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Management Of Headache In The Pediatric Emergency Department

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CME Objectives

Upon completion of this article, you should be able to:

1. Distinguish primary and secondary headaches.
2. Formulate a disease-specific treatment plan for headaches in children.
3. Determine prudent use of diagnostic imaging and laboratory testing, when indicated.

Prior to beginning this activity, see the back page for faculty disclosures and CME accreditation information.

Abstract

Headaches are a common complaint among children, with increasing frequency in adolescence. According to the Agency for Healthcare Research and Quality, more than 3 million Americans seek emergency care every year due to headaches, and one-third of them are attributable to migraines. Headaches have a significant impact on the lives of children and adolescents, resulting in school absence, decreased extracurricular activities, and poor academic achievement. Among patients, the spectrum of pathology varies widely, continually challenging healthcare providers to recognize serious, life-threatening conditions, while judiciously evaluating and treating all patients. This issue reviews the broad differential of primary and secondary headaches in the pediatric emergency department, summarizes effective strategies for diagnosis, and evaluates the current evidence supporting safe, appropriate treatment. As emergency clinicians treat increasingly more medically complex patients, they should be aware of the best current practices to evaluate and treat headaches in the pediatric population.

Case Presentations

A 14-year-old girl with no significant past medical history presents to your ED with a chief complaint of headache. She describes the pain as 9/10, bifrontal, and associated with nausea and photophobia. She denies fevers, recent illness, or any trauma. When you review her family history, you note that her mother suffers from frequent headaches. Her vital signs and physical exam are all within normal limits.

An 11-year-old boy presents to your ED with a persistent headache. His past medical history is notable for a helmet-to-helmet collision during a football game 1 month prior to presentation. Since that time, he has also complained of difficulty concentrating, dizziness, and fatigue. On your exam, you note a poorly cooperative boy with normal vital signs who is in no acute distress. He repeatedly asks you to lower your voice and refuses to cooperate with your ophthalmic exam. The remainder of your physical exam is unrevealing.

An 18-year-old male presents to your ED with severe headache and fever for the past 3 days. Your examination reveals an ill-appearing male with photophobia and neck stiffness.

How would you approach the evaluation and treatment of these headaches?

Introduction

The presence of a severe headache is anxiety-provoking in both parents and children. When treating patients with headaches in the emergency department (ED), the primary objective of the emergency clinician is to promptly recognize the life-threatening conditions requiring immediate medical or operative management. In addition, appropriate assessment and treatment of less-severe headaches have the potential to prevent unnecessary hospital admissions. A recent study evaluating patients who presented to the ED with a chief complaint of headache demonstrated that the most common cause of headache was upper respiratory infection (19.2%). Migraines, posttraumatic headaches, and tension-type headaches accounted for 18.5%, 5.5%, and 4.6%, respectively. Serious, life-threatening headaches (4.1%) including meningitis (1.6%), acute hydrocephalus (0.9%), and tumors (0.7%) were less-common etiologies.[1] For optimal assessment and management of headaches, emergency clinicians must be familiar with the broad clinical spectrum of etiologies for headache in the pediatric population.

Prevalence Of Headache

Ninety-six percent of American adults report having had a headache in their lifetime, and nearly 40% have had a significant headache at some point.[2] Among children, the prevalence of major headache ranges from 37% to 51% during the elementary school years and gradually rises to 57% to 82% by adolescence. Frequent or severe headaches (including migraines) were reported by 17% of participants in a national sample of children and adolescents.[3] Headache ranks as the third leading cause of referral to a pediatric ED.[4] The most common type of recurrent headache in childhood is migraine; in adolescence, tension headaches are the most common type of frequent headache.[5]

Estimates of the overall prevalence of headache in children vary among researchers. Secondary headaches are most frequently encountered before the age of 5 years; however, a primary headache (such as migraine) can occur as early as a few months of age. Chronic tension-type headache has been reported in 0.9% of 15-year-old children.[6]

In a widely cited study, Bille surveyed 8993 children aged 7 to 15 years and found that 59% had suffered headache at some time in their life.[7] In a systematic questionnaire of 2941 children, Sillanpaa found the prevalence of headache to be 37% at age 7 years, increasing to 69% by 14 years; migraine accounted for 2.7% and 10.6% of these headaches, respectively.[8]

Studies have shown that up to 51% of children aged 7 years and 57% to 82% of adolescents aged 15 years report recurrent headaches.[9,10] A study performed in Taiwan indicated that approximately 85% of children aged 13 to 15 years have had headache.[11] According to a large survey by Split and Neuman, 75% of children have suffered headaches by age 15 years.[12]

Critical Appraisal Of The Literature

A literature search was performed using the following databases: PubMed, Web of Science, Ovid MEDLINE®, Cochrane Database of Systematic Reviews, and Scopus. Searches were limited to those published in English. Search terms included *pediatric headache, child, children, emergency, primary headache,* and *secondary headache.* Defining the specific type of headache further refined the search, using the terms *migraine, aura, migraine equivalent, tension-type, cluster, posttraumatic, concussion, pseudotumor cerebri, intracranial hypertension, sinusitis, intracranial mass, medication overuse, seizure, infection,* and *meningitis.* The search returned 12,155 abstracts that were reviewed for relevance. The bibliographies of the relevant articles were also reviewed for additional publications. In addition, guidelines from the Agency for Healthcare Research and Quality (AHRQ) through the National Guidelines Clearinghouse (www.guidelines.gov) were reviewed. Review of the literature revealed a tremendous body of data available from adult studies, from which pediatric treatments have been extrapolated. The pediatric literature is growing, and the available data from pediatric studies are reviewed in this article.

Classification Of Headache Disorders

Headache is defined as pain located above the orbitomeatal line.[13] The International Headache Society (IHS) publishes a standardized classification system that provides diagnostic criteria and a classification scheme for headaches in general. In the IHS Classification System, headaches are grouped on the basis of origin in order to facilitate evaluation and treatment. An update to the 2004 version (ICD-2) was published as a beta version in July 2013. The ICD-3 is available in full text at: http://www.ihs-classification.org/_downloads/mixed/International-Headache-Classification-III-ICHD-III-2013-Beta.pdf.[13]

The IHS Classification System includes the following basic headache types:

- Primary headaches
- Secondary headaches
- Cranial neuralgias, central and primary facial pain, and other headaches

Primary headache disorders are those in which the symptoms cannot be attributed to another cause, and they include migraines, tension-type headaches, and cluster headaches. The most common primary headaches in children are migraine and tension-type headaches, representing 2 separate but similar diagnoses with overlapping symptoms and similar mechanisms of pain. Both migraine and tension-type headaches can be episodic or chronic and daily (ie, they present 15 or more days per month for 3 or more months).

Secondary headache disorders are those in which the headache is a symptom of an identifiable structural, metabolic, or other abnormality; etiologies include trauma, neoplasms, vascular disease, meningitis, and infection.

Primary Headache

Migraine Headache

Migraine headaches constitute the vast majority of primary headaches among children and adolescents. Pediatric migraines are often characterized by bilateral head pain, although clear localization of the pain can be difficult to obtain from children. Migraines in children are often of shorter duration than they are in adults. Migraine can be divided into 2 groups: migraine with aura and migraine without aura. These 2 groups have historically been referred to as "classic" and "common" migraines, respectively, but this terminology is now outdated.

According to Sillanpaa, migraine prevalence is around 11% at puberty (age 13 years) but increases over time.[14] In a meta-analysis of over 25,000 cases, Lewis et al found the incidence of migraine to be 2% by ages 3 to 7 years, 7% by ages 7 to 11 years, and 20% by ages 11 to 15 years.[10] The survey by Split and Neuman indicated that 4% of children have migraine by the ages of 7 through 15 years; by age 15 years, 28% have migraine.[12] Approximately 60% of all children who have onset of migraines before puberty are male. After puberty, females outnumber males 3:1. Other headache types are distributed more evenly.[15]

Migraine Without Aura

Migraine without aura is identified by at least 5 previous headache episodes that fulfill the criteria noted in **Table 1**.

Migraine With Aura

Migraines with aura are headaches that are accompanied by transient neurologic symptoms. These symptoms may occur immediately before, during, or after the headache.[13] In some situations, the headache may be mild or nonexistent.[16] Migraine with aura is seen in 14% to 30% of children with migraine, although it is difficult to know the true incidence of migraine with aura since young children might not be able to verbalize aura symptoms. A typical aura may be visual, sensory, verbal, or a combination of all 3. **(See Table 2, page 4.)** Onset of the aura is gradual, usually lasts no more than 60 minutes, and accompanies the following types of headache:

- Typical aura with migraine headache
- Typical aura with nonmigraine headache
- Typical aura without headache
- Familial hemiplegic migraine
- Sporadic hemiplegic migraine
- Basilar-type migraine

Table 1. International Headache Society Diagnostic Criteria: Migraine Without Aura

A. At least 5 attacks fulfilling criteria B-D.
B. Headache attacks lasting 4-72 hours (untreated or unsuccessfully treated).*
C. Headache has at least 2 of the following characteristics:
   1. Unilateral location†
   2. Pulsating quality
   3. Moderate or severe pain intensity
   4. Aggravation by or causing avoidance of routine physical activity
D. During headache, at least 1 of the following:
   5. Nausea and/or vomiting
   6. Photophobia and phonophobia
E. Not better accounted for by another ICHD-3 diagnosis.

*In children and adolescents (aged < 18 y), attacks may last 2-72 h.
†Migraine headache in children and adolescents (aged < 18 y) is more often bilateral than is the case in adults; unilateral pain usually emerges in late adolescence or early adult life. Migraine headache is usually frontotemporal. Occipital headache in children is rare and calls for diagnostic caution.

