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IMAGING (L MECTHLER, SECTION EDITOR)

Neuroimaging in Secondary Headache Disorders

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Abstract A secondary headache may develop de novo or in patients with a history of primary headaches, and a thorough history and neurological exam often helps to suspect a secondary etiology. The causes of secondary headaches include tumors, vascular etiologies, structural brain disorders, infection, inflammation, and alterations of cerebrospinal fluid pressure dynamics. Computed tomography (CT) is very sensitive for detecting acute hemorrhage but magnetic resonance imaging (MRI) is preferred over a head CT in subacute and non–emergent cases. Obtaining the correct diagnosis may include incorporation of intravenous contrast agents, special imaging sequences, and functional imaging techniques.

Keywords Secondary headaches · Neuroimaging · MRI · CT · Thunderclap headaches

This article is part of the Topical Collection on *Imaging*

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Introduction

Headaches are classified as primary or secondary. Primary headaches are migraine, tension-type headache, trigeminal autonomic cephalagias (TAC), and other primary headaches such as cough, exercise, stabbing, cold-stimulus, new daily persistent headaches, and thunderclap headaches [1]. Secondary headache disorders should be suspected when the history does not clearly fit a primary headache disorder. However, symptoms of secondary headache disorders can mimic those of the primary headaches, and secondary headaches can develop during the course of primary headaches. So when is it appropriate to obtain neuroimaging studies?

Neuroimaging should be considered in patients with non-acute headache and an unexplained abnormal finding on the neurological examination. Non-acute headaches include all headache syndromes that have occurred for at least 4 weeks in patent's lifetime. Also, headaches worsened by the Valsalva maneuver, headache that awakens the patient from sleep, new onset headache in older patients or progressively worsening headache increase the likelihood of significant intracranial pathology. The presence of fever, neck stiffness, systemic illness such as HIV or cancer are concerning for a secondary cause of the headaches. The following symptoms also appear to increase the likelihood for findings an abnormality of neuroimaging: rapidly increasing headache frequency, lack of coordination, localized neurological signs or subjective numbness or tingling, and headaches causing awakening from sleep [2, 3].

A lower threshold for imaging should be present in patients with atypical features or the headaches that do not fulfill the definition of migraine. There is an insufficient evidence to consider neuroimaging in tension-type headache [3].

The American Headache Society "Choosing Wisely" task force recommends not performing neuroimaging for a stable

headache that meets criteria for the diagnosis of migraine and not to perform computed tomography (CT) imaging when magnetic resonance imaging (MRI) is available, except in the emergency setting [4•].

The choice of neuroimaging modality depends upon the duration and intensity of the headache. Patients with sudden onset and peak intensity of headache usually present to emergency room (ER) and an emergent CT without contrast should be performed to rule out intracranial bleed. CT is also superior to MRI when evaluating for acute subarachnoid hemorrhage (SAH). When a patient presents to ER with fever, neck rigidity, and a headache, a head CT helps rule out a midline shift before proceeding with a lumbar puncture for suspicion of meningitis. Additional neuroimaging may be needed, depending on the cause of headache and the initial findings on CT.

Brain MRI is superior to CT scan to evaluate the posterior fossa, acute infarcts, and mass lesions. A contrast-enhanced study may be needed to look for evidence of meningitis, neoplasm, demyelination, and low cerebrospinal fluid (CSF) pressure states. However, MRI may not be as readily available, and when suspicion for a bleed remains high, one should proceed with a head CT. MRI is preferred over CT for non-emergent cases and non-acute headaches.

Herein, we illustrate the neuroimaging findings on the most frequently encountered secondary headache disorders (Table 1).

Headaches Attributed to Cranial or Cervical Vascular Disorders

Aneurysmal Subarachnoid Hemorrhage

Aneurysmal SAH most commonly presents with a thunderclap headache (TCH). TCH is an acute and severe headache, which reaches peak intensity within seconds to minutes and can start fading in an hour. Patients may describe this as the "worst headache of their life" [5]. The headache may be lateralized to the side of aneurysm. Loss of consciousness, meningitis, or focal neurological deficits may occur. The differential diagnosis of TCH also includes reversible cerebral vasconstriction syndrome (RCVS), cerebral venous thrombosis, cervical artery dissection, spontaneous intracranial hypotension, acute hypertensive crisis, colloid cyst, infections, pituitary apoplexy, and cervical artery dissection.

Table 1 Secondary headache disorders [1]

Headache attributed to trauma or injury to the head and/or neck
Headache attributed to cranial or cervical vascular disorder
Headache attributed to non-vascular intracranial disorder
Headache attributed to a substance or its withdrawal
Headache attributed to infection
Headache attributed to disorder of homoeostasis
Headache or facial pain attributed to disorder of the cranium, neck, eyes, ears, nose, sinuses, teeth, mouth, or other facial or cervical structure
Headache attributed to psychiatric disorder

The initial diagnostic test is non-contrast CT which demonstrates hyperintensity within the subarachnoid space [6••]. The sensitivity of CT for detecting SAH is 92.9 % and specificity is 100 %. If CT is performed within 6 h, the sensitivity increases to 100 %. The sensitivity seems to decline over the next 4 days to 50 %, as the aging hematoma becomes more isodense to water over time, making it harder to detect in the subacute and chronic phases [7•]. A minor bleed may be missed on the initial CT scan. If the clinical suspicion remains high, a lumbar puncture is necessary [7•] which reveals a high opening pressure and elevated red blood cell count which does not decrease from tube one to four. Xanthochromic supernatant is suggestive of SAH. Spectrophotometry may demonstrate elevated bilirubin [8].

