Indications and Imaging Modality of Choice in Pediatric Headache

Asha Sarma, MD[a,*], Tina Young Poussaint, MD[b]

KEYWORDS

• Pediatrics • Headache • Neuroimaging • Magnetic resonance imaging • Decision support

KEY POINTS

- Neuroimaging is generally not indicated for primary pediatric headache (tension, migraine, and cluster headaches).
- Practice parameters, including the 2017 American College of Radiology Appropriateness Criteria, are available to guide neuroimaging in pediatric headache.
- CT and MR imaging are first-line modalities for pediatric headache evaluation. Optimization of CT and MR imaging may decrease ionizing radiation exposure and need for procedural anesthesia.

INTRODUCTION

Pediatric headache is common and is associated with significant morbidity and economic cost.[1] Despite the increasing use of neuroimaging, findings that change clinical or surgical management are unusual in the absence of neurologic abnormalities.[2–5]

A variety of benign and emergent conditions can cause pediatric headache. This review aims to help guide when to image a child with headache and how to select the appropriate examination. Optimization of CT and MR imaging evaluation with tailored, indication-based protocoling is considered in detail, along with approaches to imaging in a range of specific clinical conditions.

EPIDEMIOLOGY

The reported prevalence of pediatric headache ranges from approximately 17% to 91%.[6,7] Prevalence increases with age throughout childhood and adolescence but headaches are still common in young children—for instance, 29% of children under age 5 have reported a headache within the previous year.[6,8] Headaches are more prevalent in boys before puberty. This trend reverses after puberty, with the female-to-male ratio increasing into adulthood.[9]

PEDIATRIC HEADACHE CLASSIFICATION

Generally, uncomplicated primary headache disorders without an underlying cause (eg, migraine, tension, cluster, and daily headaches) do not require neuroimaging.[10] Causes of secondary headache are numerous and varied.

Headaches can also be classified as acute, acute recurrent (episodic), chronic nonprogressive, chronic progressive, or new persistent (**Fig. 1**).[9] Acute recurrent (episodic) and chronic nonprogressive headaches tend to represent benign causes of headache (ie, primary headache disorders), whereas new persistent and chronic progressive headaches are more concerning for significant intracranial pathology and, therefore,

Disclosures: None.
[a] Department of Radiology, Vanderbilt University Medical Center, Monroe Carell Jr. Children's Hospital, 2200 Children's Way, Suite 1421, Nashville, TN 37232-9700, USA; [b] Department of Radiology, Harvard Medical School, Boston Children's Hospital, 300 Longwood Avenue, Boston, MA 02130, USA
* Corresponding author.
E-mail address: asha.sarma@vumc.org

Neuroimag Clin N Am 29 (2019) 271–289
https://doi.org/10.1016/j.nic.2019.01.007

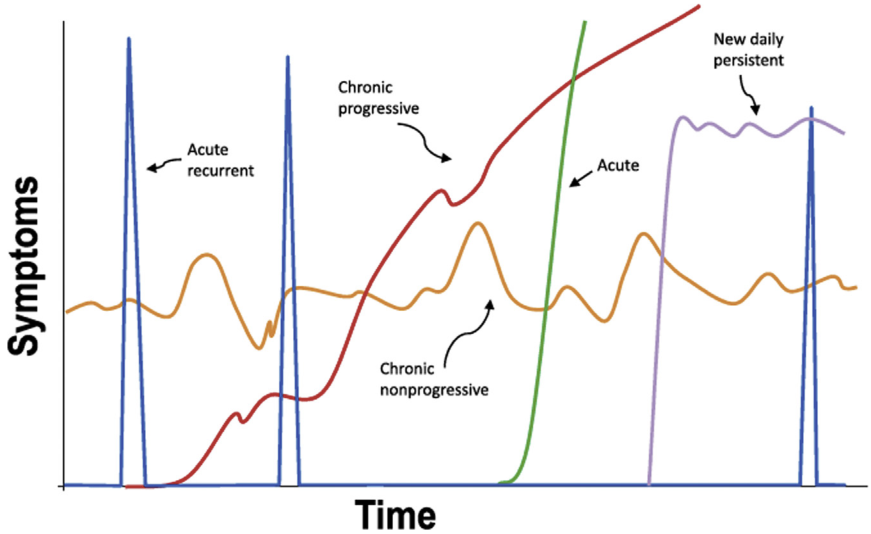

Fig. 1. Patterns of headache. (*From* Blume HK. Pediatric headache: a review. Pediatr Rev 2012;33(12):563; with permission.)

more often warrant neuroimaging.[9,11] Causes of acute single-event headaches range from self-limited viral upper respiratory infections to life-threatening hemorrhage from a vascular malformation—thus, these headaches must be carefully triaged.[9]

CLINICAL CONSIDERATIONS IN PEDIATRIC PATIENTS WITH HEADACHE

Clinical Evaluation of Pediatric Patients with Headache

Important components of the clinical evaluation for pediatric headache are included in **Table 1**.[9,12]

Knowledge of intracranial and head and neck pain–sensitive structures may help in lesion localization (**Table 2**).[13] Migraine-like pain is believed caused by cortical spreading depression, defined as sequential waves of neuronal depolarization and depressed neuronal activity, leading to release of chemicals that irritate trigeminal nerve afferents.[14]

Red Flags in Pediatric Patients with Headache

Research has been focused on determining clinical signs and symptoms predictive of serious intracranial pathology (red flags) in patients with headache.[1] Brain tumors are a common concern (although tumor is uncommon without neurologic signs).[4,15–17]

Dodick[18] established a set of red flags in pediatric patients with headache that fit the memorable mnemonic, SNOOPPPPY (**Box 1**). These red flags have not been rigorously validated in pediatric patients, and there is some disagreement in the literature about whether patients should be imaged on the basis of age at presentation, change in headache type, and occipital headache.[1] In a study of pediatric patients in the emergency setting, preschool age, recent onset of pain, occipital location, inability to describe the quality of pain, and objective neurologic signs predicted serious pathology.[19]

Summary of Current Clinical Guidelines for Neuroimaging Pediatric Patients with Headache

Relevant clinical guidelines for neuroimaging pediatric patients with headache are summarized in **Table 3**.[11,20,21]

IMAGING EVALUATION OF PEDIATRIC PATIENTS WITH HEADACHE

General Considerations

Noncontrast CT and MR imaging are the most commonly used modalities in the initial evaluation of pediatric headache. Clinical evaluation, resource availability (including anesthesia services), and risk-benefit analyses addressing radiation exposure and sedation influence choice of modality.

CT is generally more widely available than MR imaging in the pediatric emergency setting; however, efforts have been made to increase the availability of MR imaging due to concerns about carcinogenesis from ionizing radiation.[22,23] An advantage of CT over MR imaging is more rapid examination speed that reduces the need for sedation. CT is also more useful in clinically

Table 1
Salient components of the clinical evaluation of pediatric patients with headache

Headache Features	Past Medical History	Family History	Drugs and Medications	Social History	Physical and Neurologic Examination Findings	Ancillary Testing
Temporal pattern	Tumor predisposition syndromes	Migraine	Stimulants (eg, for attention-deficit hyperactivity disorder)	Social stressors	Significantly abnormal height and weight	Blood tests
Location	Sinus disease	Familial tumor syndromes	Bronchodilators	Sleep patterns	Abnormal vital signs (eg, fever, hypertension)	CSF opening pressure and analysis
Character	Cerebrovascular disease (eg, moyamoya, sickle cell disease)	Polycystic kidney disease, berry aneurysms	Drugs of abuse, including alcohol		Signs of external bleeding	Electroencephalogram
Triggers and warning signs	Bleeding disorder		Minocycline (can cause intracranial hypertension)		Altered mental status	
Red flag signs			Caffeine		Dysphasia	
					Altered motor or sensory function	
					Cranial nerve palsy	
					Abnormal vision	
					Abnormal gait	
					Papilledema, optic atrophy, intraocular hemorrhage	

Data from Refs.[10–12]

Table 2
Pain-sensitive structures of the brain, head, and neck

Intracranial Pain–sensitive Structures	Head and Neck Pain–sensitive Structures
Dural venous sinuses Anterior and middle meningeal arteries, portions of the internal carotid artery, and arterial branches near the circle of Willis Skull base dura Cranial nerves V, IX, and X Periaqueductal gray matter Sensory nuclei of the thalami	Skin, pericranium, subcutaneous tissues, muscles, extracranial arteries C2 and C3 nerves Eyes, ears, teeth, sinuses Oropharynx Nasal cavity mucosa

Data from Headache & facial pain. In: Aminoff MJ, Greenberg DA, Simon RP, editors. Clinical neurology, 9th edition. New York: McGraw-Hill; 2015.

unstable patients and in those with certain implanted devices (eg, pacemaker and cochlear implant devices).

MR imaging is generally more expensive, less available, and more time consuming than CT. It is the modality of choice, however, for the initial evaluation of most types of pediatric headache (excluding sudden-onset thunderclap headaches, which usually are first evaluated with CT) due to its superior tissue contrast resolution, which enables more optimal tissue characterization and detection of small or subtle lesions. Furthermore, unlike CT, MR imaging uses electromagnetic radiation, which confers no carcinogenic risk.

Optimization of use of gadolinium-based contrast agents is essential for MR imaging. Recent studies have shown that gadolinium deposition occurs in the brain, bone, and other organs in children with normal renal function.[24–29] The physiologic and clinical significance of brain gadolinium deposition (if any) has not yet been determined; however, judicious use of gadolinium-based contrast agents is recommended.

