouraged by both The ACEP and the American Academy of Neurology, given the potential for dependence, inadequate long-term relief, and risk of medication overuse headache.[9,10]

MIGRAINE

Background

Migraine headaches are the second most common type of primary headache, affecting 18% of US women and 9% of US men and accounting for at least 1.2 million yearly ED visits in the United States.[11,12] Migraines frequently disrupt the lives of those affected, and the frequency of these disruptions is used to evaluate severity.

Pathogenesis

The current theory focuses on neuronal dysfunction with cortical spreading of depression, resulting in neuronal and glial depolarization. This is believed to account for the different phases seen in migraines, including the prodrome, aura (when present), headache, and postdrome. Neuronal sensitization is believed to alter the response to nociceptive stimuli, resulting in varied pain responses. Serotonin and calcitonin-gene-related peptides are implicated in migraine pathophysiology and represent important targets for therapy. Endogenous and exogenous decreases in estrogen can precipitate migraine events in women.[13]

Clinical Presentation and Evaluation

Migraine headaches are characterized by recurrent pounding or throbbing pain that is typically unilateral and frequently accompanied by other symptoms, such as nausea, vomiting, and sensitivity to light, sound, or smells. A major challenge in diagnosis is the fact that many patients use the term migraine colloquially to refer to any moderate to severe intensity headache. As such, it is important to elicit specifics regarding symptomatology, frequency of headaches, and whether a physician has established the diagnosis in the past. The POUND mnemonic (see below) outlines some of the most common symptoms seen and can aid in ED diagnosis[14]:

*P*ulsatile quality of headache described.
*O*ne-day duration (<4 h suggests TTH).
*U*nilateral location.
*N*ausea or vomiting.
*D*isabling intensity.

Migraine headaches have 2 major forms: with and without aura. The International Headache Society has established criteria for both forms (**Table 2**).[3]

Table 1
Dosing/medication guide

Medication	Dosage/Route
Ibuprofen	400–600 mg by mouth every 4–6 h as needed (maximum daily dose 3200 mg)
Naproxen	440–500 mg by mouth as an initial dose followed by 220–250 mg by mouth every 8–12 h as needed (maximum daily dose 1500 mg)
Ketorolac	10–15 mg intravenous push every 4–6 h as needed
Acetaminophen	*Oral* Acetaminophen 10–15 mg/kg/dose (actual body weight), by mouth, every 4–8 h as needed (maximum 1000 mg/dose; maximum 75 mg/kg/day or 4000 mg/day, whichever is lower) *Parenteral dose* *For adults >50 kg*: 650 mg intravenously every 4 h or 1000 mg every 6 h as needed (maximum 1000 mg/dose; maximum 4000 mg/day) *For adults <50 kg*: 15 mg/kg intravenous every 6 h (maximum 750 mg/dose; maximum 75 mg/kg/day)
Dexamethasone	10 mg IV push over 2 min × 1 dose to reduce risk of migraine recurrence.
Droperidol	1.25–2.5 mg intravenous or intramuscular as a 1-time dose
Haloperidol	2.5–5 mg intravenous (maximum dose 5 mg) as a 1-time dose
Metoclopramide	10 mg slow intravenous push as a single dose over 1–2 min. Note: IV is preferred in the treatment of migraine, though it can be given intramuscularly. Additionally, some experts increase the maximum single dose to 20 mg for migraine with nausea and vomiting
Prochlorperazine	10 mg intravenous as a 1-time dose
Diphenhydramine	25–50 mg intravenous or intramuscular to prevent/treat extrapyramidal reactions
Magnesium Sulfate	1–2 g intravenous over 15–30 min as a 1-time dose Note: Particularly useful for migraine *with* aura
Sumatriptan	*Subcutaneous* 6 mg, 1-time dose (maximum 6 mg per dose; 12 mg in 24 h) May give an additional dose of 1–6 mg ≥1 h from the initial dose if migraine symptoms return or persist AND the patient had some response from the initial dose *Intranasal* *Imitrex (nasal spray):* 5, 10, 20 mg (maximum 40 mg in 24 h) May repeat dose in ≥2 h from initial dose only if there is some response from the initial dose *Onzetra Xsail (nasal powder):* 11 mg into each nostril (22 mg total) using the medication device (i.e., nose piece) (maximum 44 mg in 24 h) May repeat dose in ≥2 h from initial dose, only if there is some response from the initial dose *Tosymra (nasal spray):* 10 mg (1 actuation) as a single spray into one nostril (maximum 30 mg in 24 h) May repeat dose no more than 2 times ≥1 h from the previous dose (i.e., maximum total dose 30 mg in 24 h). Repeat dose only if there is some response from the initial dose.