Gradient echo (GRE) MRI has shown sensitivities of 94 % in the acute phase (within 4 days) and 100 % in the subacute phase (after 4 days) [9]. Fluid attenuated inversion recovery (FLAIR) sequences have sensitivities of 81 % acutely and 87 % in the subacute phase [9]. After diagnosing SAH, definitive imaging is undertaken to determine the etiology.

Ruptured aneurysms account for 80 % of non-traumatic SAH. Digital Subtraction Angiography (DSA) remains the gold standard. However, in an emergency setting and due to ease of availability, CT angiography (CTA) is the preferred imaging modality. CTA and magnetic resonance angiography (MRA) are non-invasive tests that identify aneurysms 3–5 mm or larger with high sensitivity. The diagnostic accuracy of CTA with 16- or 64-row multidetector CT was significantly higher than that of single-detector CT, especially in detecting small aneurysms of 4 mm in diameter [10]. A repeat angiogram may be necessary in 4 to 14 days if the initial angiogram is negative. The indications for repeating the angiogram include heavy blood load, small aneurysm size, and vasospasm, hematoma or thrombosis within the aneurysm [6••].

Reversible Cerebral Vasoconstriction Syndrome

Patients with RCVS typically present with sudden-onset, severe headache that is frequently recurrent, usually over the span of a week. There may or may not be associated neurological symptoms [11]. RCVS can occur in the setting of migraine, in the post-partum period, and with vasoactive medications and from drugs such as marijuana and cocaine. Brain MRI is normal in 50 % of cases [12]. Abnormal findings may develop later, revealing infarction in a watershed zone, SAH, or intracerebral hemorrhage (ICH). Edematous lesions, usually in posterior regions consistent with a posterior reversible encephalopathy syndrome (PRES)-like pattern may be seen. A MRA, CTA, or conventional angiography will reveal a

"string of beads" pattern of intracerebral arteries and their branches. The initial angiography may be negative, and a repeat study may be required in 4–5 days [6••]. These symptoms and imaging findings are reversible within days to weeks.

Cavernous Malformations

Headaches attributed to vascular malformations require that the headaches develop in close temporal relation to other symptoms and or clinical signs of vascular malformations. The headaches should improve after treatment of vascular malformations. Cavernous malformations may account for 10–15 % of intracranial and spinal vascular malformations. They are a compact mass of sinusoidal-type vessels without normal intervening brain. They appear hyperdense on CT, and on MRI as well-circumscribed masses with a rim of T2 hypointensity and marked gradient susceptibility signal on MRI [13] (Figs. 1, 2, and 3).

Cerebral Venous Sinus Thrombosis (CVST)

Headache is one of the most frequent symptoms of CVST with marked variability in the headache features. Headaches can be an isolated symptom, and may present as thunderclap or progressive headache. The headaches of CVST have no specific characteristics but are more often diffuse than localized. They can also be unilateral, usually ipsilateral to the occluded sinus. Headaches in CVST may mimic migraine, SAH, and cerebrospinal fluid (CSF) hypertension or hypotension [14, 15]. The associated symptoms include seizures, encephalopathy, and focal symptoms such as hemiparesis.

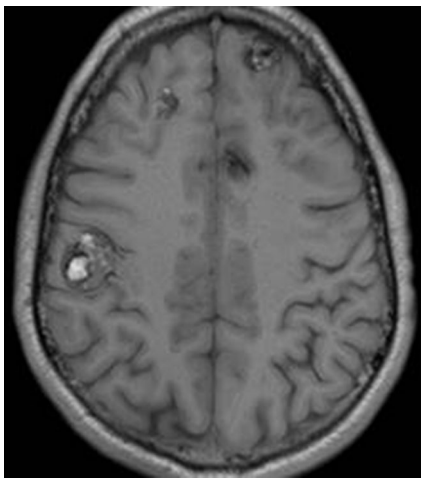

Fig. 1 A 26-year-old male with multiple cavernous malformations including prior hemorrhages. (Images courtesy of Lazlo Mechtler, MD). There are several heterogeneous areas of signal changes throughout the central nervous system parenchyma that exhibit mixed T1 hypointense and hyperintense foci

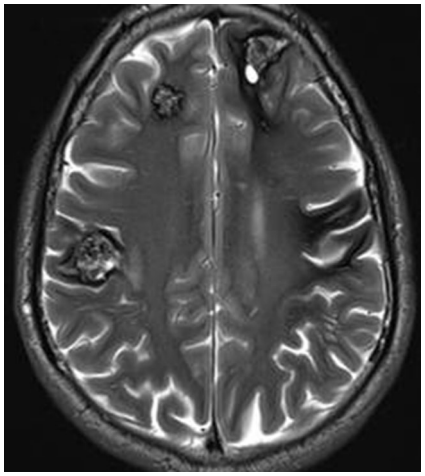

Fig. 2 A 26-year-old male with multiple cavernous malformations including prior hemorrhages. (Images courtesy of Lazlo Mechtler, MD). T2-weighted images show heterogeneous signal characteristics of the malformations

The hallmark empty delta sign on contrast-enhanced CT is seen in only 30 % of cases. Similarly, a fresh clot is visible on plain CT as a hyperdensity in superior sagittal sinus. Other findings include hemorrhagic infarcts or focal edema not respecting an arterial distribution. T2-weighted MR images seem to be superior to spin echo sequences in detecting CVST and small hemorrhages. Magnetic resonance venography (MRV) with phase contrast imaging is helpful for the diagnosis and follow-up of CVST (Fig. 4). However, MRV may not detect the thrombus, but show only absence of signal in the occluded vein [16]. Phase contrast angiography findings may be difficult to interpret due to normal anatomic variants such as sinus hypoplasia and flow asymmetry. MRI in early acute stages (1–5 days) shows an isointense signal on T1-weighted