For MR imaging, procedural sedation to minimize patient motion is often necessary to achieve diagnostic quality imaging. Although short (<3 hour), single sessions of sedation in otherwise healthy infants or children likely has no lasting neurodevelopmental consequences, studies of animals, including nonhuman primates, have raised concern about the safety of anesthetic agents in children.[30] Awareness of this issue increased after the US Food and Drug Administration issued warnings that anesthesia administered to children under age 3 for sessions longer than 3 hours or on a repeated basis might affect brain development.[31]

At Boston Children's Hospital, strategies for minimizing the need for anesthesia in children (**Box 2**) have substantially decreased the proportion of cases requiring anesthesia. Importantly, it may be unsafe to defer indicated neuroimaging studies in many instances, and sedation and general anesthesia remain vital tools.[30]

Box 1
SNOOPPPPY mnemonic for red flags in pediatric headache

S: systemic symptoms or illness

N: neurologic signs or symptoms

O: onset recent or sudden (ie, thunderclap headache)

O: occipital localization of pain

P: pattern—precipitated by Valsalva maneuver

P: pattern—positional

P: pattern—progressive

P: parents—no family history

Y: years—age less than 6

From Dodick D. Headache as a symptom of ominous disease. What are the warning signals? Postgrad Med 1997;101(5):46–50, 55-46, 62-44.

Specific CT and MR Imaging Protocol Considerations

Noncontrast MR imaging is the study of choice for most pediatric patients with headache (**Box 3**). Susceptibility-weighted imaging (SWI); MR arteriography or MR venography; perfusion sequences, such as arterial spin labeling (ASL); isotropic 3-D fast turbo spin-echo sequences; postcontrast sequences; and MR spectroscopy may be added for evaluation of specific disorders.

Using conventional acquisition parameters, headache evaluation currently requires approximately 45 minutes to an hour to complete at Boston Children's Hospital. *Fast brain*, or rapidly acquired, sequences can be performed on 3T MR imaging units to decrease acquisition time and reduce the need for anesthesia.[32] Generally, these are commercially available sequences that are modified using parallel imaging with higher

Table 3
Summary of relevant clinical guidelines for neuroimaging in pediatric headache

2002 American Academy of Neurology and Child Neurology Society	
No neuroimaging in children with recurrent headaches and a normal neurologic examination	
Imaging should be considered with abnormal neurologic examination, recent onset of severe headache, change in headache type, evidence of neurologic dysfunction	
2013 Choosing Wisely campaign (American Board of Internal Medicine and American Headache Society)	
Neuroimaging not recommended with stable migraine headaches	
MR imaging (when available) preferred over CT in the nonemergent evaluation of headache	
2017 American College of Radiology Appropriateness Criteria for Headache–Child[11]	
Type of headache (initial imaging)	Appropriate imaging modality or modalities *(usually appropriate in italics)*
Primary headache	Imaging usually not appropriate
Secondary headache	*MR imaging without or without and with contrast* MR imaging with MR arteriography or MR venography CT without or with contrast
Headache due to remote trauma	*MR imaging without contrast*
Headache attributed to infection	*MR imaging without and with contrast* MR imaging without contrast MR imaging with MR arteriography or MR venography CT without or with contrast
Thunderclap headache	*CT without contrast* *MR arteriography without contrast* *MR imaging without contrast* CTA

Data from Refs.[11,20,21]

acceleration factors, gradient echo T1-weighted imaging, and elimination of manual intersequence adjustments.[32] At Boston Children's Hospital, typical fast brain MR imaging studies are currently approximately 11 minutes in duration. Fast brain sequences are often useful in headache patients who are approximately 4 years to 7 years of age and in many older children and adolescents, including those with developmental delay. Diagnostic quality can be comparable to standard acquisitions.[32] On 1.5T MR imaging units, periodically rotated overlapping parallel lines with enhanced reconstruction imaging is sometimes used to decrease scan times. At Boston Children's Hospital, rapid axial T2-weighted echo-planar fast spin-echo imaging is often used to rapidly evaluate ventricular size in hydrocephalus patients with headache.[33]

CT may be used instead of MR imaging in emergency and critical care settings, in patients with contraindications to MR imaging, and for evaluation of new focal neurologic deficits when MR imaging is less available. Strategies to reduce

Box 2
Strategies for minimizing anesthesia in neuroimaging

- Real-time case monitoring with close communication between radiologists and technologists
- Feed and swaddle technique (infants)
- Dual-source CT
- Fast brain MR imaging sequences on 3T units
- Distraction techniques (eg, video goggles)
- Patient selection and real-time intervention by child life specialists

Box 3
Basic MR imaging protocol for imaging of pediatric patients with headache

- Sagittal T1-weighted 3D T1-weighted gradient echo sequence, reformatted into axial and coronal planes
- Axial T2-weighted sequence
- Axial fat-suppressed FLAIR sequence
- Axial DTI
- Coronal T2-weighted sequence

radiation dose in pediatric CT include patient weight/size–based parameter adjustments and selection of lower-dose parameters when higher image noise can be tolerated (eg, sinus disease, known hydrocephalus, and bone lesions). Dual-source CT can both decrease dose and decrease scan time to less than 1 second and, hence, decrease the need for sedation.

APPROACH TO IMAGING FOR PEDIATRIC HEADACHES CAUSED BY SPECIFIC CONDITIONS

Numerous underlying conditions can cause headaches in pediatric patients. This discussion briefly describes each entity, summarizing the epidemiology, clinical presentation, optimal imaging study and protocol selection, and imaging findings. **Table 4** lists adjunct sequences that may be added to routine MR imaging protocols for specific conditions.

Emergent Causes of Pediatric Headache

Hydrocephalus

Hydrocephalus is due to imbalance between production and absorption of cerebrospinal fluid (CSF) (**Fig. 2**) and is a rare cause of pediatric headache (<1%).[19,34] Early morning headache with nausea and vomiting, sometimes with changes in personality and behavior, is typical and may be accompanied by signs of elevated intracranial pressure.[35] Intracranial hemorrhage (ICH) or infection, aqueductal stenosis, Chiari malformations, and tumors are common causes.

Either CT or MR imaging may be appropriate for imaging evaluation. Radiographic shunt series are an important adjunct study.

Infection

Infection causing pediatric headaches can be broadly divided into systemic infection (eg, upper respiratory infection and febrile illnesses), head and neck infection (**Fig. 3**) (eg, sinusitis, otomastoiditis, and orbital infection), and primary central nervous system (CNS) infection (eg, meningitis, encephalitis, and abscess). Immunocompromised patients are at increased risk.

Head and neck and systemic infections are common, and sinus, mastoid, and middle ear mucosal disease and effusion are often incidentally detected even in asymptomatic pediatric patients.[36] Upper respiratory infection has been found to represent the most common cause of headache, in approximately 31% of patients.[19] It can be difficult to determine whether non-CNS systemic or head and neck infection is causal or merely concurrent with primary or other causes of secondary headache.[36] These infections are generally determined to be causal if headache symptoms parallel the course of the infection, headaches are typical for the type of infection, and infection has been clinically diagnosed.[10]

Primary CNS infections are much more rare (approximately 2% of cases presenting to the pediatric emergency department) and tend to present with systemic symptoms and laboratory abnormalities, meningismus, and neurologic signs or altered mental status.[10,19]

In suspected primary CNS infection, clinical and laboratory evaluations are essential. Headache, high fever, altered mental status, focal neurologic signs, and seizures may be indications for neuroimaging.[11] Noncontrast CT may be obtained prior to lumbar puncture when there is concern for intracranial mass effect or herniation.[11] MR imaging without and with contrast is the neuroimaging study of choice. MR imaging may help to distinguish infection from other clinical entities, detect complications (eg, meningitis, encephalitis, and intracranial abscess or empyema), and identify the causal pathogen.

Imaging is usually not indicated in acute uncomplicated sinusitis. Noncontrast CT is indicated for persistent sinusitis. Noncontrast CT or MR imaging without and with contrast are indicated when there is concern for orbital or intracranial complications.[11]

Tumor

Brain tumors are a *do-not-miss* diagnosis and a common cause of concern among patients, parents, and referring providers (**Fig. 4**). Headache is a common early symptom and presenting complaint (particularly for posterior fossa tumors), found in 62% to 88% of cases.[16] Tumors are usually accompanied, however, by neurologic signs and are found in fewer than 1% of children with isolated headaches.[16,34]

CT is often performed in the emergent setting, especially for assessment of significant mass effect, hemorrhage, acute hydrocephalus, or herniation. MR imaging performed without and with contrast is superior for presurgical evaluation, including assessment of disease extent. Advanced imaging techniques, such as diffusion-weighted imaging (DWI), perfusion imaging, and MR spectroscopy, can provide physiologic evaluation of tumors.

Brain ischemia

Headaches are a common presenting feature of ischemic stroke in children, found in

Table 4
Summary of suggested adjunct MR imaging sequences for tailoring imaging assessment in specific conditions

Hydrocephalus	T2 echo-planar fast spin-echo (*ventricle check* MR imaging) Sagittal T2-weighted fast spin-echo sequence to determine endoscopic third ventriculostomy patency
Primary CNS infection	Postgadolinium T1-weighted sequence SWI (septic emboli, hemorrhagic infection [eg, herpes encephalitis]) MR arteriography, MR venography (vasospasm, CSVT)
Head and neck infection	Postgadolinium T1-weighted gradient-echo sequence Postgadolinium fat-suppressed T1-weighted sequence (2 planes) Fat-suppressed T2-weighted sequence (2 planes) MR venography
Brain tumor	SWI MR spectroscopy ASL and/or dynamic contrast-enhanced perfusion imaging Postgadolinium T1-weighted spine imaging (assessment for CSF dissemination) MR arteriography, MR venography
Stroke	SWI ASL MR arteriography MR venography Postgadolinium T1-weighted sequence
ICH due to an underlying tumor or vascular lesion	SWI MR arteriography Postgadolinium 3D T1-weighted spin-echo (tumor) and/or gradient echo (vascular lesion) sequences
Hemiplegic migraine	SWI ASL MR arteriography
Vasculopathy (eg, PACNS, moyamoya)	SWI MR arteriography ASL, velocity selective-ASL Vessel wall imaging
CSVT	MR venography Postgadolinium 3D T1-weighted gradient echo sequence
PRES	SWI ASL MR arteriography
Chiari I malformation	T2-weighted sagittal and axial sequences of the cervical spine Phase contrast or cine sequence CSF flow study Spinal cord DTI sequence
Non-neoplastic cysts	T2-weighted balanced steady-state gradient echo sequence T2-weighted echo-planar fast spin-echo sequence Sagittal T2-weighted spin-echo sequence (pineal region lesions causing aqueductal obstruction)
Mitochondrial disease	SWI MR spectroscopy
Idiopathic intracranial hypertension	MR venography Postgadolinium 3D T1-weighted gradient echo sequence (vascular imaging is to exclude CSVT)

approximately a quarter to half of affected children, usually with accompanying focal neurologic deficits.[37,38] Headache may be a more common stroke symptom in children than in adults.[37,38] Arterial ischemia-related headaches are more common in children over 5 years of age (possibly due to greater ability to verbalize symptoms), with increasing incidence