Table 2
ICHD-3 classification diagnostic criteria for acute migraine headache without aura

Criteria	Requirements
Number of attacks *(A)*	At least 5 headache attacks, fulfilling criteria B-D
Duration *(B)*	Each attack lasts 4–72 h
Pain characteristics (At least 2 required) *(C)*	Unilateral location
	Pulsating quality
	Moderate or severe intensity
	Aggravation by or avoidance of routine physical activity
Additional Symptoms (At least 1 required) *(D)*	Nausea or vomiting
	Photophobia
	Phonophobia
Exclusion Criteria *(E)*	Not better accounted for by another disorder

Auras are reversible neurologic phenomena that precede migraine symptoms and can last for minutes at a time. Aura symptoms include visual (scintillation and/or scotomas), sensory (pins and needles, numbness), speech (aphasia), motor, brainstem, or retinal. Sensory and language symptoms typically occur in conjunction with visual symptoms. Interestingly, migraine with aura can occur without headache.[3]

Auras should not be confused with neurologic symptoms that occur with the headache itself. These symptoms can include visual, sensory, language, motor phenomena, vestibular symptoms, and can mimic acute stroke. A history of similar presentations may be present, but a stroke workup is often indicated in the absence of prior episodes. Patients may also describe prodromal symptoms, including depression, fatigue, yawning, depression, and decreased activity. These symptoms can also be present after the migraine has resolved (postdromal).

Complications

Significant complications are uncommon in migraines.[3] Status migrainosus is a debilitating migraine with or without aura that lasts for more than 72 h. Persistent aura without infarction is seen when aura symptoms last for more than 1 w in the absence of abnormalities on neuroimaging. Rarely, patients with migraine with aura can suffer from migrainous infarction, an ischemic stroke in the territory corresponding to the patient's aura. This is more common in young women. Gastrointestinal symptoms are common in migraines (e.g. nausea and vomiting), but some patients can suffer from cyclic vomiting syndrome and concomitant moderate to severe abdominal pain (i.e. abdominal migraines).

Medical/Pharmacologic Management

Patients with mild to moderate symptoms without nausea can be managed with oral medications, including acetaminophen and nonsteroidal anti-inflammatory drugs (NSAIDs).[15] However, most patients have progressed beyond this point by the time they present to the ED and require parenteral therapy.

There are a large number of parenteral treatment options for migraine headaches, but 3 classes of medications are established as first-line treatment: antidopaminergics, butyrophenones, and NSAIDs. Antidopaminergics (e.g. metoclopramide and prochlorperazine) have established efficacy both as monotherapy and in conjunction

with NSAIDs and address both nausea associated with migraines and the acute headache.[16] Though highly effective, administration of these agents can be accompanied by undesirable extrapyramidal symptoms (e.g. akathisia and dystonic reactions). Coadministration of an anticholinergic medication like diphenhydramine has been shown to reduce akathisia resulting from prochlorperazine but not from metoclopramide.[17,18] Akathisia can also be avoided by slowing the rate of administration.[19] If akathisia develops, it can be treated either with diphenhydramine or midazolam.[20]

Butyrophenones, similar to antidopaminergics, address both headache and nausea. Both parenteral haloperidol and droperidol are effective in relieving migraine symptoms.[21–23] Historically, there was a concern for prolongation of the QTc, but this has not been borne out in the literature.[24] It is reasonable to obtain an ECG before administration if feasible, and it is recommended if multiple doses are administered or if the patient is on other QTc prolonging medications. As with antidopaminergics, akathisia can occur after butyrophenone administration and can similarly be treated with diphenhydramine.

Parenteral NSAIDs (e.g. ketorolac) have shown modest efficacy as monotherapy when compared to antidopaminergics.[25] They are probably best used in combination with an antidopaminergic, though this approach has not been studied in clinical trials.