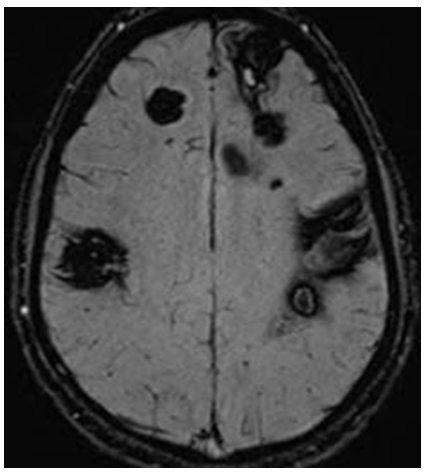

Fig. 3 A 26-year-old male with multiple cavernous malformations including prior hemorrhages. (Images courtesy of Lazlo Mechtler, MD). There are prominent hypointense blooming artifact on susceptibility-weighted images (Fig. 3)

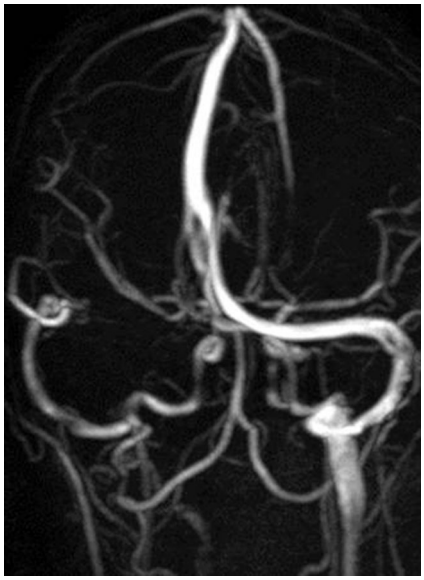

Fig. 4 A 23-year-old female experienced a severe right-sided headache 5 days after giving birth. On MR venogram, there is non-visualization of the right transverse sinus (*arrows*) consistent with venous sinus thrombosis. (Image courtesy of Lazlo Mechtler, MD)

sequence and hypointense signal on T2-weighted sequences of thrombosed sinuses. High-intensity signal on T1- and T2-weighted sequences is seen from 5 to 15 days. An isointense signal on T1-weighted sequence and high-intensity on T2-weighted sequence are present at later stages [17]. T2-weighted sequences allow visualization of hemorrhagic venous infarcts better than other sequences [18]. Acute arterial strokes and edema may be seen in diffusion-weighted images [16].

Cervical Carotid or Vertebral Artery Dissection

Cranial cervical dissection is the most frequent cause of strokes in young patients. Headache may be the only symptom and may occur without neck pain. Headaches are usually ipsilateral to the dissected vessel. They can be the first and only manifestation of internal carotid (ICA) or vertebral artery (VA) dissection. The mean duration of development of neurological symptoms from onset of headache was 4 days in ICA and 14.5 h in VA dissections in one review of 161 symptomatic patients [19]. Other common clinical manifestations include Horner's syndrome ipsilateral to a carotid dissection, cranial nerve palsies, or posterior circulation manifestations in vertebral dissection.

The preferred neuroimaging modalities include CTA and MRA. ICA dissection is characterized by a narrow eccentric lumen with increase in the external diameter of the artery. An intramural hematoma may be visualized on CTA. An intimal flap or a dissecting aneurysm may be present. VA dissection reveals an increased external diameter and crescent-shaped mural thickening in the VA.

The hematoma is isointense in the early and chronic stages. It will appear bright on T1-weighted images in the subacute stage (7 days–2 weeks).

Acutely, it is hard to visualize a dissection on T1-weighted images with fat saturation due to obscuration from the surrounding tissue. In the subacute stage, the dissection appears as a crescent-shaped hyperintensity around an eccentric flow void corresponding to the vessel lumen. MRA has poor sensitivity in diagnosing VA dissection and this modality alone may be misleading if negative. However, a contrast-enhanced MRA may show signal loss within the VA. DSA is considered the gold standard and may demonstrate a "string sign" (described as long-tapered, irregular stenosis) or "flame sign" (tapered occlusion that spares the carotid bulb) [20].

Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencepahlopathy (CADASIL) and Mitochondrial Encephalopathy, Lactic Acidosis, and Stroke-Like Episodes (MELAS)

CADASIL is an autosomal dominant arteriopathy characterized by subcortical dementia, ischemic events, and migraine with aura (MA). MA often presents early, at a mean age of 30, and may precede stroke onset by 15 years. The aura symptoms are visual or sensory, brainstem, hemiplegic, or prolonged. MA may improve when other neurological symptoms develop [21].