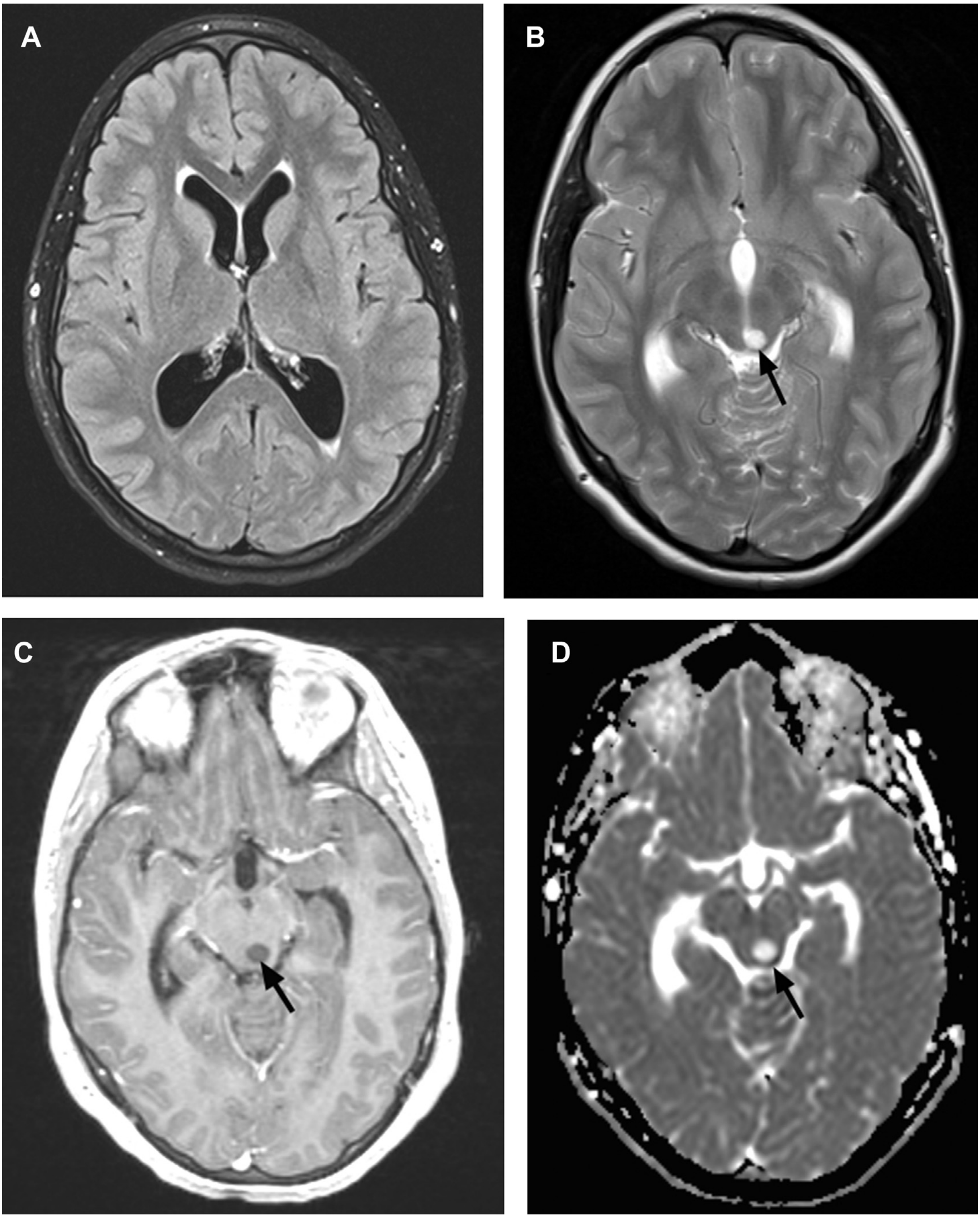

Fig. 2. A 12-year-old girl with 2 weeks of headache due to tectal glioma causing obstructive hydrocephalus. (*A*) Axial FLAIR MR imaging demonstrates mildly dilated lateral ventricles with transependymal edema. Axial T2-weighted (*B*) and postgadolinium 3D T1-weighted gradient echo images (*C*) and apparent diffusion coefficient map (*D*) demonstrate a nonenhancing small, round tectal tumor with T2 prolongation and facilitated diffusion (*arrows*). The patient underwent endoscopic third ventriculostomy.

into adolescence. Headaches are more common in vascular dissection and transient arteriopathy of childhood (approximately 70% each).[37]

CT is often the first study chosen in the emergency setting and is used to evaluate for hemorrhage or large territorial infarction (contraindications to thrombolysis).[39,40] MR imaging is more

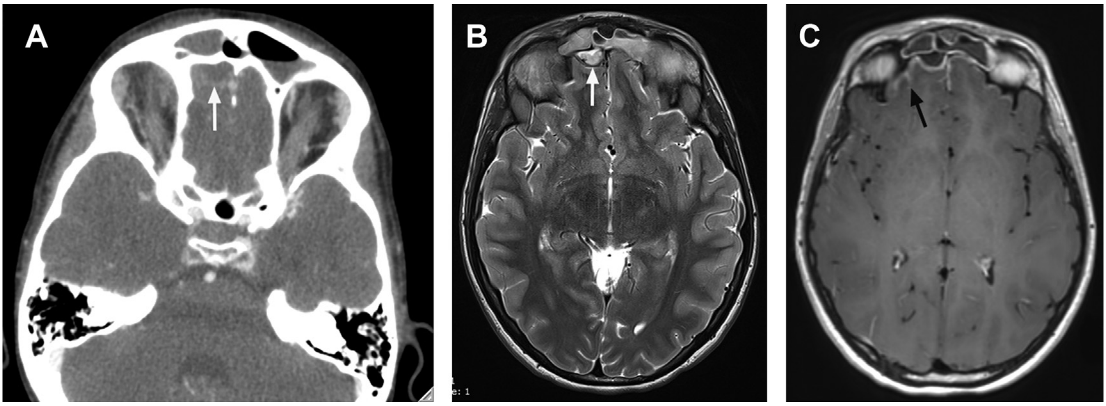

Fig. 3. A 15-year-old boy with 4 days of headache, fever, and eye swelling due to acute, complicated sinusitis. Axial CT with contrast (*A*) and axial T2-weighted MR imaging (*B*) demonstrate frontal sinus opacification with air-fluid levels and a small right frontal epidural abscess (*arrows*). Postgadolinium 3D T1-weighted spin-echo sequence (*C*) shows leptomeningeal enhancement adjacent to the abscess (*arrow*), concerning for meningitis.

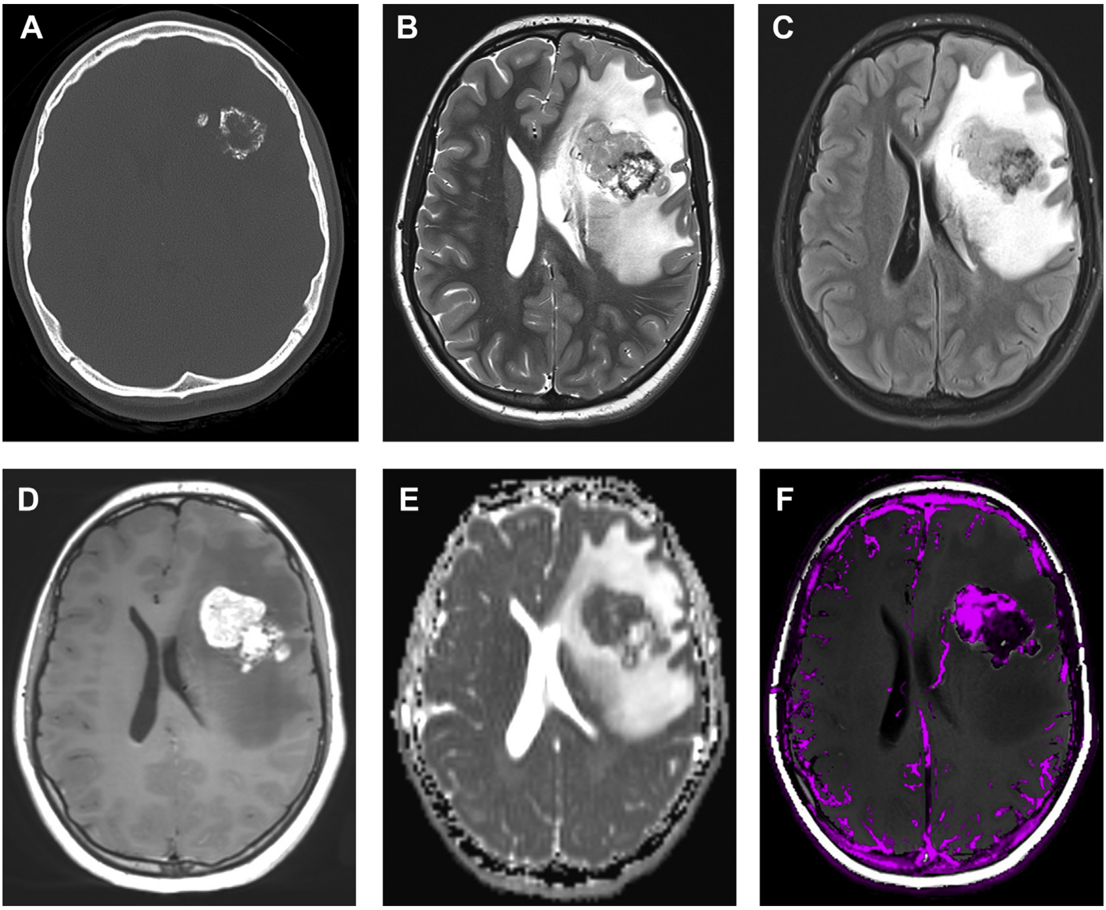

Fig. 4. A 16-year-old girl with 1 month of headache due to a brain tumor. Axial CT (*A*) and T2-weighted (*B*), FLAIR (*C*), postgadolinium 3D T1-weighted spin-echo (*D*), apparent diffusion coefficient map (*E*), and post-processed dynamic contrast-enhanced perfusion (*F*) MR images show a partially calcified, enhancing intra-axial tumor with low diffusivity, elevated blood flow, extensive surrounding vasogenic edema, and mild mass effect. The resected lesion represented anaplastic ganglioglioma.

sensitive for acute ischemia. CT and MR imaging findings vary with timing relative to the ischemic insult.