Rare Forms Of Migraine

Migraine can, rarely, be accompanied by worrisome focal symptoms. In these cases, the diagnosis of migraine should be considered a diagnosis of exclusion.

Familial hemiplegic migraine, while unusual, is seen more commonly in children than in adults. This type of headache is characterized by abrupt onset of hemiparesis, which is usually followed by a headache. Hemianesthesia may also precede the headache. Familial hemiplegic migraines represent a genetically heterogeneous autosomal dominant subtype of migraine with aura caused by mutations in genes encoding ion channels, and they are associated with other disorders, including episodic ataxia and generalized epilepsy with febrile seizures.[17]

Basilar artery migraine is a subtype of migraine with aura that is more common in girls. It is characterized by at least 2 of the following: dysarthria, tinnitus, hypacusia, diplopia, decreased level of consciousness, vertigo, ataxia, visual disturbances in both hemifields, or bilateral sensory symptoms. They may mimic posterior fossa abnormalities such as arteriovenous malformations, cavernous angiomas, neoplasms, or congenital malformations.

Ophthalmoplegic migraine, another rare variant, is defined by 2 or more headache episodes accompanied by paresis of 1 or more of the third, fourth, and/or sixth cranial nerves.[18] A magnetic resonance imaging (MRI) study with contrast is helpful to exclude parasellar, orbital fissure, and posterior fossa lesions as a cause of the cranial nerve paresis.

Retinal migraine is characterized by brief (from a few seconds to 60 min in length), sudden, monocular blackouts or "grayouts;" or bright, blind episodes of visual disturbance before, during, or after headache attacks.

"Alice in Wonderland" syndrome, as its eponym suggests, consists of unusual visual illusions and spatial distortions that precede headaches. Visual distortions include micropsia (objects appear smaller), macropsia (objects appear larger), metamorphopsia (objects appear abnormally shaped), and teleopsia (objects appear farther away).

Acute confusional migraine typically lasts 4 to 24 hours and is associated with agitation, lethargy, and impaired sensorium. Focal neurologic deficits including aphasia, anisocoria, and memory deficits may also be seen.

Migraine equivalents are periodic paroxysmal syndromes without associated headache. They are most commonly seen in young children and are caused by similar migrainous mechanisms. These include:

- **Cyclic vomiting** is characterized by stereotyped episodes of high-frequency vomiting (more than 4 times/h) with interval return to normalcy. Physical examination and extensive gastrointestinal workup are within normal limits. It typically occurs among school-aged children, but it may occur among adults.
- **Abdominal migraine** is often a precursor to more typical migraine headaches, and it refers to recurrent episodes of moderate to severe abdominal pain with associated anorexia, nausea, vomiting, and/or pallor, with return to baseline between episodes.
- **Benign paroxysmal vertigo** is defined by episodes of sudden dizziness lasting minutes to hours with interval resolution. This is more typical among young children.
- **Benign paroxysmal torticollis** typically occurs among infants or young children, and it refers to intermittent episodes of head tilt with interval resolution.

Table 2. International Headache Society Diagnostic Criteria: Migraine With Aura

A.	At least 2 attacks fulfilling criteria B-D.
B.	One or more of the following fully reversible aura symptoms:
	1. Visual
	2. Sensory
	3. Speech and/or language
	4. Motor
	5. Brainstem
	6. Retinal
C.	At least 2 of the following:
	4. At least 1 aura symptom spreads gradually over ≥ 5 minutes, and/or 2 or more symptoms occur in succession
	5. Each individual aura symptom lasts 5 to 60 minutes
	6. At least 1 aura symptom is unilateral
	7. The aura is accompanied, or followed within 60 minutes, by a headache
H.	Not better accounted for by another ICHD-3 diagnosis, and transient ischemic attack has been excluded.

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Tension-Type Headache

Tension-type headaches are similar to migraines in terms of the onset of symptoms. Convergence theorists argue that tension-type headaches and migraines lie within a spectrum of the same disorder;[19] however, the pain is typically described as a band-like sensation around the head, and it may be associated with neck and/or shoulder pain. These headaches often become worse as the day progresses and can last for days. They may be associated with stressful events at home or at school, and they may be temporarily relieved by sleep. **(See Table 3.)**

Cluster Headache

Cluster headaches have been described as the most painful type of primary headache. Diagnostic crite-

ria include attacks of severe, unilateral pain lasting 15 to 180 minutes and occurring, in series, up to 8 times per day. **(See Table 4.)** Classically, the pain originates orbitally or temporally and is associated with nasal congestion, rhinorrhea, sweating, and ipsilateral lacrimation, conjunctival injection, miosis, ptosis, or eyelid edema. Cluster headaches can be further divided into episodic and chronic types. Episodic cluster headaches can last anywhere between 7 days and 1 year, with periods of remission lasting 1 month or longer. Cluster headaches are considered chronic after 1 year of symptoms or when pain-free periods last < 1 month.

Table 5 (page 6) summarizes the most common forms of primary headache.

Pathophysiology

Primary Headache

Migraine

Migraine pain attacks involve complex factors, and the precise mechanism has not yet been clearly delineated. Although much remains to be discovered, the pain in migraine attacks appears to be multifactorial.

Trigeminovascular System Activation

One suggested mechanism of migraine includes neurotransmitter activation of the trigeminovascular system, where proinflammatory mediators (substance P, calcitonin-gene-related peptide, vasoactive intestinal peptide) are released at the level of the meningeal and basal cerebral vessels following trigeminal nerve stimulation. Upstream triggers remain unclear; however, a cycle of neurogenic inflammation occurs downstream, causing further stimulation of nerve endings that results in afferent pain signals transmitted to the thalamus and cerebral cortex. This mechanism provides a target for commonly used migraine medications (such as the triptans), which activate serotonin receptors $5HT_{1B}$ and $5HT_{1D}$ to cause vasoconstriction, decreased release of proinflammatory mediators, and dampened transmission of afferent pain impulses.

It remains unclear whether these afferent pathways in migraineurs develop abnormally from external insult or are genetically determined. Evidence suggests that the brains of migraineurs have cortical hyperexcitability due to defective catecholamine release and low magnesium levels. White-matter T2 MRI hyperintensities are observed more frequently in migraineurs with aura, especially in the posterior circulation territories; however, the pathophysiologic implication of this remains unclear. Based, in part, on studies by Burstein et al of migraineurs with cutaneous allodynia, theories about chronic transformation of migraine suggest spatial and temporal progression followed by peripheral and central nociceptor sensitization that alters pain perception.[21]

Cortical Spreading Depression

Another mechanism thought to result in migraine headache has its origin in the brainstem. After

Table 3. International Headache Society Diagnostic Criteria: Tension-Type Headache

A. Minimum of 10 episodes and fulfilling criteria B-D.*
B. Headache lasting from 30 minutes to 7 days.
C. Headache has at least 2 of the following characteristics:
    1. Bilateral location
    2. Pressing/tightening (nonpulsating) quality
    3. Mild or moderate intensity
    4. Not aggravated by routine physical activity such as walking or climbing stairs
D. Both of the following:
    5. No nausea or vomiting
    6. No more than 1 of photophobia or phonophobia
E. Not better accounted for by another ICHD-3 diagnosis.

*Tension-type headaches can be subclassified into 1 of 3 categories:

- Infrequent episodic: At least 10 episodes occurring on < 1 day/mo on average (< 12 days/y) and fulfilling criteria B-D.
- Frequent episodic: At least 10 episodes occurring on 1-14 days/mo, on average, for > 3 mo (≥ 12 and < 180 days/y) and fulfilling criteria B-D.
- Chronic: Occurring ≥ 15 days/mo, on average, for > 3 mo (≥ 180 days/y) and fulfilling criteria B-D.

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Table 4. International Headache Society Diagnostic Criteria: Cluster Headache

A. At least 5 attacks fulfilling criteria B-D.
B. Severe or very severe unilateral orbital, supraorbital, and/or temporal pain lasting 15-180 minutes (when untreated).
C. Either or both of the following:
    1. At least 1 of the following symptoms or signs, ipsilateral to the headache:
        b. Conjunctival injection and/or lacrimation
        c. Nasal congestion and/or rhinorrhea
        d. Eyelid edema
        e. Forehead and facial sweating
        f. Forehead and facial flushing
        g. Sensation of fullness in the ear
        h. Miosis and/or ptosis
    2. A sense of restlessness or agitation
C. Attacks have a frequency between 1 every other day and 8 per day for more than half of the time when the disorder is active.
D. Not better accounted for by another ICHD-3 diagnosis.

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trauma or disturbances in local ion concentrations (hydrogen, potassium, and glutamate), cortical spreading depression takes place. Cortical spreading depression is characterized by neuronal activation followed by suppression; cerebral blood flow changes in parallel with hyperperfusion, followed by hypoperfusion. Cortical spreading depression activates central nervous system nociceptors (possibly through the release of nitric oxide, atrionatriuretic factor, activation of noradrenergic pathways, and/or changes in cerebral blood flow). Cortical spreading depression also causes neurogenic inflammation, which stimulates the release of several different neurotransmitters that lead to cerebral vasodilatation and activation of central nervous system nociceptors. The onset of the aura in migraine headache may also be mediated by cortical spreading depression.