The triptan class of medications is serotonergic receptor agonists that mainly play a role in aborting migraines early in their course. Sumatriptan is available as a subcutaneous injection but is inferior to antidopaminergic agents and is commonly associated with adverse events that limit its utility in the ED.[26]

A number of agents have limited data regarding efficacy in migraine management and may be reserved for refractory cases. While steroids have not been shown to improve acute migraine pain, substantial evidence exists to endorse the use of dexamethasone in reducing headache recurrence.[27,28] Despite frequent use, magnesium has not been consistently shown to provide benefit in patients with migraines.[29] Intravenous fluids, without significant signs of dehydration, do not play a significant role in treatment.[26] Calcitonin gene-related peptide receptor antagonists are an emerging class of medications in migraine management. Outpatient data have been underwhelming and no ED studies have shown any benefit. Valproic acid is often considered in refractory cases but has demonstrated similar efficacy to ketorolac, prochlorperazine, and metoclopramide.[30] Ketamine and propofol have both been studied with mixed results.[31–33]

TENSION-TYPE HEADACHE

Background

TTH is a common condition characterized by bilateral, bandlike pain of mild to moderate intensity. It is the most common primary headache in the general population, with a lifetime prevalence between 30% and 78%.[2,34–36] Studies suggest that TTHs are more common in women than men, may be more prevalent in White Americans than Black Americans, and occur less frequently among the elderly.[34] TTH is classified by symptom frequency into infrequent episodic, frequent episodic, and chronic TTH.[3]

Pathogenesis

The precise mechanisms underlying TTH remain uncertain and arise from a complex interplay of peripheral and central mechanisms. Episodic TTH is driven by peripheral nociceptive activation, often linked to myofascial trigger points and muscle tenderness, while chronic TTH involves central sensitization, increased pain sensitivity, and impaired pain modulation.

Clinical Presentation

Patients typically report a mild to moderate, bilateral, non-throbbing headache described as "dull," "pressure," or "head fullness," and often compared to a "tight cap," "band-like," or a "heavy weight on the head or shoulders." While the pain is usually mild to moderate, it can occasionally be severe. Pericranial muscle tenderness is the most common abnormal finding in TTH, often reported in the head, neck, or shoulders and exacerbated during headache episodes.[3]

Evaluation

Patients with TTH should be evaluated for their headaches' frequency, duration, and potential triggers. Stress, mental strain, head or neck movements, sleep disturbances, dehydration, alcohol, and sunlight exposure are commonly reported triggers.[37] The causal role of these triggers remains unclear and a headache diary may help identify and manage individual triggers. Risk factors for secondary headaches should also be assessed.

The diagnosis of TTH relies on symptoms consistent with typical features, fulfillment of the International Classification of Headache Disorders (ICHD)-3 criteria (**Table 3**), and normal neurologic examination findings apart from increased pericranial muscle tenderness.[3] Always consider alternative diagnoses in patients unresponsive to initial therapies.

Medical/Pharmacologic Management

The first-line medication is typically an oral NSAID, such as ibuprofen, naproxen, ketoprofen, or aspirin.[36,38] These agents are preferred due to their effectiveness and lower risk of medication overuse headaches compared to acetaminophen, codeine, or butalbital. However, in patients who cannot tolerate NSAIDs or have contraindications to them, acetaminophen is considered a first-line agent.[39]

Table 3
ICHD-3 classification diagnostic criteria for episodic and chronic tension-type headaches

Criteria	Episodic TTH	Chronic TTH
Frequency (A)	≥10 episodes of headache fulfilling criteria B through D *Infrequent*: <1 d/mo on average (<12 d/y) *Frequent:* 1–14 days/month on average (≥12 and <180 days/year)	Headache ≥15 d/mo on average for >3 mo (≥180 d/y) and fulfilling criteria B through D
Duration (B)	30 min to 7 d	Hours to days or unremitting
At least two of the following criteria (C)	Bilateral location Pressing or tightening (non-pulsating) quality Mild to moderate intensity Not aggravated by routine physical activity (e.g. walking, climbing stairs)	
Both of the following (D)	No nausea or vomiting	Neither moderate or severe nausea nor vomiting
	No more than one of photophobia or phonophobia	No more than one of photophobia, phonophobia, or mild nausea
Exclusion (E)	Not better accounted for by another ICHD-3 diagnosis	