Intracranial hemorrhage due to underlying vascular lesions

Nontraumatic acute ICH in pediatric patients is often caused by underlying vascular lesions (eg, arteriovenous malformations [AVMs] [Fig. 5], aneurysms, and cavernous malformations). Noncontrast CT is usually the initial study for evaluation of pediatric patients with thunderclap headache and suspected acute ICH.

Headache is relatively common in patients with AVM even without ICH, with a wide range of reported incidence of 9% to 70% of cases at presentation.[41] Headaches commonly localize to the side of the lesion. Occipital location is a risk factor for headache, which is often associated with visual symptoms.[41]

Less than 5% of intracranial arterial aneurysms present in children. Most affect children older than 5 years of age, and aneurysms are approximately twice as common in boys.[42] They are more commonly found at the internal carotid artery bifurcation and in the posterior circulation than in adults, and mycotic, giant, and posttraumatic aneurysms are more common.[42] Headache is a common presenting symptom, affecting 82% of patients, with a median 2-week prodrome.[42] Seizure and cranial neuropathies are more common in children than in adults.[42]

CMs are vascular malformations comprised of dilated endothelium-lined blood vessels without

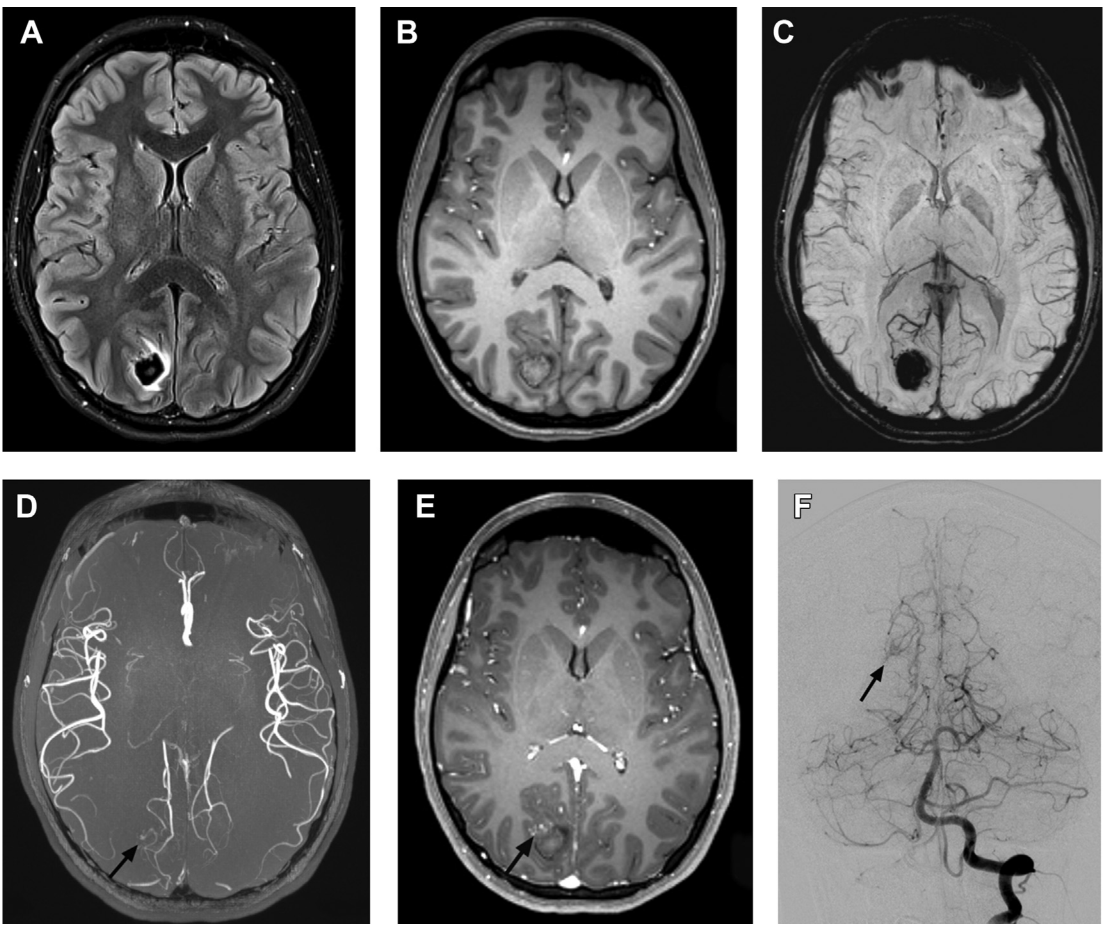

Fig. 5. A 15-year-old girl with headache and visual field cut due to an AVM-related hemorrhage. Axial FLAIR (*A*), 3D T1-weighted gradient echo (*B*), and SWI (*C*) MR images demonstrate a small right occipital hematoma with mild surrounding vasogenic edema. Axial time-of-flight MR arteriography maximum intensity projection image (*D*) and postgadolinium 3D T1-weighted gradient echo (*E*) demonstrate a small vascular nidus (*arrows*). (*F*) Left vertebral artery injection image from a conventional angiogram obtained several months later, after resolution of the hematoma, demonstrates the nidus (*arrow*). The nidus was angiographically occult at presentation due to compression of the small vessels by the hematoma. The AVM was resected.

muscular and adventitial layers.[43,44] Approximately one-quarter present in pediatric patients.[43] In children under 6 years old, lesions tend to be larger and are more likely to bleed than in children over 12 years old. At presentation, headaches (approximately 3%) are less common than seizures (38%), focal neurologic deficits (42%), and intracranial hypertension (29%).[43]

For evaluation of underlying vascular lesions, CT arteriography and MR imaging, including precontrast and postcontrast vascular imaging, may be complementary. Soft tissue contrast resolution of CTA is lower than that of MR imaging, but its higher spatial resolution and decreased sensitivity to motion artifact can be helpful in detecting abnormal vessels. If no underlying lesion is found, conventional angiography and/or delayed follow-up imaging after resolution of any hematoma that may be compressing abnormal vessels or obscuring tumor (usually weeks to months after the acute hemorrhage) is indicated. Conventional contrast angiography is also used for image-guided intervention.

Vascular Causes of Headache Without Acute Hemorrhage

Hemiplegic migraine

Hemiplegic migraine is a rare migraine variant (approximately 4% in 1 large pediatric series)[45] that mimics cerebral infarction (**Fig. 6**).[10,45] Migraine with aura is accompanied by fully reversible motor, visual, sensory, and/or speech and language deficits that usually last less than 72 hours but may persist for weeks.[10] Familial cases are often caused by membrane channel mutations (eg, CACNA1A, ATP1A2, and SCN1A).[46]

MR imaging may be normal during an acute episode or include findings of unilateral cortical swelling and hyperintensity on fluid-attenuated