Genetic Predisposition

Migraine headaches likely have a genetic predisposition; nearly 70% of pediatric patients with migraine have a family history of migraine headache. Some individuals with familial hemiplegic migraine have been found to have several genetic mutations in ion channels responsible for neurotransmitter release within the central nervous system.[22]

Tension-Type Headache

The heterogeneous pathogenesis of tension-type headache is not completely understood. A combination of pericranial muscular factors, abnormal nociceptive mechanisms, and poor emotional coping mechanisms exist, all possibly linked to brainstem serotonergic interneurons, leaving certain patients more susceptible to developing tension-type headaches. Furthermore, central and peripheral sensitization of pain fibers may be involved. Insufficient endogenous pain responses (either central or peripheral) are likely responsible for chronic conversion of episodic tension-type headache.[20]

Cluster Headache

The pathophysiology of cluster headache is not completely understood. It has been suggested that acute attacks involve activation of the trigeminovascular system, with greater symptoms and neuropeptide concentrations measured within the ophthalmic branch of the trigeminal nerve. Theories also suggest hypothalamic involvement, particularly the posterior hypothalamus, since functional imaging studies have demonstrated activity among patients with cluster headaches. Furthermore, the periodic nature and associated neuroendocrine changes with cluster headaches implicates the hypothalamus.[23]

Secondary Headache

Posttraumatic Headache

Posttraumatic headaches are common and may be associated with somatic, psychological, or cognitive disturbances. The pathogenesis remains unclear. The initial, acute phase is nociceptive; however, there is inadequate evidence to explain the chronic head pain that follows. Traumatic injury may serve as a trigger for the trigeminovascular mechanism well described among migraineurs.[24]

Idiopathic Intracranial Hypertension

Idiopathic intracranial hypertension (pseudotumor cerebri) is caused by the expansion of 1 or more of

Table 5. Typical Primary Headache Characteristics

Headache Type	Location	Characteristics	Patient Appearance	Duration	Associated Symptoms
Migraine headache	Unilateral 60%-70%; bilateral 30%	Gradual onset, crescendo pattern; pulsating; moderate-to-severe intensity; aggravated by routine physical activity	Seeks rest in dark, quiet room	1 hour to 3 days	Nausea, vomiting, photophobia, phonophobia, +/- aura (visual, speech/motor deficits)
Tension-type headache	Bilateral	Pressure or tightness that waxes and wanes	Variable	Variable	Typically, none (may have anorexia, photophobia, or phonophobia)
Cluster headache	Unilateral only; usually begins periorbitally or temporally	Pain begins quickly, reaches a crescendo within minutes; pain is deep, continuous, excruciating, and explosive in quality	Remains active (or hyperactive, pacing)	15 minutes to 3 hours	Ipsilateral lacrimation, nasal congestion, rhinorrhea, pallor, sweating, Horner syndrome

the intracranial fluid spaces, ie, the vasculature, extracellular fluid compartment, or cerebrospinal fluid space. Several drugs have been implicated as causative agents, including oral contraceptives, steroids, minocycline, tetracycline, penicillin, gentamicin, indomethacin, thyroid hormone, lithium carbonate, and hypervitaminosis A. Of interest, prepubertal children with idiopathic intracranial hypertension have a lower incidence of obesity compared to adults, and there is no gender predilection. Similar to adult patients, children are at risk for the development of permanent visual loss.[25]

Other Causes

Headache related to meningeal irritation may be caused by infection (meningitis), inflammation (eg, from a tumor), or hemorrhage (eg, from vascular malformation or malignant hypertension). In addition, sinus disease is often associated with headache.[26]

Differential Diagnosis

The differential diagnosis of headaches is broad. Headaches may range from bothersome nuisances to life-threatening problems. See **Table 6** for an expanded list of primary and secondary headaches.

Table 6. Differential Diagnosis For Pediatric Headache

Primary Headache

- Migraine headache
- Tension-type headache
- Cluster headache

Secondary Headache, Serious Causes

- Alcohol intoxication
- Aneurysm
- Brain abscess
- Carbon monoxide poisoning
- Cerebrovascular abnormality
- Cerebral contusion
- Dental infection
- Drug toxicity
- Encephalitis
- Glaucoma
- Glioma
- Hydrocephalus
- Hypoglycemia
- Infection (bacterial, viral, fungal, mycobacterial)
- Malignant hypertension
- Meningioma
- Meningitis
- Metastasis
- Neuroblastoma
- Osteomyelitis
- Sickle cell disease
- Sinus tumor
- Subarachnoid hemorrhage
- Subdural hematoma
- Substance abuse
- Temporal arteritis
- Trauma
- Vasospasm

Secondary Headache, Non-Life Threatening Causes

- Allergies
- Caffeine toxicity or withdrawal
- Conjunctivitis
- Contusion
- Dental infection
- Extraocular muscle strain
- Glaucoma
- Otitis
- Poor nutrition
- Postlumbar puncture headache
- Sinusitis
- Temporomandibular joint dysfunction
- Vitamin A toxicity

Emergency Department Evaluation

History

When evaluating a child presenting with headache, a thorough history is paramount in determining an accurate diagnosis that will ultimately guide appropriate therapy. Although the majority of headaches are not life threatening, rapid recognition of secondary headache emergencies is vital. A complete history should include a clear description of the headache, mode of onset, duration, severity, and associated symptoms, as well as medication history and family history. Since young children are often unable to articulate specifics, parent or caregiver observations are important, including descriptions of any behavior changes (refusal to participate in age-appropriate play, preference for the dark, etc.).

The following symptoms and signs warrant consideration of further investigation for secondary headache:[27,28]

- First or worst headache ever; sudden ("thunderclap") onset
- Increasing severity or frequency
- Change in headache character
- Awakening from sleep because of headache
- Occurring exclusively in the morning or late at night
- Associated with severe vomiting, particularly in early morning
- Headache associated with straining (cough, urination, defecation)
- Poor response to therapy
- Abrupt alteration in mental status
- Papilledema
- Focal neurologic deficit
- High-risk populations (patients with sickle cell disease, immune deficiency, malignancy, coagulopathy, pregnancy, neurocutaneous syndromes, congenital heart disease, or recent head trauma)

Fortunately, the majority of patients with serious underlying neuropathology with acute headache have recognizable signs and symptoms. Older children may be able to characterize the quality and severity of the headache, which may prove helpful in identifying a specific diagnosis. For example, unilaterality suggests migraine headache, although this may be an unreliable indicator in children aged < 10 years who often report bilateral pain. Also, unilateral headaches may occur with focal infections, such as sinusitis or dental abscess. Furthermore, the location may provide clues to a specific diagnosis, especially in the case of cluster headaches, which are typically associated with retro-orbital or temporal pain.

In addition to providing an alternative classification scheme, certain temporal patterns are associated with specific diagnoses. Acute-onset

headaches that are unlike previous episodes are usually indicative of a secondary headache, and they are often caused by infection or trauma. Acute recurrent headaches, on the other hand, are more suggestive of primary headaches (eg, migraines or tension-type headaches). Chronic progressive headaches that occur several times per week and worsen in severity and frequency over time are concerning for more ominous structural pathology, including neoplasms, abscesses, or hemorrhages. Chronic nonprogressive headaches that have not changed in quality or character are usually attributable to tension-type headaches, but migraines may also be responsible. Unlike acute recurrent headaches, these headaches persist for years and are usually associated with psychological factors.

Disease-Specific History Findings

Migraine Headache

Clinicians should suspect migraine headache in any child who presents with recurrent episodes of incapacitating headaches. Children may have some type of premonitory symptoms before the onset of headache, including irritability and fatigue.[29] Common differences between pediatric and adult presentation of migraine include lack of throbbing, absence of lateralization, and shorter duration of the attack.[30,31] For a list of common migraine triggers, see **Table 7**.

An aura is an acute-onset, transient neurologic dysfunction that may precede a migraine. It typically lasts a few minutes and may present in a variety of forms:

- Visual symptoms: brief changes in visual acuity, flashes of light, straight or broken lines, colors, illusions of shape, and hallucinations
- Sensory symptoms: paresthesias or altered body impression
- Motor symptoms: weakness or paralysis
- Verbal disturbance: dysarthria or aphasia
- Cognitive deficits: confusion or amnesia

Table 7. Factors Known To Precipitate Migraine Headaches

- Stress/anxiety
- Menstruation
- Oral contraceptives
- Physical exertion/fatigue
- Lack of sleep
- Sunlight or screen glare
- Hunger
- Foods/beverages with nitrates, glutamate, caffeine (chocolate, coffee), tyramine (cheese, nuts), salt, monosodium glutamate (MSG)
- Reading/refractive error
- Cold foods
- High altitude
- Seizures
- Traumatic injury
- Infection

The diagnosis of migraine (with or without aura) has been further clarified by the IHS; see **Tables 1 and 2** (pages 3-4) for diagnostic criteria. Migraine is more likely if patients report preceding motor, sensory, or vertiginous symptoms before the onset of throbbing unilateral pain with associated nausea, vomiting, or abdominal pain. Typically, patients report a family history of migraine and find relief with sleep.

Tension-Type Headache

Tension-type headaches are common in children. Distinguishing tension-type headaches from migraine headache may be difficult. In general, tension-type headaches tend to be milder and patients are less likely to seek medical attention. Tension-type headaches typically occur during times of obvious stress, with reports of continuous pain that involves the neck and occiput. No nausea, vomiting, or abdominal pain occurs, and a family history of migraine is less likely. In a subgroup of patients with tension-type headaches, some patients have obvious symptoms of depression (eg, depressed mood, feelings of worthlessness, anhedonia, or anorexia). In this subgroup, headache relief typically follows when the depression is treated. For more specific diagnostic criteria, please see **Table 3,** page 5.