T2-weighted and FLAIR MRI sequences show large, confluent white matter hyperintensities, which are predominant in the anterior temporal and superior frontal white matter (Fig. 5). Lesions extend into arcuate fibers in the temporopolar and paramedian superior frontal lobes [22]. Subcortical

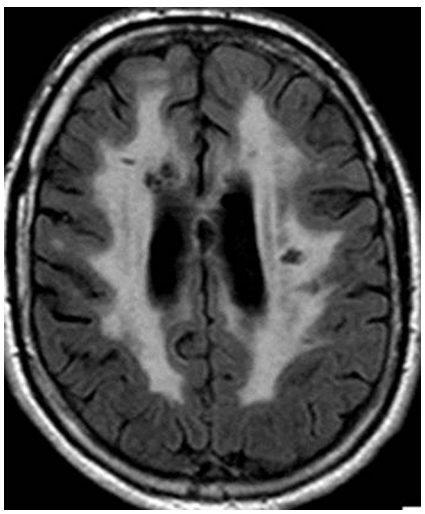

Fig. 5 A 52-year-old man with presenile dementia and migraines has typical imaging findings of CADASIL. (Images courtesy of Lazlo Mechtler, MD). FLAIR axial imaging demonstrates widespread confluent white matter hyper intensities and lacunar infarcts

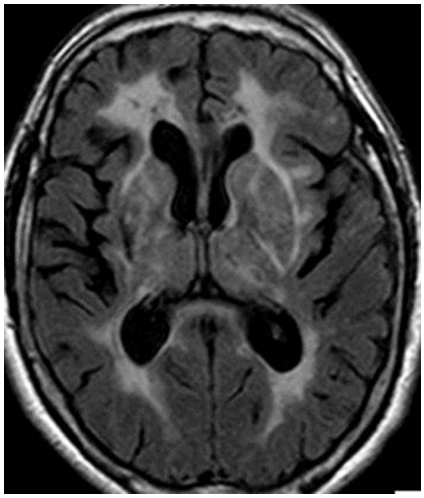

Fig. 6 A 52-year-old man with presenile dementia and migraines has typical imaging findings of CADASIL. (Images courtesy of Lazlo Mechtler, MD). More circumscribed hyper intense lesions are also seen in the basal ganglia, thalamus, and pons

infarcts and cerebral microbleeds may also be detected on MRI (Fig. 6).

MELAS is a mitochondrial disorder that presents in childhood (3 months–12 years). Patients experience various neurological manifestations such as seizures, hemiparesis, hemianopia, and cortical blindness. Migraine headaches are common and recurrent. MRI may show multiple areas of infarction with predilection for the parieto-occipital areas, and progression to generalized atrophy [23].

Pituitary Apoplexy

Pituitary apoplexy results from acute hemorrhage, infarction, or both in a pre-existing pituitary adenoma. The clinical syndrome is characterized by sudden headache, vomiting, visual impairment, and meningismus caused by accumulation of blood and edema leading to compression of vessels and surrounding structures. Sheehan syndrome should be distinguished from pituitary apoplexy. Sheehan syndrome is commonly seen after excessive postpartum bleeding and a prolonged period of hypotension, leading to hemorrhagic infarction in a normal pituitary gland [24].

MRI is the investigation of choice. A non-contrast head CT may not be sufficient to detect pituitary apoplexy. A CT specifically looking at the sella turcica may show evidence of hemorrhage as hyperdensity. CT may not differentiate between cystic or degenerative changes from a previous hemorrhage. CT also becomes less specific in chronic and subacute stages. MRI of the sellar region is more specific, and the preferred neuroimaging study. The appearance of hemorrhage varies based on the degradation of hemoglobin. In the hyperacute stages (1 to 2 h), the tumor may be isointense on T1-weighted image and hypointense on T2-weighted images. In acute phase (1–2 days), hemorrhage is hyperintense on T1-weighted images and hypointense on T2-weighted images. In subacute phase (3–15 days), there is hyperintensity on T1- and T2-weighted images. A fluid level may be noted within the hemorrhagic region in chronic stages (>15 days). T2*-weighted gradient echo imaging is the most sensitive sequence for detection of intracranial hemorrhage. Contrasted T1-weighted images are not helpful, as the hyperintensity may be mistaken for contrast-enhanced tumor [25–27].

Headaches Attributed to non-Vascular Intracranial Disorders

Spontaneous Intracranial Hypotension (SIH)

Headaches which worsen when upright and improve in the supine position may result from low CSF pressure. However, it is not unusual for postural component to be absent or headaches to evolve into a more chronic daily headache pattern. There may be associated symptoms such as hearing loss, tinnitus, visual blurring, and neck or interscapular pain [28••].

Head CT serves little diagnostic value although subdural fluid collections or increased tentorial enhancement may be seen. Brain MRI with gadolinium classically reveals diffuse pachymeningeal enhancement which is often seen as uninterrupted, non-nodular enhancement (Fig. 7). Engorgement and enhancement of cerebral venous sinuses and the pituitary gland are commonly present. Coronal and sagittal images may show descent of the cerebellar tonsils, flattening of optic chiasm, and decreased size of subarachnoid cisterns. Spine MRI abnormalities include extra-arachnoid fluid collections,

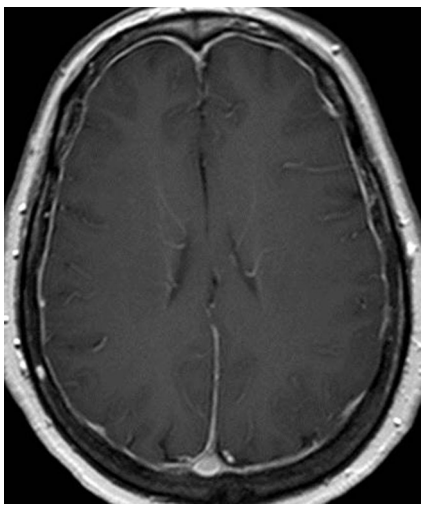

Fig. 7 Intracranial hypotension. A 51-year-old woman initially experienced orthostatic headaches that evolved into a constant, daily headache. Note the diffuse enhancement of the pachymeninges. (Image courtesy of Lazlo Mechtler, MD)

extra-dural extravasation of fluid, meningeal diverticula, spinal dural enhancement and engorgement of epidural venous plexus, and intradural spinal veins [28••, 29].