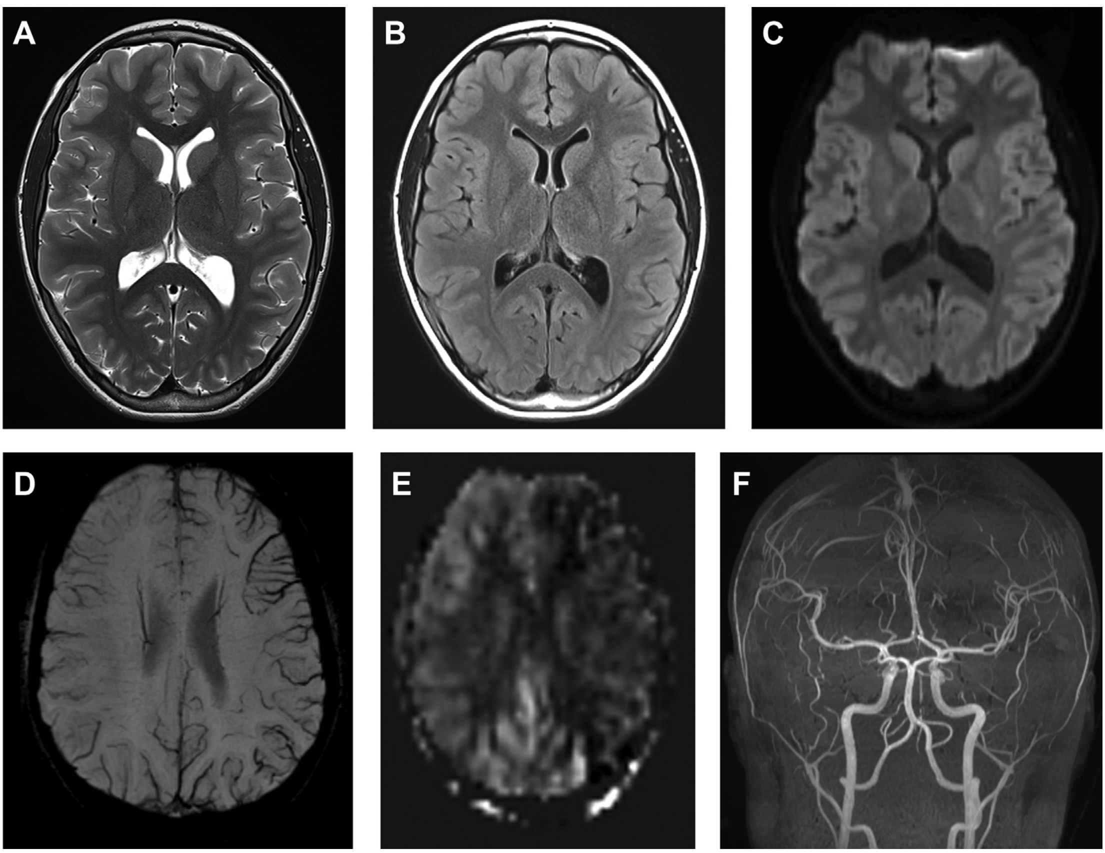

Fig. 6. A 14-year-old girl with headache due to hemiplegic migraine, presenting with transient aphasia and right-sided weakness. Axial T2-weighted (*A*), FLAIR (*B*), and DWI (*C*) MR images are normal. Axial SWI (*D*) demonstrates asymmetric venous prominence in the left cerebral hemisphere, suggesting increased tissue oxygen extraction. ASL (*E*) shows decreased flow in the left cerebral hemisphere, and coronal maximum intensity projection 3-D reconstruction from time-of-flight MR arteriography (*F*) shows pruning of distal left middle cerebral artery branches. Imaging abnormalities resolved on 3-week follow-up MR imaging (not pictured).

inversion recovery (FLAIR) and T2-weighted imaging, decreased perfusion, diminished caliber of vessels on MR arteriography, and asymmetric prominence of cortical veins on SWI due to increased tissue oxygen extraction and elevated venous deoxyhemoglobin. Normal DWI excludes ischemic injury. Imaging abnormalities resolve on follow-up.[46–48] Transient neurologic deficits with similar imaging findings can be seen even without migraine in an entity that has been termed *regional cerebral hypoperfusion*.[49]

Vasculitis

In childhood primary angiitis of the CNS (PACNS), clinical and imaging features vary by type (nonprogressive, progressive, and small-vessel angiography-negative, biopsy-positive types).[50] Serum inflammatory markers are typically elevated.[51] Ischemia-induced headache accompanied by focal neurologic deficits or seizures is common in angiography-positive disease.[50]

MR imaging is the imaging modality of choice. Time-of-flight MR arteriography may overestimate or underestimate abnormalities.[50] ICH may affect decisions pertaining to thrombolysis. Perfusion imaging is helpful for evaluating tissue at risk. CTA may be useful to evaluate MR arteriography abnormalities that are suspected to represent artifact, whereas conventional angiography is the gold standard for evaluation of the vessels.[50,51] Using postgadolinium T1-weighted spin-echo imaging at 3T, thickening and increased enhancement of the vessel wall may be detected even in nonstenotic arteries.[52]

Cerebral sinovenous thrombosis

Cerebral sinovenous thrombosis (CSVT) is rare in children (Fig. 7).[53] Although seizures are the most common presenting clinical feature overall (a majority of cases are in neonates and infants under 6 months), older children typically present with headaches (32%–68%) and motor symptoms.[53,54]

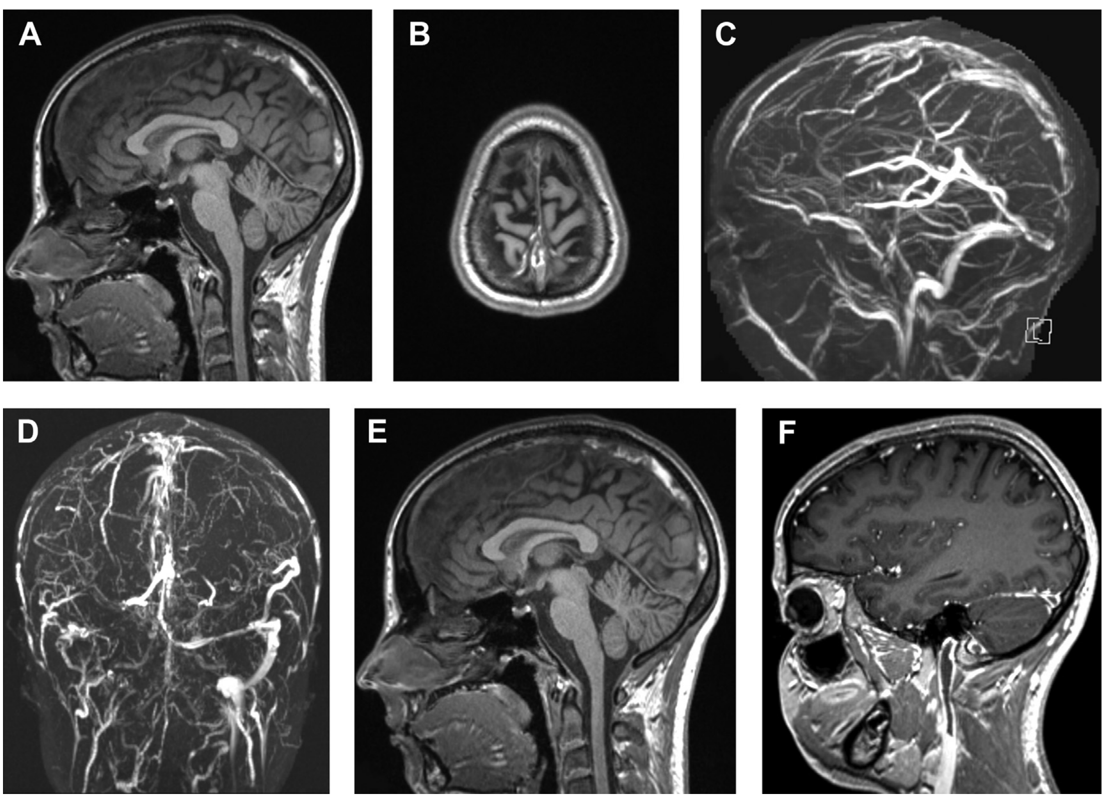

Fig. 7. A 16-year-old girl with headache, ataxia, and papilledema due to CSVT 10 days after influenza infection. Sagittal and axial 3D T1-weighted gradient echo MR images (*A*, *B*) demonstrate extensive hyperintense thrombus within the superior sagittal sinus and tributary cortical veins. Sagittal and coronal maximum intensity projection 3-D reconstructions from time-of-flight MR venography (*C*, *D*) demonstrate loss of flow-related enhancement within the superior sagittal sinus and right transverse and sigmoid sinuses and upper internal jugular vein. Postcontrast sagittal 3D T1-weighted gradient echo (*E*, *F*) demonstrates filling defects in the same distribution. MR imaging showed findings of elevated intracranial pressure (not pictured).

Complications may include hemorrhagic infarction (40%) and hydrocephalus.[54]

When CSVT is suspected, CT venography or MR venography is indicated even if initial CT is normal. On noncontrast CT, involved veins may appear hyperdense (>70 Hounsfield units) and distended. Absent flow-related enhancement on MR venography may correspond to intravenous filling defects on CT venography and postcontrast 3D T1-weighted gradient echo imaging.[54] Involvement of multiple veins is common (70%).[53] MR imaging can evaluate for edema, ischemia, and hemorrhagic infarction with high sensitivity and specificity.[54]

Posterior reversible encephalopathy syndrome

Posterior reversible encephalopathy syndrome (PRES) results from failed cerebrovascular autoregulation or cytotoxic endothelial injury. Risk factors include renal or autoimmune disease, malignancy, hypertension (85%), and chemotherapy or immunosuppressive therapy. Seizure (90%), encephalopathy, headache (40%), and visual disturbances are common in pediatric patients.

CT is often the initial imaging modality for suspected PRES; however, MR imaging should be obtained even when CT is normal. Findings on CT and MR can include hemispheric edema. In typical PRES, this most often presents in the parietal and occipital lobes, followed by the frontal lobes and cerebellum.[55] Atypical PRES is more common in children than adults, with relatively greater involvement of the frontal and temporal lobes and deep gray matter.[56] More frequent involvement of the cerebellum has not been universal across studies.[57] Low diffusivity and hemorrhage are overall less common atypical features that may be more frequent in children.[56] Perfusion sequences may show regional hypoperfusion and hyperperfusion, and angiographic imaging may show diffuse arterial constriction, narrowing, and beading.[58]

Moyamoya vasculopathy

Moyamoya vasculopathy is characterized by progressive steno-occlusive disease of the internal carotid artery and its terminal branches, with resultant formation of puff-of-smoke lenticulostriate and leptomeningeal collaterals (**Fig. 8**). Headaches are common before and after revascularization surgery and may resemble migraines, although may be refractory to usual pharmacologic therapy. Headaches may be caused by hypoperfusion or redistributed blood flow.[59]

Although conventional angiography is usually necessary to define disease extent, MR imaging with MR arteriography plays an important role at diagnosis and in follow-up for assessment of sulcal bright signal on FLAIR (ivy sign), infarction, vascular steno-occlusive disease, and collateral vessels. ASL may show regions of decreased flow due to increased path length of labeled spins through leptomeningeal collateral vessels; velocity selective ASL (vs-ASL) may reflect perfusion more accurately.[60]

Chiari I Malformation

Chiari I malformation is defined by cerebellar tonsillar descent to at least 5 mm below the foramen magnum. Headache is the most common presenting symptom, with Chiari I malformation identified in approximately 6% of children imaged for headache and 12% with the malformation presenting with headache (**Fig. 9**).[61,62] Chiari I malformation frequently coexists with primary headache and many patients meeting imaging criteria are asymptomatic; therefore, clinical correlation is required. Specific clinical features linking headaches to Chiari I malformation include occipital location; precipitation by Valsalva maneuver; short duration; and bulbar, cerebellar, lower cranial nerve, and upper cervical spinal cord signs.[10] Greater tonsillar descent is associated with greater headache severity.[63] Occipital headaches and those that are precipitated by Valsalva-like maneuvers are more likely to respond to surgical intervention.[10]