Cluster Headache

In cluster headaches, the headaches occur in groups or clusters, localizing to 1 side of the head. Patients also have clear nasal discharge, congestion, and watery red eye on the ipsilateral side of the head as the headache. Cluster headaches often awaken a patient from sleep, and among children, most often occur in adolescents. For more specific diagnostic criteria, see **Table 4,** page 5.

Sinus Headache

Recurrent headache, though most often caused by migraine and tension-type headaches, is attributable to sinus headache in approximately 1% of cases.[32] Among this small subset, patients complain of a throbbing headache that is worse in the morning or that occurs at the same time each day but may vary with changes in head position (eg, head in the dependent position). With ethmoid disease, pain may be referred to behind the ipsilateral eye; with frontal sinusitis, pain may occur just above the inner canthi of both eyes. Acute bacterial sinusitis may present with persistent symptoms of nasal congestion and cough lasting more than 10 days without clinical improvement. Patients often experience headache, abrupt onset of severe symptoms of high fever (> 39°C), purulent nasal discharge, and facial pain (not present in small children due to the small size of the maxillary sinuses) lasting for 3 to 4 consecutive days. They may also report an antecedent viral upper respiratory infection 5 to 6 days prior to onset.[33]

Head Trauma/Posttraumatic Headache

Posttrauma headaches frequently follow closed-head trauma; headache is a key feature of postconcussive syndrome. Acutely, the patient may complain of headache shortly after the injury, which may worsen and be accompanied by vomiting, lethargy, or seizures (although these may be the earliest symptoms of an intracranial hemorrhage). In chronic cases, headache, dizziness, and personality changes may be present for months after the initial injury. Trauma may also serve as a trigger for migraine headaches.[34]

Intracranial Masses

Patients with intracranial masses may complain of pain localized to the region of the mass, but if a diffuse rise in intracranial pressure exists, the headache may be generalized. Some distinguishing historical features of intracranial masses include severe occipital headache or headache that is exacerbated by sneezing, coughing, any Valsalva maneuver, or change in head position. Pain awakens the patient from sleep or is worse in the morning. Nonetheless, morning headaches – once thought to be a hallmark of raised intracranial pressure – may also occur from etiologies other than intracranial masses, including migraines, cluster headaches, sinusitis, or idiopathic intracranial hypertension. Projectile vomiting without nausea or focal seizures may also occur.

Idiopathic Intracranial Hypertension (Pseudotumor Cerebri)

Idiopathic intracranial hypertension produces headaches similar to headaches in conditions with raised intracranial pressure. Patients typically report vomiting and vision problems such as double vision, blurred vision, or transient visual loss.

Malignant Hypertension

Malignant hypertension, which presents as headache in the setting of hypertension (blood pressures exceeding the 95th percentile for age-based and height-based norms) may be signs of end-organ damage.

Meningitis

Meningeal irritation due to inflammation, infection, or hemorrhage results in the acute onset of diffuse severe headache. Neck pain or stiffness (particularly in bacterial meningitis) and alteration in consciousness may be present. Of note, meningeal signs may not be apparent (or may be delayed) in viral meningitis.

Epilepsy

Children with a prior history of epilepsy may have a generalized or focal headache after a seizure. Headaches may also accompany the aura prior to a seizure.

Medication Overuse Headache

Chronic use of all medications used to treat headaches (such as analgesics or vasoconstrictors) can result in medication overuse headache. It is defined as the development of a different type of headache or worsening of a migraine or tension-type headache, resulting in chronic daily headaches. It develops after use of medications such as analgesics or triptans on > 10 days/mo or after use of over-the-counter analgesics for > 15 days/mo for 3 months' duration.

Physical Examination

A detailed physical examination, including a complete neurologic examination, will facilitate an accurate diagnosis and help to exclude worrisome causes of headache. In most patients with primary headache disorders, the general physical and neurological examinations are normal.[35] Abnormal vital signs (especially the presence of fever, elevated blood pressure, or bradycardia) raise suspicion of worrisome pathology (eg, increased intracranial pressure). Careful evaluation of the skin for rashes or cutaneous lesions (eg, petechiae, purpura for meningococcemia, ash-leaf spots for tuberous sclerosis, café-au-lait spots for neurofibromatosis) may also suggest a specific etiology of secondary headaches.

A thorough neurologic examination should be performed to assess the level of consciousness and to evaluate cranial nerve function, tone, reflexes, strength, or sensation. Evaluate the neck for nuchal rigidity and the head for hematomas or other signs of trauma. Perform funduscopic examination, looking for papilledema or signs of intraocular or retinal hemorrhage (seen mostly in nonaccidental trauma), which may also suggest increased intracranial pressure or trauma.

Certain disease entities have specific physical examination findings to consider. For most primary headaches, the physical examination will be completely normal; however, some children with complicated migraines may have focal neurologic abnormalities such as weakness, sensory changes, or ataxia. With tension-type headache, patients may report pain on palpation of the posterior neck muscles.[36] Among patients with cluster headaches, ipsilateral lacrimation, nasal congestion, rhinorrhea, facial sweating, miosis, ptosis, eyelid edema, or conjunctival injection may be notable.[23]

Among secondary causes, the physical examination findings vary widely. For example, sinus headache sufferers' physical findings include pale, edematous nasal mucosa, boggy turbinates, clear or yellow nasal discharge, pain on palpation of frontal or maxillary sinuses, and failure of the affected sinuses to transilluminate. In acute closed-head injuries, the child may have evidence of skull fracture, altered level of consciousness, focal neurologic deficits, abnormalities in cranial nerve function (III, VI),

or motor deficits. In chronic injuries, the physical examination findings are often normal. Patients with headaches due to an intracranial mass often have focal neurologic abnormalities, especially if they have had headaches for several months; physical examination abnormalities include signs of intracranial hypertension such as papilledema, cranial nerve VI palsy, ataxia, spasticity of the lower extremities, and indications of brain dysfunction regarding language, motor control, or vision (depending on the location of the lesion). Early in the course of the mass lesion, the physical examination findings may be normal. Children with intracranial abscesses may have alteration of the level of consciousness only during the acute presentation. Children with seizures due to metabolic or abnormal brain architecture may have baseline neurologic deficits (eg, hypertonia, hemiparesis), but those with uncomplicated idiopathic epilepsy may have a normal physical examination. In cases of meningeal irritation, fever (meningitis) or hypertension (malignant hypertension) may be present as well as altered consciousness, nuchal rigidity, or hemorrhage of the fundus (suggesting subarachnoid hemorrhage secondary to hypertension). Among patients with depression, anxiety, or psychological stress, psychological examination may demonstrate diminished activities of daily living and pubertal emotional fluctuations.

Diagnostic Studies

Brain Imaging

In general, imaging in primary headache patients is not useful for diagnosis, as only 1% yield significant findings.[37,38] This is not surprising, since central nervous system neoplasms, for example, are uncommon in children aged < 15 years, with an annual incidence of only 3 per 100,000 (0.003%).[39] Furthermore, headaches associated with space-occupying lesions typically have associated focal deficits. In 2 separate retrospective studies of 200 and 72 patients with brain tumor-associated headaches, abnormal neurologic deficits were present in 88% and 94%, respectively.[40,41]

The AHRQ assembled an expert committee in 2012 with the American College of Radiology (ACR) to update its guideline on imaging, entitled ACR Appropriateness Criteria® Headache—Child.[42] This comprehensive review conceded that discerning primary from secondary headaches is challenging, especially considering that the research, to date, is limited to retrospective case series and prospective studies from selective populations of headache subsets. Therefore, the AHRQ recommends:

Imaging Recommendations For Primary Headaches:

- Imaging is not indicated for:
  - Typical migraine with or without aura
  - Common migraine of > 6 months duration with a positive family history
  - Nonprogressive migraine headache
- MRI, when readily available, is recommended to rule out structural lesions in certain primary headaches, including:
  - Migraines with neurologic deficit
  - Ophthalmologic migraine with unilateral ptosis or complete third-nerve palsy
  - Basilar artery migraine syndrome
  - Acute confusional migraine syndrome that persists
  - Progressive chronic headache
  - Hemiplegic migraine
  - Seizures and postictal headache

Imaging Recommendations For Secondary Headaches:

- Imaging is recommended
- MRI is recommended for:
  - Neurologic signs or symptoms of increased intracranial pressure
  - Intracranial hemorrhage (along with magnetic resonance [MR] angiography)
  - Meningitis
  - Encephalitis
  - Brain abscess (MRI is the study of choice)
- CT of the head without intravenous contrast is recommended for:
  - Sudden severe headaches (thunderclap headaches)
  - Exclusion of impending herniation prior to lumbar puncture
  - Subdural empyema or other intracerebral complications of sinusitis or mastoiditis
  - Trauma
  - Cases where MRI is not available
- CT angiography or conventional angiography is recommended for:
  - Subarachnoid hemorrhage
  - Infarction
  - Arterial dissection
  - Aneurysm
- MR angiography is less sensitive, but it may prove an adequate substitute for CT angiography
- CT venography or MR venography is recommended for venous sinus thrombosis

While MRI is a preferred imaging study for ruling out most secondary headache etiologies and sparing young children ionizing radiation exposure, it is often not readily available within the ED. In addition, children commonly require sedation to tolerate the duration of an MRI study. Therefore, the risks of anesthesia and delayed imaging should not be ignored. A quick or fast brain MRI sequence is emerging as an

available alternative for the evaluation of hydrocephalus, since the 4- to 5-minute scan time eliminates the need for sedation. However, these scans are limited in the detection of subtle masses, hemorrhages and abnormalities. To date, there are no formal recommendations regarding its use.[43]

Conicella et al identified clinical features that are useful to recognize life-threatening intracranial conditions for pediatric patients presenting with a chief complaint of headache in the ED.[1]