For severe or refractory cases, parenteral options, such as ketorolac, metoclopramide, and chlorpromazine, may provide rapid relief, particularly for patients with disabling symptoms.[40,41] Combination therapies containing caffeine (e.g. acetaminophen-aspirin-caffeine or acetaminophen-caffeine) may provide additional analgesic benefits for patients without response to NSAIDs or acetaminophen, but carry a higher risk of side effects like nausea or dizziness.[42,43]

Triptans, muscle relaxants, and local injections are treatments with limited or uncertain benefits.[44] While non-pharmacologic options like heat, ice, and massage lack robust evidence for acute TTH relief, they may offer adjunctive benefits for some patients, particularly as a preventive strategy. For chronic TTH, preventive therapy and addressing comorbid conditions, such as anxiety or depression are often necessary.

CLUSTER HEADACHE

Background

Cluster headache is the most prevalent and well-characterized subtype of trigeminal autonomic cephalalgia (TAC), which includes paroxysmal hemicrania, short-lasting unilateral neuralgiform headache attacks, and hemicrania continua.[45] Affecting approximately 0.1% of the population, cluster headaches primarily occur in males and typically begin between ages 20 and 40.[46,47] While tobacco use and head trauma are noted to be associated with cluster headaches, their role in its pathogenesis remains unclear.[48,49]

Pathogenesis

The pathogenesis of cluster headache remains unclear, but the leading theory suggests hypothalamic activation triggers the trigeminal-autonomic reflex, likely via a trigeminal-hypothalamic pathway.[45] Across TACs, this process likely involves activation of the ophthalmic division of the trigeminal nerve, parasympathetic outflow from the facial nerve, and neuropraxia of postganglionic fibers, which contribute to autonomic symptoms.

Clinical Presentation

Cluster headache presents as severe, unilateral pain in the orbit, periorbital region, or temple, often described as sharp or stabbing. The intensity is excruciating, causing restlessness, pacing, or rocking as patients struggle to remain still during an attack. Some may also experience cutaneous allodynia. Attacks are accompanied by ipsilateral cranial autonomic symptoms, including conjunctival injection, lacrimation, nasal congestion, rhinorrhea, eyelid edema, forehead or facial sweating, miosis, and ptosis.

Cluster headaches occur in cycles, with attacks lasting 15 to 180 min and occurring multiple times per day over weeks to months, followed by remission periods lasting months to years. They occur in 2 forms: episodic and chronic. The episodic form, affecting 80% to 90% of patients, includes remission periods lasting at least 3 mo. In contrast, chronic cluster headache persists for over a year without remission or with remissions shorter than 3 mo.[3,50] Some patients report circadian and seasonal patterns, with attacks more frequent at night and during spring or autumn. Alcohol and heat exposure are common triggers.[50,51]

Evaluation

Patients with suspected cluster headaches should be evaluated for attack frequency, duration, and the previously discussed triggers and risk factors. A detailed history and physical examination are essential to distinguish cluster headaches from other

headache syndromes. The diagnosis of cluster headache relies on symptoms consistent with typical features and fulfillment of the ICHD-3 criteria (**Table 4**).[3]

Neuroimaging, such as MRI, may be necessary in patients suspected of this diagnosis as secondary structural causes, such as pituitary adenomas, may mimic its presentation and are readily picked up by MRI.[52] Additional imaging of the face, sinuses, or vascular structures is reserved for atypical cases. Unless symptoms or physical examination suggest an alternative diagnosis, routine laboratory tests, electrophysiologic studies, and lumbar puncture are unnecessary.

Medical/Pharmacologic Management

First-line acute treatment should include 100% oxygen therapy at 12 to 15 L/min via a non-rebreather mask, followed by subcutaneous sumatriptan if there is no relief. Both treatments have shown high efficacy in rapidly terminating attacks and are supported by national guidelines and expert consensus.[53–56] Sumatriptan administered subcutaneously is the most effective pharmacologic option but is contraindicated in patients with ischemic heart disease, uncontrolled hypertension, or peripheral vascular