Advanced MR imaging sequences, such as cardiac-gated phase contrast for CSF flow assessment, pulse-gated cine for cerebellar tonsillar motion, quantitative volumetric assessment of the posterior fossa and biometry of the skull base, and spinal cord diffusion tensor imaging (DTI), have been investigated but are not currently in wide clinical use.[64]

Non-neoplastic Cysts and Their Complications

Arachnoid cyst

Arachnoid cysts form when CSF accumulates between the walls of a duplicated arachnoid membrane (**Fig. 10**). Most are found in children and young adults, commonly as an incidental finding.[61,65] Incidence in pediatric patients with headache is approximately 1% to 3%.[61] Rare complications include enlarging cyst (children under 4 years of age), intracystic and subdural hemorrhage (especially after trauma), and obstructive hydrocephalus.[65,66]

MR imaging assessment may include a balanced steady-state gradient echo sequence for delineation of the cyst wall. At Boston

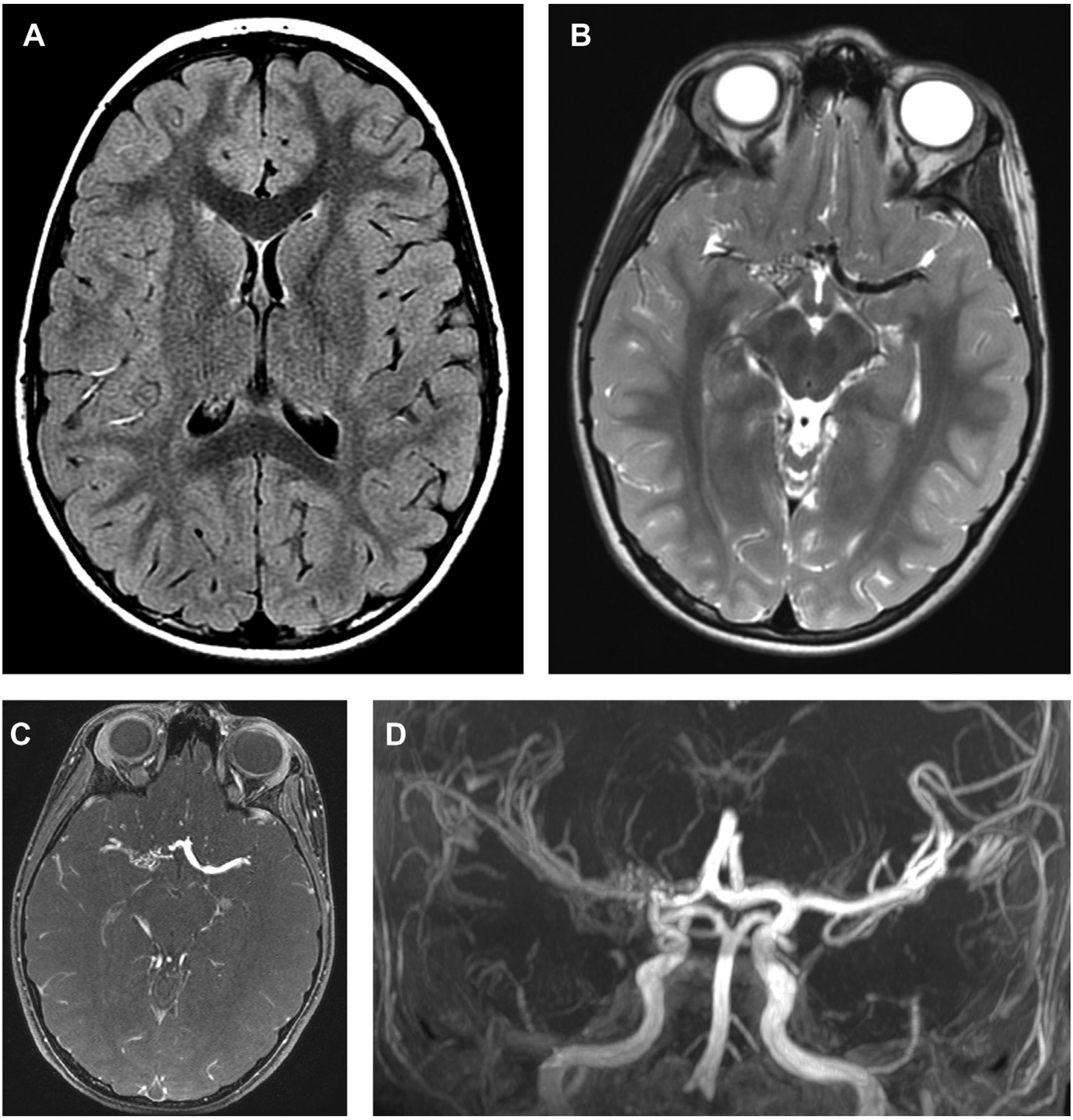

Fig. 8. A 9-year-old girl with headaches and right-sided moyamoya vasculopathy. (*A*) Axial FLAIR MR imaging demonstrates ivy sign connoting leptomeningeal collateral flow in the right cerebral hemisphere. Axial T2-weighted image (*B*), axial time-of-flight MR arteriography image (*C*), and coronal time-of-flight MR arteriography maximum intensity projection 3-D reconstruction (*D*) demonstrate severe narrowing of the right internal carotid artery terminus and the A1 and M1 segments, with numerous middle cerebral artery (MCA) cistern collaterals and pruning of distal MCA branches. The patient was treated with pial synangiosis.

Children's Hospital, echo-planar fast spin-echo imaging often is used to follow arachnoid cysts for changes in size or fluid reaccumulation after surgery.

Colloid cyst

Colloid cysts, believed to arise from endodermal remnants at the roof of the embryonic diencephalon, are rare in children, with fewer than 8% presenting before 15 years of age.[65]

Approximately 50% to 60% of symptomatic patients have headaches, often with papilledema. Precipitous frontal headaches and nausea and vomiting that are relieved when the patient is lying down are characteristic.[65] Acute hydrocephalus may result in sudden neurologic deterioration or death. Noncontrast CT and/or MR imaging demonstrate a round lesion at the roof of the third ventricle near the foramen of Monro, which is hyperdense

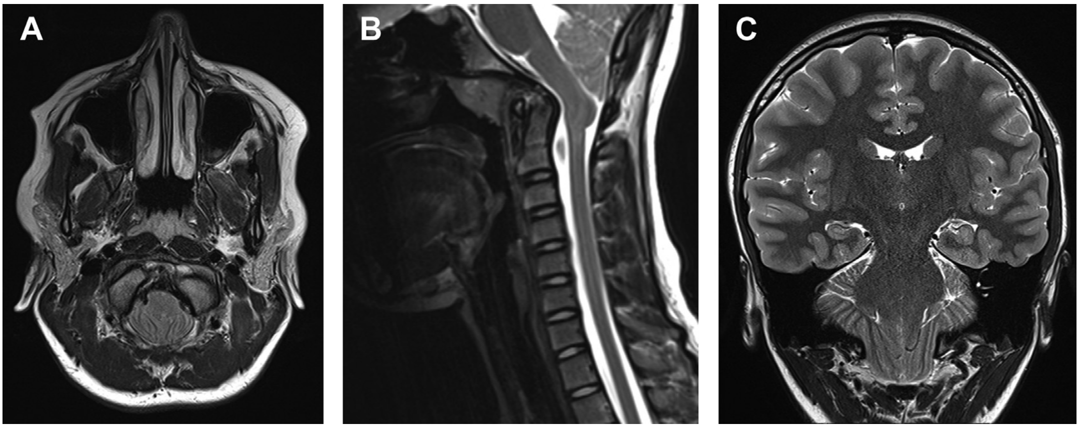

Fig. 9. A 13-year-old girl with brief headaches and syncopal episodes due to Chiari I malformation. Axial, sagittal, and coronal T2-weighted MR images (*A–C*) show cerebellar tonsillar descent below the foramen magnum, with crowding of the adjacent CSF spaces and dorsal bump at the cervicomedullary junction. The patient underwent posterior fossa decompression.

on CT, with variable signal intensity on MR imaging.[65]

Miscellaneous Conditions

Mitochondrial disease

Migraine-like headaches that have been attributed to vasculopathy are common in mitochondrial encephalopathy with lactic acidosis and stroke-like episodes (MELAS).[10] Both tension-type and migraine-like headaches, however, are common even in mitochondrial disorders without vasculopathy, suggesting pathogenesis related to abnormal mitochondrial metabolism.[67]

In MELAS, neuroimaging findings include stroke-like lesions crossing vascular territories; deep gray matter changes including basal ganglia mineralization; white matter abnormalities and parenchymal volume loss; and elevated lactate in the parenchyma and CSF on MR spectroscopy.[68]

Idiopathic intracranial hypertension

Idiopathic intracranial hypertension is rare in children, in particular those under 10 years of age, with most cases of intracranial hypertension being secondary (**Fig. 11**). The predilection for female and obese patients that has been described in adults has not been observed in pediatric patients. Headache is the most common symptom, occurring in greater than 90% of affected patients.[69]

Hartmann and colleagues[70] found no significant difference between children and adults in MR

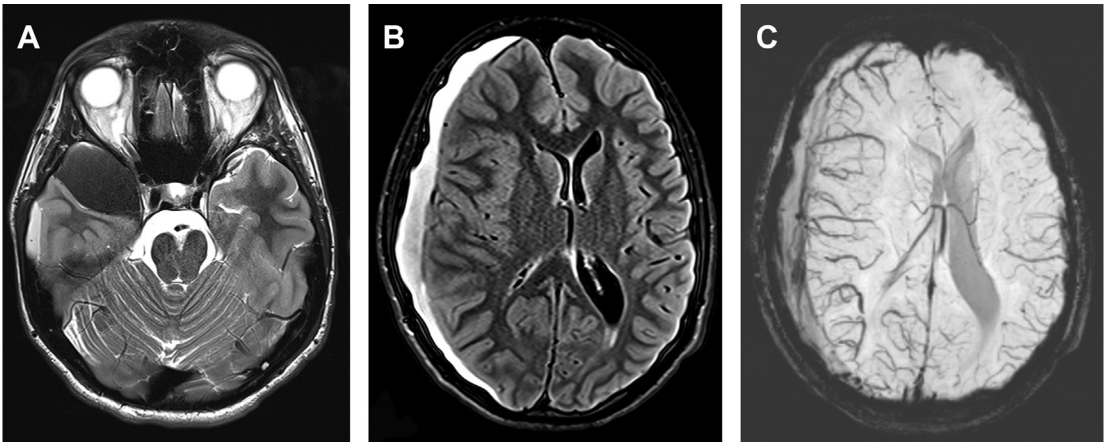

Fig. 10. A 17-year-old boy with 2 months of headache, presenting with worsening headache and vomiting due to hemorrhage complicating an arachnoid cyst. Axial T2-weighted MR image (*A*) demonstrates a right middle cranial fossa arachnoid cyst containing hypointense blood products. Axial FLAIR (*B*) and SWI (*C*) demonstrate a right holohemispheric subdural hematoma with mass effect and venous congestion in the right cerebral hemisphere.