- Age: preschool*
- Onset of headache: < 2 months
- Pain location: unable to describe or occipital region*
- Pain quality: unable to describe or constrictive*
- Pain intensity: very intense*
- Associated neurological signs: focal neurological deficits, papilledema, ataxia, altered mental status

*Statistically significant associations ($P < .05$)

Laboratory Studies

There is little evidence to suggest a specific panel of laboratory diagnostic tests for the evaluation of headache in the ED. Laboratory studies are not helpful in primary headaches and should be directed by presenting symptoms and signs of suggested secondary headaches. Examples include renal function testing in malignant hypertension and carboxyhemoglobin levels in suspected carbon monoxide poisoning. Outpatient genetic testing may be of some use in a small subset of patients for evaluation of familial hemiplegic migraine.[44]

Lumbar Puncture

A lumbar puncture is not routinely indicated in the evaluation of a primary headache; however, it can offer important clinical information in the evaluation of a secondary headache, including cell count, protein level, glucose level, opening pressure, and routine samples for culture and Gram stain. Therefore, lumbar puncture is indicated for the evaluation of suspected meningitis, headache in the immunocompromised patient, low pressure headaches, idiopathic intracranial hypertension, and subarachnoid hemorrhage.[4]

Lumbar puncture has long been considered the most sensitive test in the diagnosis of subarachnoid hemorrhage;[45] however, imaging technology has advanced, and a review article of the adult literature by McCormack and Hutson supports imaging alone with CT followed by CT angiography for the evaluation of subarachnoid hemorrhage. This review states the pretest risk of missing an aneurysmal subarachnoid hemorrhage to be < 1%;[46] however, these studies have not been replicated among children.

Electroencephalography recordings are not routinely helpful in the evaluation of pediatric headaches.[47,48] Electroencephalography abnormalities have been detected in certain migraine and tension-type headache populations,[49] as well as headache associated with an underlying seizure disorder,[50] yet it is likely of little clinical utility in the ED.

Likewise, electromyelography recordings are of little diagnostic utility for headache in the pediatric ED; however, recent evidence supports its use during biofeedback therapy for outpatient complementary management of tension-type headache.[51]

Treatment

General Principles

There are many treatment options available for managing pediatric headaches in the ED, and interprovider practice varies widely;[52] however, some principles apply to pain and headache in general. For instance, addressing triggers and somatic and psychiatric comorbidities as well as setting goals and expectations will improve therapeutic success. Decreasing stimulation within the ED visit and educating patients about their illness with age-appropriate terminology may serve to reduce stress, in part, by disrupting the vicious cycle. Although it may be of little use during the acute phase, encouraging patients who provide vague or limited historical details to maintain a headache diary will prove useful upon follow-up or in the event of return to the ED or to their primary provider. It should include headache frequency, duration, associated symptoms, and use of medications. Advocate for stress reduction, encourage regular routines (with balanced meals, sufficient fluid intake, physical exercise, and sleep), and discuss coping strategies.

In terms of medication use, abortive analgesics should be used as early as possible with appropriate age- and weight-based doses to alleviate pain. Patients should be cautioned about medication overuse upon discharge. Opioids and benzodiazepines serve no role in the management of primary headaches and should be used sparingly, especially in cases of secondary headaches, due to risks for hemodynamic compromise and rebound effects. Where indicated, it is also important to rehydrate patients with intravenous fluids and address nausea with antiemetics.[55] Prochlorperazine and metoclopramide are used because of their additional benefits in treating the underlying migraine. In situations where those medications are contraindicated, ondansetron can be used.

Disease-Specific Pharmacological Interventions

Abortive Pharmacological Interventions For Migraine

Nonsteroidal Anti-Inflammatory Drugs And Acetaminophen

Nonsteroidal anti-inflammatory drugs (NSAIDs) and acetaminophen are effective first-line agents with proven effectiveness in the acute treatment of migraines. Ibuprofen is 1 of only 2 medicines to receive level A recommendation from the American Academy of Neurology in the acute treatment of migraine in children.[53] In a double-blinded, placebo-controlled crossover study of 88 migraineurs between the ages of 4 and 15 by Hamalainen et al, ibuprofen (10 mg/kg orally) was superior, with 68% pain relief at 2 hours in comparison to acetaminophen (15 mg/kg orally) with 54% pain relief at 2 hours.[54] A similar study compared ibuprofen to placebo among 138 children between the ages of 6 and 12 and demonstrated success with 76% pain relief at 2 hours.[55] Brousseau et al further investigated the effects of another nonsteroidal anti-inflammatory pain reliever, ketorolac (0.5 mg/kg intravenously, max 30 mg), in a double-blinded study among 62 children with migraine aged 7 to 18 years. This study demonstrated pain relief in 55.2% of patients at 1 hour and a 93% response rate when combined with prochlorperazine.[56] Of note, acetaminophen and ibuprofen are contraindicated in liver failure and active gastrointestinal bleeding, respectively.

Dopamine Receptor Antagonists

Dopamine receptor antagonists (such as chlorpromazine, prochlorperazine, and metoclopramide) treat pain and nausea, but also have antihistaminic and anticholinergic effects. Prochlorperazine has the best supporting evidence for acute treatment of migraine in children in an ED setting. A randomized double-blinded controlled trial in a pediatric ED setting demonstrated 84.8% pain relief at 1 hour among 62 children, compared with ketorolac, which only had 55.2% success.[56] A 2012 Canadian prospective cohort study by Trottier et al further attests to the acute relief provided by prochlorperazine at 1 hour; however, nearly two-thirds of the 79 participants in this study experienced relapse within 1 week after discharge.[57] A 2001 retrospective review of 20 pediatric ED patients by Kabbouche et al demonstrated relief of intractable migraines with intravenous prochlorperazine, with 90% of patients reporting improvement after 1 hour and 95% reporting improvement after 3 hours; however, complete pain relief was reported among 50% and 60%, respectively.[58]

Chlorpromazine and metoclopramide have similar activity, but further studies are needed to compare the 2 agents. All dopamine receptor antagonists can have side effects that include dystonia and akathisia, while prochlorperazine and chlorpromazine are specifically associated with a risk for QT prolongation.

Of note, recently Leung et al compared reduction in headache pain scores (as the primary outcome), length of stay in the ED, hospital admission rate, and ED readmission rate within 48 hours between patients with migraine who received standardized combination therapy with patients receiving various migraine therapies. Standardized treatment consisted of intravenous combination therapy involving normal saline fluid bolus, ketorolac, prochlorperazine and diphenhydramine. Metoclopramide was substituted during prochlorperazine shortages. Of interest, patients receiving standardized treatment had a significant decrease in pain score, length of stay, and hospital admission rate without changes in the ED return rates.[59]

Triptans

Several triptans, including almotriptan (6.25-12.5 mg orally), rizatriptan (5-10 mg orally), zolmitriptan (2.5-5 mg orally), and sumatriptan (5-20 mg intranasally or 6 mg subcutaneously), have demonstrated success in double-blind placebo controlled studies in children. Sumatriptan nasal spray is the other medication that has level A evidence for use in migraine in children. Linder et al confirmed 66.7% to 72.9% pain relief at 2 hours in 866 patients between ages 12 and 17 who were given almotriptan; it is currently approved by the United States Food and Drug Administration (FDA) for that age group for acute migraine treatment.[60] Intranasal sumatriptan has shown successful pain relief at 2 hours in 3 additional studies: Ueberall and Wenzel with 86% in 14 patients, ages 6 to 9 years; Ahonen et al with 66% in 83 patients, ages 8 to 17 years; and Winner et al with 64% in an impressive 653 patients aged 12 to 17 years.[61-63]

Rizatriptan has also demonstrated efficacy in children. Ahonen et al showed 73% to 74% relief with rizatriptan at 2 hours in 96 patients between ages 6 and 17 years, the same age group for which it is FDA approved.[64] Finally, zolmitriptan appears effective in 2 studies, with 58.1% relief for 171 patients between ages 12 to 17 years at 1 hour (intranasally)[65] and 62% to 64% relief at 2 hours for 32 patients between the ages of 6 to 18 years (orally).[66] Triptans are contraindicated in patients with a history of stroke, cardiovascular disease, uncontrolled hypertension, and hemiplegic migraine; in pregnancy, it is a category C drug.

Dihydroergotamine

Dihydroergotamine (DHE) is an ergot alkaloid that functions as an effective intravenous abortive / vasoconstrictor. It has proven successful in managing status migrainosus in children in the inpatient setting; however, no randomized controlled trials are currently available. One retrospective chart review of 32 patients demonstrated 74.4% of patients were pain free after hospitalization that included intravenous

DHE (0.5-1 mg every 8 h), along with intravenous rehydration and antiemetic pretreatment.[67] Oral DHE has poor bioavailability and is no more effective than placebo.[68] Like the triptans, DHE is contraindicated in patients with uncontrolled hypertension, pregnancy, cardiovascular disease, and stroke.

Prophylactic Pharmacological Interventions For Migraine

There are limited data to suggest that migraine prophylaxis is of use in treating childhood migraine. Prophylaxis may be indicated if lifestyle modification and nonpharmacologic prophylaxis prove ineffective, or if attacks occur with sufficient frequency (usually 3-4/mo) and severity, leading to adverse events such as frequently missed school; however, prophylaxis is typically managed on an outpatient basis by neurology specialists. Medications (such as propranolol, amitriptyline, cyproheptadine, sodium valproate, and topiramate), supplementation (including vitamin B12, coenzyme Q10, riboflavin), and nonpharmacologic techniques (such as relaxation, biofeedback, and cognitive behavioral therapy) have been used, but the data remain inconclusive.