Patients with a high suspicion of intracranial hypotension are often treated presumptively with epidural blood patches prior to undergoing studies to identify the location of the leak. The preferred study for identifying the site of a spinal leak is debatable. CT myelography remains the gold standard. The procedure allows measurement of the CSF opening pressure (OP) and usually localizes the site of leak. However, there is considerable radiation exposure with this technique, approximately 105 mGy per one description of the technique for identifying high flow leaks [30]. The fast and slow flow leaks may be challenging to identify, as they may not be readily visible, and often need a thorough approach. Dynamic CT myelography or digital subtraction myelography may help to locate high flow leaks [30]. The yield of detecting slow flow leaks is facilitated by delayed CT after 3–4 h. Gadolium myelography (spine MRI with intrathecal contrast) and positive pressure myelography may be considered if the aforementioned studies are inconclusive, though reported results have been variable [28••, 29].

A diagnostic algorithm was proposed to detect suspected spinal CSF leaks [31]. The authors propose MRI brain with and without contrast and non-contrast MRI of the spine for the initial imaging studies. If they are negative, and clinical suspicion is high, MR myelography with intrathecal contrast is obtained. For positive initial studies, an epidural blood patch is recommended. However, if symptom relief remains inadequate after a second epidural blood patch, they recommend proceeding with additional imaging (digital subtraction or CT myelography) to localize the site of the leak.

Idiopathic Intracranial Hypertension (IIH)

The pseudotumor cerebri syndrome (PTCS) can arise as a primary disorder (IIH) or from secondary causes such as exogenous agents or venous sinus thrombosis. Patients often present with headaches, which are non-specific and may have features of migraine or tension-type headaches. The headaches may evolve into a chronic daily headache pattern [32••]. Other common symptoms include transient visual obscurations, pulsatile tinnitus, neck or radicular pain, and visual changes such as blurred vision and diplopia. The most common diagnostic sign is papilledema on the fundoscopic exam. Visual acuity and visual fields using perimetry need to be documented on these patients because of the potential for visual loss [32••]. A lumbar puncture opening pressure of at least 250 mm CSF supports the diagnosis of PTCS, along with papilledema and a normal neurological exam [33].

CT or MRI of the brain is required prior to performing the diagnostic lumbar puncture to rule out space occupying lesion. CT should be normal and without evidence of intracranial mass or hydrocephalus. MRI findings may help support the diagnosis, particularly in the absence of papilledema. Thin sections through the orbital and pituitary region may reveal distension of the perioptic nerve subarachnoid space, flattening of posterior aspect of the globe, and protrusion of the optic nerve head into the vitreous [34] (Fig. 8). Sagittal views frequently reveal an empty sella (Fig. 9) or tonsillar descent. If there is no evidence of papilledema but patients otherwise meet the diagnostic criteria, a diagnosis of PTCS can be suggested based on brain MRI findings of empty sella, flattening of posterior aspect of the globe, and distention of perioptic subarachnoid space with or without tortuous optic nerve [35, 36]. The lateral ventricle size is not affected in patients with IIH [37]. A MR venogram may show transverse sinus stenosis [36]. A venogram is indicted in atypical patients such as men, children, and patients on oral contraceptives, non-obese females or women over age 45 years to exclude venous sinus thrombosis as a secondary cause.

Colloid Cyst

The headaches arising from a colloid cyst of the third ventricle are often thunderclap and recurrent. There may be a reduced level or loss of consciousness. The headaches may improve in the supine position and are often located in the bilateral frontoparietal or frontooccipital regions. The cyst may cause obstructive hydrocephalus as it is located close to foramen of Monro (Fig. 10). There is no restricted diffusion and hyperintensity is seen on FLAIR sequences. On T1-weighted images,

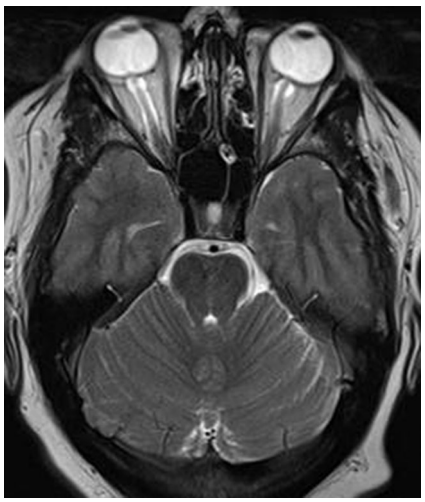

Fig. 8 Idiopathic Intracranial Hypertension in a 36-year-old female with history of headaches and papilledema. (Images courtesy of Lazlo Mechtler, MD). There are tortuous optic nerves with prominent optic nerve sheaths, and flattening of the posterior globes