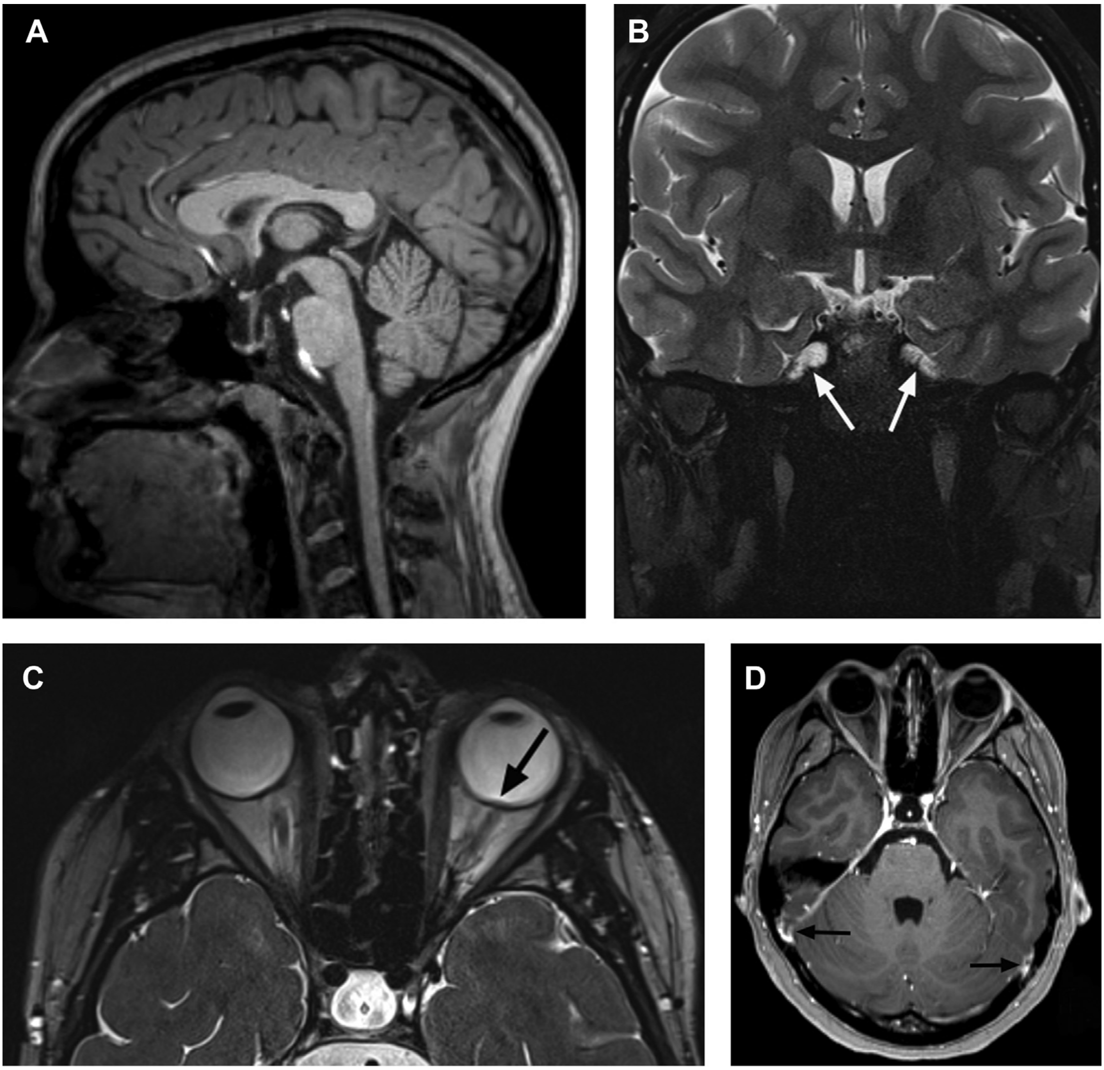

Fig. 11. A 17-year-old girl with headache, blurry vision, and vomiting due to idiopathic intracranial hypertension. Sagittal 3D T1-weighted gradient echo image (*A*) demonstrates a partially empty sella. Coronal T2-weighted MR image (*B*) demonstrates enlarged Meckel caves (*arrows*). Axial T2-weighted MR image (*C*) demonstrates flattening of the posterior globes and subtle elevation of the left optic papilla (*arrow*). Axial postgadolinium T1-weighted 3D T1-weighted gradient echo (*D*) shows flattening of the transverse sinuses at the sinodural angle (*arrows*). There was no evidence of CSVT.

imaging findings of optic nerve tortuosity and optic nerve head protrusion, increased perioptic CSF, tonsillar herniation, and enlargement of Meckel cave. Optic nerve enhancement was more common in prepubertal patients, and scleral flattening, transverse sinus stenosis, meningoceles, and sellar changes were less common.

SUMMARY

Pediatric headache is a common clinical problem and reason for neuroimaging referral. The yield of imaging is low in patients with primary headache disorders. There are many secondary causes of pediatric headache, for which neuroimaging tends to be more useful. Multiple published clinical guidelines are available to guide imaging modality selection. Tailored CT and MR selection and protocoling are key for optimal use and should include incorporation of shortened examinations, which decrease the need for anesthesia in pediatric neuroimaging.

REFERENCES

1. Trofimova A, Vey BL, Mullins ME, et al. Imaging of children with nontraumatic headaches. AJR Am J Roentgenol 2018;210(1):8–17.

2. Gilbert JW, Johnson KM, Larkin GL, et al. Atraumatic headache in US emergency departments: recent trends in CT/MRI utilisation and factors associated with severe intracranial pathology. Emerg Med J 2012;29(7):576–81.
3. Larson DB, Johnson LW, Schnell BM, et al. Rising use of CT in child visits to the emergency department in the United States, 1995-2008. Radiology 2011;259(3):793–801.
4. Rho YI, Chung HJ, Suh ES, et al. The role of neuroimaging in children and adolescents with recurrent headaches–multicenter study. Headache 2011; 51(3):403–8.
5. Abu-Arafeh I, Macleod S. Serious neurological disorders in children with chronic headache. Arch Dis Child 2005;90(9):937–40.
6. Lateef TM, Merikangas KR, He J, et al. Headache in a national sample of American children: prevalence and comorbidity. J Child Neurol 2009;24(5): 536–43.
7. Abu-Arafeh I, Razak S, Sivaraman B, et al. Prevalence of headache and migraine in children and adolescents: a systematic review of population-based studies. Dev Med child Neurol 2010;52(12): 1088–97.
8. Scheller JM. The history, epidemiology, and classification of headaches in childhood. Semin Pediatr Neurol 1995;2(2):102–8.
9. Blume HK. Pediatric headache: a review. Pediatr Rev 2012;33(12):562–76.
10. Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders, 3rd edition. Cephalalgia 2018;38(1):1–211.
11. Hayes LL, Palasis S, Bartel TT, et al. ACR appropriateness criteria® headache-child 2017. Available at: https://acsearch.acr.org/docs/69439/Narrative/. Accessed February 23, 2018.
12. Rothner AD. The evaluation of headaches in children and adolescents. Semin Pediatr Neurol 1995;2(2): 109–18.
13. Aminoff MJ, Greenberg DA, Simon RP. Headache & facial pain. Clinical neurology. 9th edition. New York: McGraw-Hill; 2015. Available at: https://accessmedicine.mhmedical.com/content.aspx?bookid=1194&sectionid=78427211. Accessed July 23, 2018.
14. Borsook D, Maleki N, Burstein R. Migraine. In: Zigmond MJ, Coyle JT, editors. Neurobiology of brain disorders: biological basis of neurological and psychiatric disorders. 1st edition. Cambridge (MA): Academic Press; 2015. p. 693–708.
15. DeVries A, Young PC, Wall E, et al. CT scan utilization patterns in pediatric patients with recurrent headache. Pediatrics 2013;132(1):e1–8.
16. The epidemiology of headache among children with brain tumor. Headache in children with brain tumors. The Childhood Brain Tumor Consortium. J Neurooncol 1991;10(1):31–46.
17. Ahmed MA, Martinez A, Cahill D, et al. When to image neurologically normal children with headaches: development of a decision rule. Acta Paediatr 2010; 99(6):940–3.
18. Dodick D. Headache as a symptom of ominous disease. What are the warning signals? Postgrad Med 1997;101(5):46–50, 55-46, 62-44.
19. Conicella E, Raucci U, Vanacore N, et al. The child with headache in a pediatric emergency department. Headache 2008;48(7):1005–11.
20. Lewis DW, Ashwal S, Dahl G, et al. Practice parameter: evaluation of children and adolescents with recurrent headaches: report of the Quality Standards Subcommittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society. Neurology 2002;59(4):490–8.
21. Loder E, Weizenbaum E, Frishberg B, et al. Choosing wisely in headache medicine: the American Headache Society's list of five things physicians and patients should question. Headache 2013; 53(10):1651–9.
22. Goske MJ, Applegate KE, Boylan J, et al. The Image Gently campaign: working together to change practice. AJR Am J Roentgenol 2008;190(2):273–4.
23. Scheinfeld MH, Moon JY, Fagan MJ, et al. MRI usage in a pediatric emergency department: an analysis of usage and usage trends over 5 years. Pediatr Radiol 2017;47(3):327–32.
24. McDonald JS, McDonald RJ, Jentoft ME, et al. Intracranial gadolinium deposition following gadodiamide-enhanced magnetic resonance imaging in pediatric patients: a case-control study. JAMA Pediatr 2017;171(7):705–7.
25. McDonald RJ, McDonald JS, Kallmes DF, et al. Intracranial gadolinium deposition after contrast-enhanced MR imaging. Radiology 2015;275(3): 772–82.
26. McDonald RJ, McDonald JS, Kallmes DF, et al. Gadolinium deposition in human brain tissues after contrast-enhanced MR imaging in adult patients without intracranial abnormalities. Radiology 2017; 285(2):546–54.
27. Radbruch A, Haase R, Kieslich PJ, et al. No signal intensity increase in the dentate nucleus on unenhanced T1-weighted MR images after more than 20 serial injections of macrocyclic gadolinium-based contrast agents. Radiology 2017;282(3): 699–707.
28. McDonald RJ, McDonald JS, Dai D, et al. Comparison of gadolinium concentrations within multiple rat organs after intravenous administration of linear versus macrocyclic gadolinium chelates. Radiology 2017;285(2):536–45.
29. Lord ML, Chettle DR, Grafe JL, et al. Observed deposition of gadolinium in bone using a new