Table 4
ICHD-3 classification diagnostic criteria for cluster headaches

Criteria	Cluster Headache
Number of attacks *(A)*	At least 5 attacks fulfilling criteria B through D
Severity *(B)*	Severe or very severe unilateral orbital, supraorbital, and/or temporal pain lasting 15–180 min when untreated; during part (but less than half) of the active time course of cluster headache, attacks may be less severe and/or of shorter or longer duration
Symptoms: Either or both of the following (*C*)	At least one of the following symptoms or signs ipsilateral to the headache: a. Conjunctival injection and/or lacrimation b. Nasal congestion and/or rhinorrhea c. Eyelid edema d. Forehead and facial sweating e. Miosis and/or ptosis A sense of restlessness or agitation
Frequency *(D)*	Attacks have a frequency between one every other day and 8 per day; during part (but less than half) of the active time course of cluster headache, attacks may be less frequent
Exclusion criteria *(E)*	Not better accounted for by another ICHD-3 diagnosis
Episodic Cluster Headache	
A	Attacks fulfilling criteria for cluster headache and occurring in bouts (cluster periods)
B	At least 2 cluster periods lasting from 7 d to 1 y (when untreated) and separated by pain-free remission periods of 3 mo or more
Episodic Cluster Headache	
A	Attacks fulfilling criteria for cluster headache
B	Attacks occurring without a remission period, or with remissions lasting less than 3 mo, for at least 1 y

disease. If the patient cannot tolerate subcutaneous therapy, intranasal triptans are appropriate and administered contralateral to the side of the headache.[56] Oxygen therapy is well-tolerated and relieves nearly two-thirds of patients, making it the preferred option.

For patients who do not respond to first-line therapies, alternatives include intranasal lidocaine and ergot derivatives, such as oral ergotamine or intravenous dihydroergotamine. Intranasal lidocaine, in particular, is easy to administer and has minimal systemic side effects, making it an attractive option in the ED. However, its efficacy is lower than triptans.[57] Ergots may be useful in select patients, particularly for nighttime attacks, due to their longer half-life than triptans. However, they should not be used within 24 h of triptan administration due to the risk of vasospasm and are contraindicated in pregnancy.[58] Robust data regarding these medications' efficacy in ED patients are lacking.

A plan for prophylaxis or mitigation of recurrent episodes should be considered for cluster headache patients before discharge from the ED. Prevention of recurrent attacks is essential, as patients with cluster headaches have an increased risk of suicide.[50,59] Verapamil is the most widely used preventive agent and is efficacious in treating both episodic and chronic cluster headaches, but requires a relatively long-titration time, making outpatient follow-up crucial.[60] Glucocorticoids can be used for short-term prophylaxis, particularly in episodic cluster headaches with short active periods, or as a bridge while titrating verapamil. There are no data favoring 1 glucocorticoid regimen over another. An example regimen might include prednisone 100 mg once a day for 3 d and then tapering by decreasing the dose 10 mg every third day. In refractory cases, alternatives, such as galcanezumab, lithium, and topiramate, have been studied, though their roles remain secondary.[55]

Other Medical Management

Nerve blocks with local anesthetics are effective adjuncts to standard medical management, with increasing evidence supporting their efficacy, particularly for occipital and sphenopalatine ganglion nerve blocks.[61] The greater occipital nerve block, which involves injecting local anesthetic near the greater occipital nerve (with or without an additional injection near the lesser occipital nerve), was found to be superior to placebo but not as effective as standard treatment with metoclopramide.[62,63] This block can be combined with a supraorbital block.[64] The sphenopalatine block involves placing a cotton-tipped applicator soaked in local anesthetic into the naris until it reaches the posterior pharynx. Data for this treatment show improvement in migraine headaches.[65,66]

Patients with chronic headache syndromes may be treated with botulinum toxin injections (Botox) in the outpatient setting. The most common side effects after injection are pain, swelling, and erythema at the injection site. Uncommon complications that may prompt ED presentation include ptosis, dysphagia, and weakness in the shoulders and neck. These effects occur when the toxin spreads past the injection site and will typically resolve on its own. Rarely, the spread of toxin beyond local areas can cause breathing issues. Supportive care with non-invasive or invasive ventilation is indicated in these cases.