While hypomagnesemia has been implicated in the pathogenesis of migraines, the available data regarding the efficacy of magnesium replacement for migraine prevention remain mixed. Of the 4 available randomized controlled trials, 2 studies showed successful prevention, while 2 studies showed no significance.[69-72] Magnesium has been shown to treat migraine with aura, though it only mitigates symptoms of photophobia and phonophobia across all migraineurs.[73] Despite this inconclusive evidence, a paucity of pediatric studies, and known possible side effects including diarrhea, hypotension, and gastrointestinal discomfort, magnesium supplementation is typically well tolerated, safe to use in pregnancy, and may provide migraine prevention. However, it cannot be used in conjunction with metoclopramide due to cerebral vasodilatory effects.

Valproate has demonstrated pain relief among adults but, to date, little evidence supports its use among children. Mathew et al showed improvement with valproate among 73% of adults within 30 minutes,[74] while Edwards et al demonstrated relief among 60% of adults (equivalent to DHE 1 mg intravenously plus metoclopramide 10 mg intravenously).[75] In contrast, a prospective randomized double-blind trial by Tanen et al pointed out that valproate was significantly inferior to prochlorperazine for pain relief among adult patients.[76]

There are no pediatric data supporting corticosteroid use in the management of migraine; however, studies among adults suggest a role for a short course of corticosteroids in prevention of headache recurrence for headaches that have lasted > 72 hours.[77,78]

Tension-Type Headache Treatment

Most episodic tension-type headaches are mild and self limited, but when they progress to chronic tension-type headaches, patients may seek emergency medical care. There are limited research data exploring the most effective treatment for tension-type headaches; however, most migraine medications can provide relief for tension-type headaches as well. First-line therapies include acetaminophen and NSAIDs. Adult literatures suggests a role for amitriptyline (a tricyclic antidepressant) in tension-type headache prevention. Currently, there are no placebo-controlled studies among children investigating tension-type headache prophylaxis.[79]

Cluster Headache Treatment

Sumatriptan and inhalation of 100% oxygen, via high flow, at 15 L/min or via a demand-valve oxygen delivery system can provide acute pain relief for cluster headaches. A 2008 Cochrane review supported the use of normobaric oxygen therapy for cluster headaches, but it was inconclusive with regard to hyperbaric oxygen therapy.[80] It is theorized that oxygen administered at higher pressures produces vasoconstriction (while preserving tissue oxygenation), provides serotonergic agonism, and plays a role in immunomodulation via substance P. A 2010 Cochrane review demonstrated the benefits of triptans over placebo in managing cluster headaches.[81] Zolmitriptan (orally or intranasally) and sumatriptan (subcutaneously or intranasally) improved headache relief and pain-free responses when compared to placebo. One study comparing intranasal zolmitriptan 10 mg to subcutaneous sumatriptan 6 mg showed that sumatriptan was superior in providing pain relief.[81] A small retrospective study of 11 children with cluster headaches showed that steroids were useful in preventing recurrence.[82] Finally, intranasal lidocaine may have some usefulness as an alternative adjunctive therapy for treatment of cluster headache among adults, though its true efficacy remains debatable.[83]

Secondary Headache Treatment

For most cases of secondary headaches, treatment relies on addressing the primary problem; however, a few specific headache treatments are worthy of mention.

Sinusitis Treatment

The mainstay of treatment includes appropriate antibiotic coverage. NSAIDs and acetaminophen may provide useful analgesia, but they may lead to medication-overuse headaches if used for > 10 days per month. In addition, high-dose intranasal corticosteroids may offer an additional therapeutic benefit in the treatment of headache associated with sinusitis.[84]

Medication Overuse Headache Treatment

Medication-overuse headache, a syndrome characterized by the chronic conversion of episodic headaches, results from the frequent use of medications that were meant to treat migraines. Opioids, triptans, acetaminophen, and NSAIDs are some of the most common offenders, ironically leading to worsening frequency and intensity of migraine headaches. Several mechanisms have been suggested, though the leading theory hypothesizes that neuronal remodeling leads to increased stimulation by migraine triggers with features similar to behavioral hypersensitivity observed in cases of cutaneous allodynia.[85] Medication-overuse headache has been recognized by the IHS as a syndrome in which headaches occur on 15 or more days per month, during which time the pain reliever is overused regularly for 3 or more months.[86] Anxiety, depression, and substance abuse disorders are frequent comorbid conditions.

Primary treatment for medication-overuse headache includes removal of the offending agent, typically the overused acute analgesic. A 2013 prospective multicenter randomized, double-blinded placebo-controlled study of 96 patients by Rabe et al demonstrated that a course of prednisone (100 mg/day for 5 days) may reduce the need for additional analgesics for medication-overuse headache sufferers during abstinence.[87] Notably, patients with medication-overuse headache attributed to narcotic analgesics had a longer, more challenging recovery than their triptan counterparts.[88]

Intracranial Space-Occupying Lesion Treatment

Intracranial hemorrhage or mass can cause headache. Given the life-threatening nature of these etiologies, particular attention should be paid to the ABCs (airway, breathing, and circulation), with the goal of appropriate oxygenation and ventilation. Surgery is typically indicated to evacuate the hemorrhage or remove the mass.

Idiopathic Intracranial Hypertension Treatment

Reducing cerebrospinal fluid volume by lumbar puncture has been documented to be beneficial for both diagnosis and treatment of idiopathic intracranial hypertension. Theoretically, decreasing production of cerebrospinal fluid by using carbonic anhydrase inhibitors may relieve the pressure-like pain associated with idiopathic intracranial hypertension; however, well-done studies conclusively documenting the efficacy of carbonic anhydrase inhibitors are lacking.[89]

Malignant Hypertension Treatment

Blood pressure control with antihypertensives may be indicated. Expert consultation with pediatric nephrology should be considered.

Meningeal Inflammation Treatment

The treatment goal for meningeal inflammation is treatment of the underlying cause, such as infection (antibiotics), or subarachnoid hemorrhage (surgical evacuation of intracranial hemorrhage); nimodipine (calcium channel blocker) can be used to reduce vasospasm, but its safety and effectiveness have not been established in children.

Postlumbar Puncture Headache Treatment

Though uncommon in younger children, postlumbar puncture headaches may occur in older children and adolescents. A 2011 Cochrane review of postdural puncture headache demonstrated that caffeine, gabapentin, theophylline, and hydrocortisone provide pain relief that surpasses placebo. Sumatriptan and adrenocorticotropic hormone showed no effect.[90]

Controversies And Cutting Edge

Intranasal Ketamine

A 2013 randomized controlled double-blind trial by Afridi et al offers evidence in support of intranasal ketamine, a novel therapy for migraineurs with aura. To date, there is no abortive agent that has demonstrated relief of the aura phase of migraine. Prior animal studies looking at the effect of ketamine on migraine have established its role in blocking cortical spreading depression. The successful relief of aura severity in the 18 subjects who received 25 mg of intranasal ketamine adds further support to this theory. Although this study was conducted exclusively among adults, the ease of administration and common use of ketamine in the pediatric emergency setting make this an attractive avenue for future study among children.[91]

Low-Dose Propofol

A small 2012 retrospective study by Sheridan et al suggested that propofol may be effective for the abortive treatment of intractable migraine headache in the pediatric ED.[92] This is the first study among children that supports the use of propofol, which was first described among adults by Krusz et al in 2000.[93] In the case-control study by Sheridan et al, 7 pediatric migraineurs who were given subanesthetic doses of propofol (average 0.56 mg/kg per bolus, ranging 10-50 mg per bolus, up to 3 boluses) were compared to matched controls who had received NSAIDs, diphenhydramine, and prochlorperazine for abortive therapy. Patients who received propofol reported a significant reduction in pain scores (80.1% vs 61.1%). The study cites a decreased length of stay among patients receiving propofol (122 min vs 203 min), but this was not statistically significant. No patients experienced adverse side effects such as apnea, hypoventilation, or hypoxia, despite monitoring by providers trained in moderate and deep

sedation; however, 2 patients who received propofol still required admission for further therapy. Although this study offers encouraging support for propofol as a safe alternative abortive agent for treating status migrainosus among children, larger prospective studies are needed before conclusions can be drawn.[92]

Disposition

Indications For Referral

Complicated headache patterns that fail to respond to treatment may require a pediatric neurology consultation. Patients suffering from chronic recurrent headaches should be referred to a pediatric neurologist for further management. Psychiatric support may also prove useful in treating any comorbid mood disturbance or anxiety. Secondary headaches often require specialist management. Consultation with a surgeon or neurosurgeon is appropriate for headache caused by mass lesions, intracranial hemorrhage, or abscess.

Criteria For Admission

Inpatient management is indicated for headaches that require continuous intravenous medications or for rehydration, chronic daily headaches that have failed outpatient treatment, status migrainosus, analgesic rebound headache, or headaches of unclear etiology that are severe, worsening, or associated with an abnormal neurologic examination.

Complications

Children with frequent or severe headaches are more likely to have emotional or behavioral problems, including conduct problems and inattention-hyperactivity. They are more likely to struggle with family relationships, friendships, classroom learning, and leisure activities due to their chronic headaches. In general, they report lower satisfaction with their health or life, in general, and they experience greater stress, fatigue, depression, and somatic symptoms, similar to patients with other chronic illnesses.[94] Headache can lead to psychological impairment and decreased quality of life, especially for persons who experience chronic migraine. Migraineurs are even more impaired than those who suffer from tension-type headache, in terms of medication use, school nurse visits, and school absences. Headaches cause more than 3,000,000 bedridden days per month in the United States and, per migraineur, 2 missed school days per month.