noninvasive in vivo biomedical device: results of a small pilot feasibility study. Radiology 2018;287(1):96–103.

30. Andropoulos DB, Greene MF. Anesthesia and developing brains - implications of the FDA warning. N Engl J Med 2017;376(10):905–7.
31. FDA Drug Safety Communication: FDA approves label changes for use of general anesthetic and sedation drugs in young children. In: US Food and Drug Administration. 2017. Available at: https://www.fda.gov/Drugs/DrugSafety/ucm554634.htm. Accessed March 23, 2018.
32. Prakkamakul S, Witzel T, Huang S, et al. Ultrafast brain MRI: clinical deployment and comparison to conventional brain MRI at 3T. J Neuroimaging 2016;26(5):503–10.
33. Robson CD, MacDougall RD, Madsen JR, et al. Neuroimaging of children with surgically treated hydrocephalus: a practical approach. AJR Am J Roentgenol 2017;208(2):413–9.
34. Yilmaz U, Celegen M, Yilmaz TS, et al. Childhood headaches and brain magnetic resonance imaging findings. Eur J Paediatr Neurol 2014;18(2):163–70.
35. Ahad R, Kossoff EH. Secondary intracranial causes for headaches in children. Curr Pain Headache Rep 2008;12(5):373–8.
36. Vieira Neto RJ, Teixeira KCS, Guerreiro MM, et al. Paranasal sinus disease in children with headache. J Child Neurol 2017;32(12):1014–7.
37. Headache far more common stroke symptom in children than adults. In: American Heart Association/American Stroke Association Newsroom. 2017. Available at: http://newsroom.heart.org/news/headache-far-more-common-stroke-symptom-in-children-than-adults. Accessed April 3, 2018.
38. Mallick AA, Ganesan V, Kirkham FJ, et al. Childhood arterial ischaemic stroke incidence, presenting features, and risk factors: a prospective population-based study. Lancet Neurol 2014;13(1):35–43.
39. Rivkin MJ, deVeber G, Ichord RN, et al. Thrombolysis in pediatric stroke study. Stroke 2015;46(3):880–5.
40. Salmela MB, Mortazavi S, Jagadeesan BD, et al. ACR appropriateness criteria((R)) cerebrovascular disease. J Am Coll Radiol 2017;14(5S):S34–61.
41. Ellis JA, Mejia Munne JC, Lavine SD, et al. Arteriovenous malformations and headache. J Clin Neurosci 2016;23:38–43.
42. Garg K, Singh PK, Sharma BS, et al. Pediatric intracranial aneurysms–our experience and review of literature. Childs Nerv Syst 2014;30(5):873–83.
43. Bilginer B, Narin F, Hanalioglu S, et al. Cavernous malformations of the central nervous system (CNS) in children: clinico-radiological features and management outcomes of 36 cases. Childs Nerv Syst 2014;30(8):1355–66.
44. Osborn AG. Vascular malformations. In: Osborn AG, Hedlund GL, Salzman KL, editors. Osborn's brain: imaging, pathology, and anatomy. 2nd edition. Philadelphia: Elsevier; 2018. p. 155–96.
45. Pacheva IH, Ivanov IS. Migraine variants–occurrence in pediatric neurology practice. Clin Neurol Neurosurg 2013;115(9):1775–83.
46. Bosemani T, Burton VJ, Felling RJ, et al. Pediatric hemiplegic migraine: role of multiple MRI techniques in evaluation of reversible hypoperfusion. Cephalalgia 2014;34(4):311–5.
47. Fedak EM, Zumberge NA, Heyer GL. The diagnostic role for susceptibility-weighted MRI during sporadic hemiplegic migraine. Cephalalgia 2013;33(15):1258–63.
48. Altinok D, Agarwal A, Ascadi G, et al. Pediatric hemiplegic migraine: susceptibility weighted and MR perfusion imaging abnormality. Pediatr Radiol 2010;40(12):1958–61.
49. Lehman LL, Danehy AR, Trenor CC 3rd, et al. Transient focal neurologic symptoms correspond to regional cerebral hypoperfusion by MRI: a stroke mimic in children. AJNR Am J Neuroradiol 2017;38(11):2199–202.
50. Moharir M, Shroff M, Benseler SM. Childhood central nervous system vasculitis. Neuroimaging Clin N Am 2013;23(2):293–308.
51. Aviv RI, Benseler SM, Silverman ED, et al. MR imaging and angiography of primary CNS vasculitis of childhood. AJNR Am J Neuroradiol 2006;27(1):192–9.
52. Mandell DM, Mossa-Basha M, Qiao Y, et al. Intracranial vessel wall MRI: principles and expert consensus recommendations of the American Society of Neuroradiology. AJNR Am J Neuroradiol 2017;38(2):218–29.
53. Wasay M, Dai AI, Ansari M, et al. Cerebral venous sinus thrombosis in children: a multicenter cohort from the United States. J Child Neurol 2008;23(1):26–31.
54. Hedlund GL. Cerebral sinovenous thrombosis in pediatric practice. Pediatr Radiol 2013;43(2):173–88.
55. Bartynski WS. Posterior reversible encephalopathy syndrome, part 1: fundamental imaging and clinical features. AJNR Am J Neuroradiol 2008;29(6):1036–42.
56. Gupta V, Bhatia V, Khandelwal N, et al. Imaging findings in pediatric posterior reversible encephalopathy syndrome (PRES): 5 years of experience from a Tertiary Care Center in India. J Child Neurol 2016;31(9):1166–73.
57. Habetz K, Ramakrishnaiah R, Raina SK, et al. Posterior reversible encephalopathy syndrome: a comparative study of pediatric versus adult patients. Pediatr Neurol 2016;65:45–51.
58. Osborn AG. Acquired metabolic and systemic disorders. In: Osborn AG, Hedlund GL, Salzman KL, editors. Osborn's brain: imaging, pathology, and

anatomy. 2nd edition. Philadelphia: Elsevier; 2018. p. 1017–70.
59. Seol HJ, Wang KC, Kim SK, et al. Headache in pediatric moyamoya disease: review of 204 consecutive cases. J Neurosurg 2005;103(5 Suppl):439–42.
60. Zun Z, Hargreaves BA, Rosenberg J, et al. Improved multislice perfusion imaging with velocity-selective arterial spin labeling. J Magn Reson Imaging 2015; 41(5):1422–31.
61. Schwedt TJ, Guo Y, Rothner AD. "Benign" imaging abnormalities in children and adolescents with headache. Headache 2006;46(3):387–98.
62. Tubbs RS, McGirt MJ, Oakes WJ. Surgical experience in 130 pediatric patients with Chiari I malformations. J Neurosurg 2003;99(2):291–6.
63. Wu YW, Chin CT, Chan KM, et al. Pediatric Chiari I malformations: do clinical and radiologic features correlate? Neurology 1999;53(6):1271–6.
64. Fakhri A, Shah MN, Goyal MS. Advanced imaging of Chiari 1 malformations. Neurosurg Clin N Am 2015; 26(4):519–26.
65. Osborn AG. Nonneoplastic cysts. In: Osborn AG, Hedlund GL, Salzman KL, editors. Osborn's brain: imaging, pathology, and anatomy. 2nd edition. Philadelphia: Elsevier; 2018. p. 867–901.
66. Wu X, Li G, Zhao J, et al. Arachnoid cyst-associated chronic subdural hematoma: report of 14 cases and a systematic literature review. World Neurosurg 2018;109:e118–30.
67. Kraya T, Deschauer M, Joshi PR, et al. Prevalence of headache in patients with mitochondrial disease: a cross-sectional study. Headache 2018; 58(1):45–52.
68. Malhotra K, Liebeskind DS. Imaging of MELAS. Curr Pain Headache Rep 2016;20(9):54.
69. Rogers DL. A review of pediatric idiopathic intracranial hypertension. Pediatr Clin North Am 2014;61(3): 579–90.
70. Hartmann AJ, Soares BP, Bruce BB, et al. Imaging features of idiopathic intracranial hypertension in children. J Child Neurol 2017;32(1):120–6.