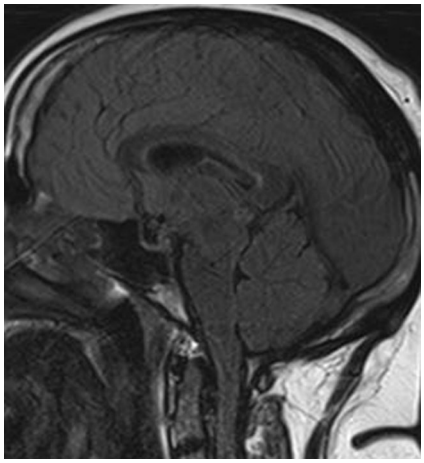

Fig. 9 Idiopathic Intracranial Hypertension in a 36-year-old female with history of headaches and papilledema. (Images courtesy of Lazlo Mechtler, MD). Sagittal T1-weighted imaging shows an empty sella and a crowded posterior fossa consistent with a diagnosis of idiopathic intracranial hypertension

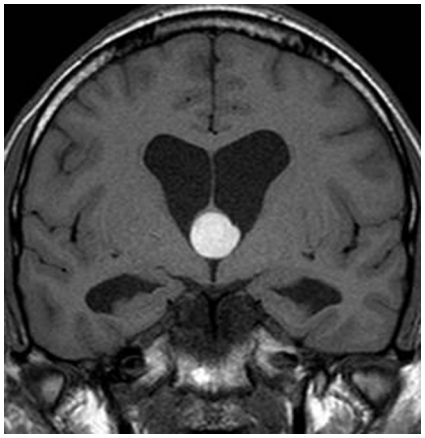

Fig. 11 Colloid cyst in a 23-year-old male with the insidious onset of headache. (Images courtesy of Lazlo Mechtler, MD). This same mass is hyperintense compared to brain on T1WI (B, *arrow*) reflecting cholesterol concentration. Colloid cysts generally do not enhance after contrast administration

the central portion of the mass is hyperintense, whereas the periphery is isointense (Fig. 11). The central portion is markedly hypointense, while the peripheral portion is isointense on T2-weighted images (Fig. 12) [38].

Chiari Malformation Type 1 (CM1)

CM1 headaches are often precipitated by cough or Valsalva maneuver. There may be associated symptoms of brainstem, cerebellar, or cervical cord dysfunction. The associated headaches are occipital or suboccipital, and generally last longer than primary cough headaches which last several seconds to a few minutes (although some patients experience mild to moderate headache for 2 h) [1]. MRI is required for the diagnosis to obtain detailed sagittal images. Brain MRI shows at least 5 mm of caudal descent of the cerebellar tonsils which appear "peglike" and pointed (Fig. 13). Other MRI findings include crowding of subarachnoid space at craniocervical junction and kinking of the medullary cervical junction and brainstem [39].

Tonsillar herniation of less than 5 mm does not exclude the diagnosis, if other features on brain MRI are present and patient is symptomatic. Asymptomatic tonsillar ectopia may be differentiated from symptomatic CM1 using CSF flow studies. An abnormal CSF flow pattern is seen in CM1 [40]. A spinal MRI is necessary to look for syringomyelia which may be seen in 40 % of the patients, commonly located between the C-4 and C-6 levels [41]. Tonsillar ectopia may also be caused by disorders producing high and low CSF pressure, including mass lesions, which are distinct from a true Chiari malformation (Fig. 14).

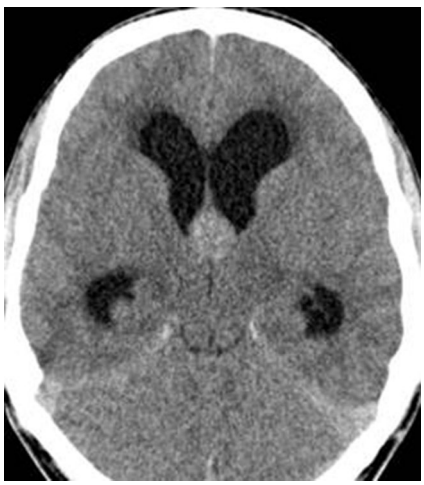

Fig. 10 Colloid cyst in a 23-year-old male with the insidious onset of headache. (Images courtesy of Lazlo Mechtler, MD). Non-enhanced CT demonstrates a spherical hyperdense mass (*arrow*) within the anterior third ventricle and ventriculomegaly

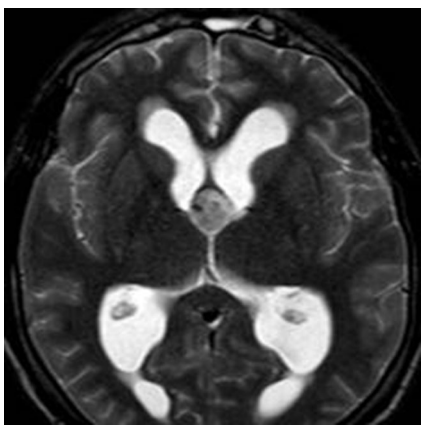

Fig. 12 Colloid cyst in a 23-year-old male with the insidious onset of headache. (Images courtesy of Lazlo Mechtler, MD). T2-weighted imaging reveals subependymal CSF extension into the brain parenchyma. The cyst demonstrates mixed hypo- and hyperintensity, the "black hole" effect

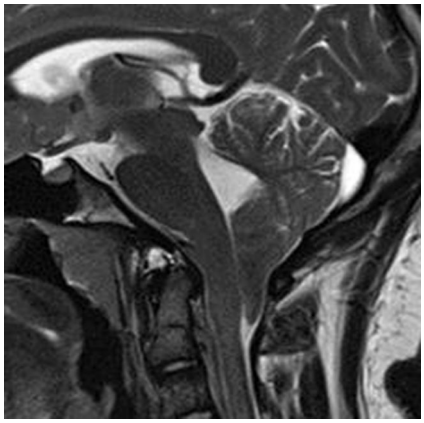

Fig. 13 Chiari 1 malformation in a 15-year-old female with a history of constant headaches. Low-lying peg-shaped cerebellar tonsils are seen lying below the foramen magnum, extending inferiorly approximately 24 mm to the level of the body of the axis. (Image courtesy of Lazlo Mechtler, MD)

Brain Tumor

Headaches caused by brain tumors have been classically described as severe, early morning or nocturnal headaches associated with nausea and vomiting. However, several studies have shown that headaches arising from brain tumors can present with the same phenotype as primary headache disorders such as migraine or tension-type headaches. Most patients have atypical features and only 17 % of patients fit the classic brain tumor headache description [42].