DISCUSSION

Challenges in Diagnosis

Diagnosing primary headaches is difficult due to overlapping symptoms across headache types. Variability in patient-reported symptoms, treatment response, and

frequency further complicate classification. Distinguishing primary from secondary headaches, such as subarachnoid hemorrhage, meningitis, or cerebral venous sinus thrombosis, is critical. Secondary headaches can mimic migraines or TTHs, leading to misdiagnosis if red flags—sudden onset, focal neurologic deficits, or immunocompromised status—are missed. Overuse of neuroimaging in low-risk patients increases unnecessary testing, while underuse in high-risk patients can delay life-threatening diagnoses.

Challenges in Management

Treatment efficacy varies, requiring trial-and-error with different pharmacologic agents. Medication overuse can worsen headache frequency and severity. Pain control must be balanced against medication side effects, such as QT prolongation, nephrotoxicity, and cardiovascular risks associated with antidopaminergics, triptans, and NSAIDs.

Setting realistic patient expectations is the key. Headache management often requires multiple medication trials and lifestyle modifications. Acute treatments may not work immediately or universally, sometimes necessitating combination therapy. Preventive strategies, including avoiding triggers, optimizing sleep, and stress reduction, are essential for patients with frequent or disabling headaches. Proper follow-up ensures ongoing treatment adjustments and helps patients recognize when changes in headache pattern or severity warrant re-evaluation.

Special Populations

Pregnancy and lactation

Acetaminophen and metoclopramide are first-line, with caffeine and diphenhydramine as adjuncts. Nerve blocks are a safe option. NSAIDs should be avoided in the third trimester and minimized in the first. Triptans, ondansetron, and magnesium may be considered with careful risk-benefit assessment. Critical diagnoses to consider include preeclampsia, cerebral venous thrombosis, and pituitary apoplexy.[66]

Pediatrics

Non-invasive treatments are preferred, with hydration, rest, and trigger avoidance playing a larger role. For pharmacologic management, weight-based dosing is essential with special attention to age restrictions (e.g. triptans not for <5 y, promethazine not for <2 y). NSAIDs and acetaminophen are first-line agents. Other medications have limited evidence in pediatrics and are reserved for severe or refractory cases, with common parenteral options including prochlorperazine with diphenhydramine and ketorolac. Alternatives include metoclopramide or dihydroergotamine. Promethazine or ondansetron may be added for nausea.

Older adults

NSAIDs pose higher risks of gastrointestinal bleeding, renal dysfunction, and cardiovascular events. Polypharmacy must be carefully managed due to drug interactions and impaired metabolism. Additionally, older adults are at higher risk for secondary headaches and warrant careful evaluation, including imaging as needed.

SUMMARY

Primary headaches, including migraines, TTHs, and trigeminal autonomic cephalgias, are common ED presentations. While benign, they must be differentiated from dangerous secondary headaches through history-taking, red flag recognition, and selective imaging. Treatment should be tailored to headache type when possible, with

parenteral therapy often required in the ED. Opioids should be avoided due to their association with dependence and medication overuse headaches. Special populations require adjusted approaches: NSAIDs should be avoided in late pregnancy, pediatric patients need weight-based dosing, and older adults require careful medication selection. Preventive measures, including lifestyle modifications and prophylactic medications, are critical for patients with frequent or severe headaches. Proper follow-up and patient education are essential to long-term headache management, reducing unnecessary ED visits and optimizing care.

CLINICS CARE POINTS

- Headaches are one of the most common reasons patients present to the emergency department. The vast majority of headaches in the ED are primary. Still, secondary headaches must always be considered. Use the SNNOOP10 mnemonic to screen for dangerous secondary causes.
- The aim of the emergency clinician should be to alleviate the distress caused by these non-life-threatening headaches and ensure a clear outpatient treatment plan, including preventive therapy where appropriate and discussion of strategies to avoid medication overuse headaches.
- Routine neuroimaging is unnecessary for primary headaches unless red flags are present—computed tomography (CT) or MRI should be reserved for cases where secondary causes cannot be excluded based on history and examination.
- Opioids should be avoided due to poor efficacy and the risk of medication overuse headache; instead, focus on targeted treatment with NSAIDs, antiemetics, intravenous (IV)fluids, and other adjuncts, such as nerve blocks.
- Consider special populations carefully, including pregnant or lactating patients, pediatric patients, or the elderly, to rule out secondary causes of headaches and to tailor treatment to population and patient-specific factors.

DISCLOSURES

The authors have no disclosures.

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