Prognosis

Approximately one-third of children with recurrent headaches will experience remission. An initial diagnosis of migraine portends a more troublesome course, as those with tension-type headache have higher rates of remission than those with migraine. A long-term cohort study of children with headache demonstrated that at 20-year follow up, among 77 respondents, only 27% were headache free, 33% had tension-type headache, 17% had migraines, and 23% had migraines and tension-type headaches. Eight percent of these patients described their headaches as moderate to severe, although 66% of the patients considered their current state as an improvement.[95] Risk factors that predict persistence include female gender, maternal history of headache, and a psychiatric diagnosis.[96] A multidisciplinary approach has proven to be effective for children and adolescents with migraine.[97]

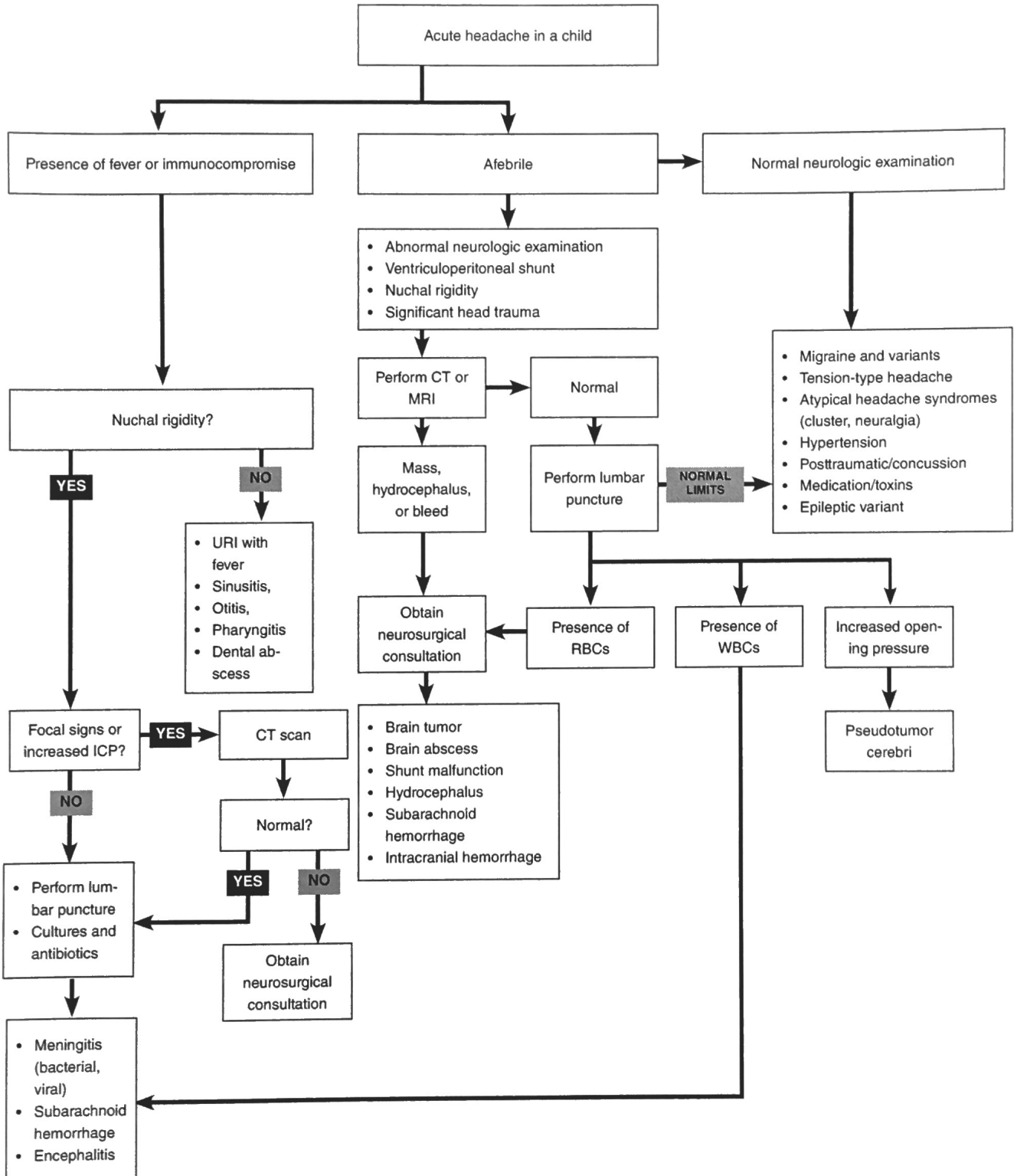
Patient Education

For patient information, including support resources and handouts about pediatric headaches, patients should consult the National Headache Foundation (www.headaches.org) and American Headache Society Committee for Headache Education (www.achenet.org).

Summary

Headache is a common complaint among pediatric ED patients. While the majority of headaches are due to primary etiologies that include migraine headaches, tension-type headaches, and cluster headaches, secondary headaches must always be considered by all emergency clinicians. Rapid recognition of the signs of increased intracranial pressure can be life-saving. When approaching the pediatric headache patient, the most important step is diagnosis. Familiarity with the diagnostic criteria of primary headaches will help with establishing a cause. Unless secondary headache is suspected, there is limited role for the use of neuroimaging or diagnostic testing. Once a diagnosis is made, treatment can be tailored, implementing the most current evidence-based interventions. Currently, there are few large, prospective, randomized control trials guiding therapy, leaving significant opportunity for further research.

Clinical Pathway For Diagnosis Of Pediatric Headache In The Emergency Department



Abbreviations: CT, computed tomography; ICP, intracranial pressure; RBC, red blood cell; MRI, magnetic resonance imaging; URI, upper respiratory infection; WBC, white blood cell.

For class of evidence definitions, see page 17.

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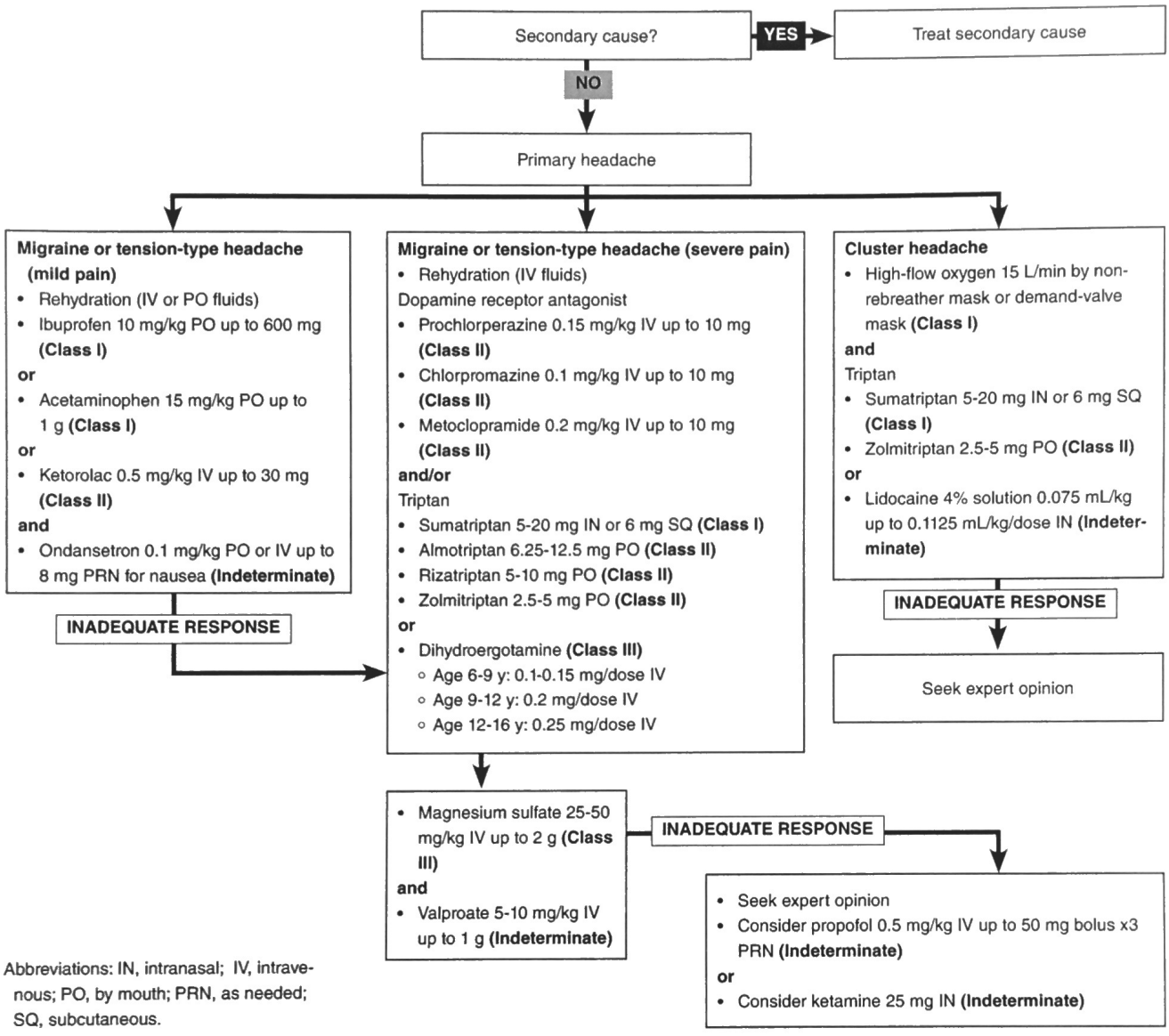
Clinical Pathway For Treatment Of Pediatric Primary Headache



Abbreviations: IN, intranasal; IV, intravenous; PO, by mouth; PRN, as needed; SQ, subcutaneous.