Headaches may occur ipsilateral to the tumor. Infratentorial tumors may cause occipital headaches [42–44]. Any headaches which are associated with red flags such as change in pattern, progression, associated neurological signs, systemic symptoms, precipitation with exertion, or onset after age 50 years should be further evaluated especially to rule out an intracranial mass. A space-occupying lesion may produce hydrocephalus and obstruction of CSF outflow, emphasizing the importance of fundoscopy to detect papilledema.

A head CT with contrast to rule out a space-occupying lesion or hydrocephalus is usually the first imaging modality. However, a contrast-enhanced MRI should be done especially to evaluate the meninges, posterior fossa, and pituitary region. The pattern and signal characteristics MRI may suggest the tumor type (Fig. 15). A contrast-enhanced study will also define enhancement and vascular distribution. For example, malignant gliomas may enhance heterogeneously on contrasted MRI. MR spectroscopy may help differentiate infiltrative from circumscribed tumors [45]. Functional MRI, positron emission tomography (PET), single photon emission CT (SPECT) and some other modalities which may help grade brain tumors, detect the metabolic rates, or differentiate tumor from surrounding edema.

Headaches Attributed to Infections

Brain Abscess

Brain abscess is focally encapsulated pus caused by various organisms. This can result from hematogenous dissemination, or local extension from a sinus, odontogenic, or otic source [46]. Headache is present in 69 % of patients along with fever and focal neurological signs. The classic triad of fever, headache, and focal neurological deficits is present in only 20 % of patients [47]. Vomiting and change in mental status may indicate raised intracranial pressure. A lumbar puncture could be obtained if there is no concern for raised intracranial pressure or significant mass effect. However, CSF examination is

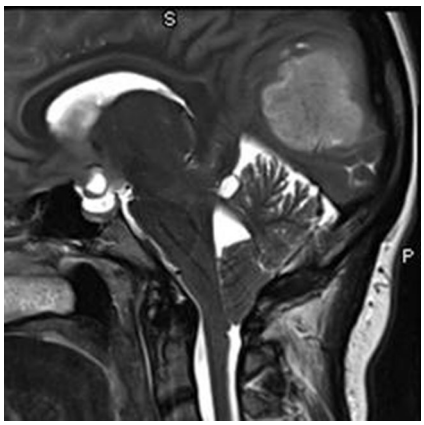

Fig. 14 Sagittal T2-weighted image shows tonsillar ectopia (*thin arrows*) resulting from mass effect produced by a meningioma. There is expansion of the chiasmatic recess of the third ventricles, exerting mass effect on the optic chiasm, which is thinned (*arrowheads*). (Image courtesy of Lazlo Mechtler, MD)

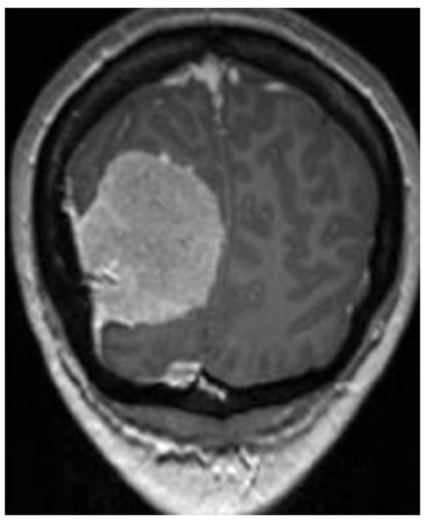

Fig. 15 Axial T1-weighted MRI with contrast shows a large right parieto-occipital meningioma with midline shift and a dural tail (*arrows*). (Image courtesy of Lazlo Mechtler, MD)

reported to be normal in 16 % of patients. CSF cultures are positive in 24 % of patients [47].

Brain abscess development can be divided in four stages. The first stage is early cerebritis (1–4 days) followed by late cerebritis at 4–10 days. Capsule formation is divided into early (11 to 14 days) and late (>14 days) stages [46].

CT scan is not as sensitive as MRI, with false negative results in 6 % of patients [47].However, CT can be obtained in the emergency setting and is useful to detect focal brain edema. A contrast-enhanced study may reveal early cerebritis with an irregular area of low-density and later ring enhancement in the capsule formation stages, secondary to mass effect [46, 48]. MRI is the preferred imaging study and should be performed with contrast. In the early phases, MRI reveals hypointensity on T1-weighted images and hyperintensity T2-weighted signal with patchy enhancement. In later stages, the hypointensity on T1-weighted images is better demarcated, and high signal intensity within the cavity and surrounding parenchyma is seen on T2-weighted images. The abscess cavity shows a hyperintense rim on non-contrasted T1-weighted images and a hypointense rim on T2-weighted image. Ring-enhancing lesions, satellite lesions, central necrosis, or cerebral edema are well visualized on a MRI. Thickness, irregularity, and nodularity of the enhancing ring are suggestive of tumor or fungal infection [49]. The lesions demonstrate restricted diffusion which may help differentiate an abscess from a necrotic tumor, which is hypointense or shows variable intensities on DWI [50]. MR spectroscopy (MRS) helps to differentiate brain abscess and other cystic lesions. There could be elevated amino acids, lactate, succinate, acetate, and alanine in untreated brain abscess or soon after initiation of treatment [46].

Headaches or Facial Pain Attributed to Disorders of the Cranium, Neck, Eyes, Ears, Nose, Sinuses, Teeth, Mouth, or Other Facial or Cervical Structures

Rhinosinusitis

The ICHD-3 beta has defined sinusitis-related headaches as headaches getting worse in parallel with worsening of rhinosinusitis [1]. There should be an improvement or resolution of headaches with improvement or resolution of rhinosinusitis. Headaches may be exacerbated by manual pressure applied over the paranasal sinuses. A CT scan of the sinuses may demonstrate mucosal thickening, air–fluid levels, thickening, or clouding. CT scan with contrast is indicated to rule out a suppurative process and intracranial infection. However, 42 % of asymptomatic individuals may have mucosal abnormalities on CT [51].

Sphenoid sinusitis presents differently and is potentially associated with significant mortality and morbidity. Headaches are a common symptom and may worsen with exertion. Symptoms frequently present in acute sinusitis such as nasal discharge, stuffiness, and post nasal drip are less common in sphenoid sinusitis. Other common symptoms include periorbital pain, pain in facial nerve distribution, photophobia, and lacrimation. Plain radiographs are the initial study but may be normal due to soft tissue overlap. If the clinical suspicion remains high, a CT scan or MRI should be obtained [52, 53] (Figs. 16 and 17).

Painful Cranial Neuropathies and Other Facial Pains

Trigeminal Neuralgia

Trigeminal Neuralgia (TN) is characterized by brief episodes of recurrent electric-like pain in the trigeminal nerve distribution, usually affecting the maxillary or mandibular division unilaterally. Classical trigeminal neuralgia is due to neurovascular compression by the superior cerebellar artery. Secondary etiologies include demyelinating disease and infiltration of the nerve root, Gasserian ganglion, or nerve by a tumor or amyloid, and small infarcts or angiomas in the brainstem. The recommendations to proceed with neuroimaging in classical TN are inconclusive, as vascular compression

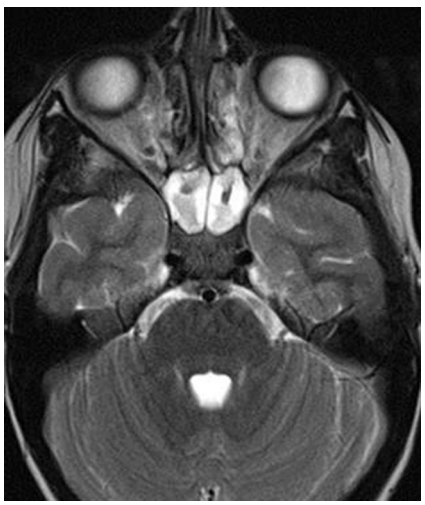

Fig. 16 A 7-year-old girl was evaluated for headaches that gradually increased in frequency and severity. Imaging revealed pansinusitis. (Images courtesy of Lazlo Mechtler, MD). There are chronic inflammatory changes in the sphenoid sinuses and ethmoid air cells (*arrows*)

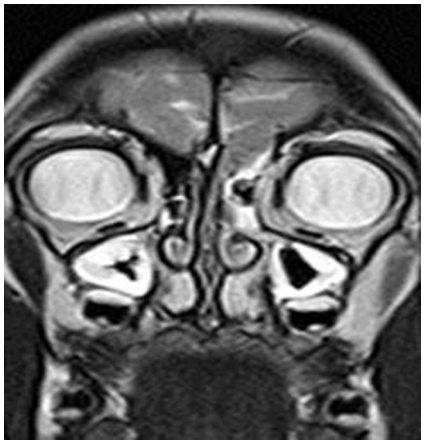

Fig. 17 A 7-year-old girl was evaluated for headaches that gradually increased in frequency and severity. Imaging revealed pansinusitis. (Images courtesy of Lazlo Mechtler, MD). Similar changes are present in the maxillary sinuses and ethmoid air cells (*arrows*)

of trigeminal nerve can be seen in normal individuals [54, 55]. A brain MRI will help to rule out structural lesions and secondary causes. The addition of gadolinium is useful to evaluate for demyelinating plaques and leptomeningeal disease. Further imaging techniques such as three-dimensional fast imaging employing steady-state acquisition (3-D-FIESTA) and constructive interference in steady state (CISS) magnetic resonance imaging, diffusion tensor imaging (DTI), fast inflow with steady-state procession (FISP), and constructive interference in steady state (CISS) have been used to identify neurovascular compression and evaluation of the trigeminal nerve root anatomy for pre-surgical planning [56–59].

Conclusion

A thorough history and neurological exam often help differentiates primary and secondary headache disorders. Neuroimaging is appropriate when there is a high clinical suspicion for a secondary etiology. Thunderclap and exertional should always be evaluated with imaging although these features can be present in patients with primary headache disorders. The diagnostic imaging modality with the highest yield is recommended, taking into account the potential risks of radiation exposure.

Compliance with Ethics Guidelines

Conflict of Interest Dr. Priyanka Chaudhry and Dr. Deborah I. Friedman each declare no potential conflicts of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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- •• Of major importance

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