Class Of Evidence Definitions

Each action in the clinical pathway section of *Pediatric Emergency Medicine Practice* receives a score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Non-randomized or retrospective studies: historic, cohort, or case control studies
- Less robust randomized controlled trials
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

Significantly modified from: The Emergency Cardiovascular Care Committees of the American Heart Association and representatives from the resuscitation councils of ILCOR: How to Develop Evidence-Based Guidelines for Emergency Cardiac Care: Quality of Evidence and Classes of Recommendations; also: Anonymous. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Part IX. Ensuring effectiveness of community-wide emergency cardiac care. *JAMA.* 1992;268(16):2289-2295.

This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

Risk Management Pitfalls For Pediatric Headache Management

The greatest medical pitfall in pediatric headache management is failure to make an accurate diagnosis. Effective treatment of headache relies on identification of the underlying cause. In the setting of an abnormal physical examination or certain historical red flags, a headache must be considered a secondary headache until otherwise ruled out.

1. **"I thought the teenager with unilateral facial numbness was having an atypical migraine, so I sent her home with a triptan and told her to follow up with her pediatrician."**
   Careful history-taking and thorough neurological examination can help make the correct diagnosis. A high index of suspicion is needed to avoid missing a secondary headache. Remember that primary headaches are diagnoses of exclusion.

2. **"The patient was really sick and I didn't want to sterilize the cultures, so I made sure to perform the lumbar puncture before giving antibiotics."**
   When faced with a decompensating patient with possible meningitis, do not delay the administration of life-saving antibiotics. Lumbar puncture is meant to aid in diagnosis; if you already know the treatment is needed urgently, do not wait.

3. **"The patient has a history of multiple concussions, so I figured his progressively worsening headache was just part of a posttraumatic headache."**
   Concussions and previous head injuries can be challenging to manage, but it is important to recognize acute on chronic changes or progression of symptoms as possible clues to more ominous pathology such as intracranial hemorrhage or venous thrombosis.

4. **"The patient was only 13, so I didn't bother to check a urine pregnancy test."**
   Among female adolescents who are of childbearing age, eclampsia must be considered until pregnancy has been ruled out. In addition, some migraine medications, such as triptans and DHE, are contraindicated or discouraged in pregnancy. Urine pregnancy tests are inexpensive, readily available in the ED, and generally more reliable than the average teenager.

5. **"The patient has had the same headache for 2 months, so I got a head CT to find out why."**
   Chronic headaches without progression of symptoms or other red flags do not always require emergent head imaging. In fact, an MRI (which can be arranged as an outpatient) may provide a more thorough evaluation and avoid unnecessary exposure to ionizing radiation.

6. **"He said he gets sinus headaches all the time, so I gave him a prescription for amoxicillin and sent him on his way."**
   Sinusitis can cause headache; however, these patients are more likely to suffer from under-recognized primary headaches such as migraines and tension-type headaches. Judicious use of antibiotics is necessary to prevent resistance, and diagnosis-specific medications are important to address the pain.

7. **"The patient was in so much pain, I had to give him additional doses of morphine."**
   Narcotics play little role in the management of headaches and no role in the management of primary headaches. They may provide a quick fix, but this effect is fleeting and is typically followed by rebound headaches that have been recognized as medication overuse headaches.

8. **"He kept saying his headaches bothered him the most in the mornings – I thought he just didn't want to go to school."**
   Early-morning headache is a red flag for an intracranial space-occupying lesion. A thorough history and physical examination should help differentiate this worrisome secondary headache from behavioral misconduct. Beware of drawing such conclusions before life-threatening pathology has been effectively ruled out.

9. **"She had papilledema on examination after a fall from a 3-story window, so I ordered an MRI right away."**
   A good fundoscopic examination should be performed on every patient. Since papilledema may suggest increased intracranial pressure, it is important to remember that timeliness is key. Even if MRI is available, if you have concern for an acute bleed with potential for rapid decompensation, CT would be your imaging modality of choice.

10. **"This was her third visit to the ED with status migrainosus in the last 2 months, so I started her on cyproheptadine to prevent a fourth visit."**
    Evidence for use of migraine prophylaxis in children is poor. If indicated, migraine prophylaxis should be administered by the patient's medical home (primary care provider or neurologist) with a plan in place for good follow-up care. Lack of follow-up when starting chronic medications may lead to medication overuse or hazardous, unchecked medication side effects.

Case Conclusions

You diagnosed the 14-year-old girl with a classic migraine and prescribed ibuprofen 600 mg orally, metoclopramide 10 mg intravenously, and a 20 mL/kg bolus of normal saline. She experienced relief and was ultimately discharged to continue ibuprofen 600 mg every 6 hours as needed for pain. Although she did not yet meet the technical criteria for migraine (since she had not had 5 previous episodes), empiric migraine therapy should not be denied.

The 11-year-old boy failed to cooperate with a full examination to eliminate neurologic deficit, so you ordered a quick-brain MRI, which was unremarkable. You subsequently diagnosed him with a postconcussive headache with migraine features. Because he was unable to tolerate oral medications, you prescribed ketorolac 15 mg and ondansetron 4 mg intravenously, and he experienced some relief. You consulted neurology, and they subsequently started him on amitriptyline 25 mg at bedtime with plans to follow up with neurology as an outpatient.

The 18-year-old male appeared ill, but he remained clinically stable. You ordered prompt blood culture and lumbar puncture before administering empiric antibiotics for bacterial meningitis within an hour of presentation. His cerebrospinal fluid cell count was remarkable for white blood cell count of 580/mm^3 and glucose of 20 mg/dL. He required 2L normal saline in fluid resuscitation, but remained hemodynamically stable. Gram stain was notable for gram-negative Diplococcus and his culture later grew out Neisseria meningitidis. You admitted him for intravenous antibiotic therapy and he was eventually discharged with a peripherally inserted central catheter to finish antibiotic therapy at home.

Time- And Cost-Effective Strategies

- While neuroimaging can largely rule out serious intracranial pathology, it comes at a price. Often, a pediatric patient requires sedation in order to lie still for a radiology study, which confers all the risks and medical expenditures inherent to the anesthetics, including airway implementation and the imaging modality involved. Lifetime radiation exposure cannot be ignored. Remember that when evaluating a child with headache, it is critically important to perform a thorough history and complete physical examination.
- Patients with complex neurologic complaints may require transfer to another facility for a higher level of care, increasing the likelihood of repeated neuroimaging studies if information exchange is not readily available. Interestingly, a 2013 longitudinal study by Bailey et al looking at health information exchange among EDs failed to demonstrate significant cost savings with information exchange; however, implementation reduced the overall use of diagnostic imaging.[98]

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study will be included in bold type following the references, where available. The most informative references cited in this paper, as determined by the author, will be noted by an asterisk (*) next to the number of the reference.

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CME Questions

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1. **An 11-year-old girl with recurrent headaches presents to the ED with a bilateral headache that she describes as "pulsating" for the past 5 hours and associated with nausea. On physical examination, she is afebrile and has photophobia. Her headache would best be classified as:**
   a. Migraine headache
   b. Tension-type headache
   c. Meningitis
   d. Cluster headache

2. **A 12-year-old male is brought into the ED complaining of unilateral, icepick-like stabbing pain emanating from behind his right eye with unilateral tearing. His headache would best be classified as:**
   a. Migraine headache
   b. Tension-type headache
   c. Cluster headache
   d. Posttraumatic headache

3. **A 12-year-old boy presents with a bitemporal headache that comes and goes throughout the day. On physical examination, he has no photophobia or phonophobia. His headache would be classified as:**
   a. Migraine headache
   b. Tension-type headache
   c. Meningitis
   d. Cluster headache

4. **All of the following are considered red flags that would warrant further diagnostic evaluation EXCEPT:**
   a. Nausea
   b. Increasing severity or frequency
   c. Awakening from sleep because of headache
   d. Abrupt alteration in mental status

5. **A mother brings her 13-year-old daughter into the ED because she has had daily headaches of the same quality and severity for the past month, which always respond to acetaminophen or ibuprofen. The next BEST step would:**
   a. Initiate triptan therapy
   b. Obtain noncontrast head computed tomography
   c. Perform a lumbar puncture
   d. Provide reassurance and encourage follow-up with her primary care physician

6. **A 7-year-old boy presents with headache and ataxia. To rule out a space-occupying lesion, which of the following would be the BEST imaging modality?**
   a. Noncontrast head CT
   b. Contrast head CT
   c. Noncontrast head MRI
   d. Contrast head MRI

7. **A 16-year-old girl with a history of migraines presents with a headache. She reports that this feels like every other migraine headache she has experienced in the past. What further workup is indicated in the ED?**
   a. Complete blood count
   b. Lumbar puncture
   c. Basic metabolic panel
   d. All of the above
   e. None of the above

8. **Which of the following medications has the best evidence for the treatment of pediatric migraines?**
   a. NSAIDs
   b. Dopamine-receptor agonists
   c. Opioids
   d. Triptans

9. **A 15-year-old girl presents with a migraine headache. Which of the following treatment options would be INAPPROPRIATE for acute management?**
   a. Sumatriptan nasal spray
   b. Dihydroergotamine intravenously with metoclopramide
   c. Cyproheptadine and propranolol orally
   d. Ibuprofen orally

10. **Frequent use of which of the following medications may lead to a medication overuse headache?**
    a. NSAIDs
    b. Opioids
    c. Triptans
    d. All of the above
    e. None of the above

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