Evaluation of Headaches in Urgent Care Part 1: Emergent Headaches

Urgent message: Identifying potentially life-threatening or emergent causes of headaches can be challenging in urgent care. Emergent headaches are the focus of the first of a two-part series, aimed at aiding practitioners in appropriate evaluation and management.

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Introduction

The presentation of headaches in the urgent care setting often elicits some healthy angst. Differentiating between potentially life-threatening headaches and more benign varieties can be challenging, especially when the signs and symptoms are often subtle and nonspecific. This article will highlight features associated with the emergent headache syndromes with which patients may more commonly present in the urgent care setting.

Eliciting emergent causes of headaches can be challenging. Some of the key history questions to focus on are: onset of the headache, location, severity, associated neurological symptoms, history of similar headaches and how *this* headache differs from prior headaches.

For the purpose of this article, emergent headaches are classified as those that threaten life or pose a substantial risk of permanent disability. The majority of patients with emergent headaches present directly to the emergency department (ED), but some do present in the urgent care setting. It is likely that the latter presentations are more subtle in presentation and/or early in the disease process, although that has not been well studied. It is vital that urgent care providers be vigilant in their assessment and include serious etiologies of headache in their differential diagnosis.

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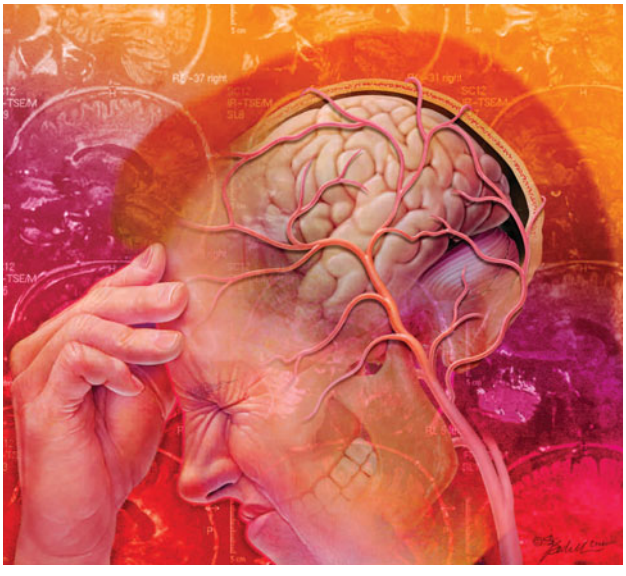

Intracranial Hemorrhage Headache

The classic presentation for an intracranial bleed (ICB) is abrupt onset of severe headache (97% of cases) with peak severity within seconds or minutes of onset (**Table 1**).[1,2]

Table 1. Pearls on Intracranial Hemorrhage-Related Headache

Symptoms
- Sudden onset.
- Maximum intensity reached within minutes.
- Often severe ("worst headache of life")
- Usually nausea or vomiting
- +/- neurological findings

Risk factors
- Positive family history of SAH
- Hypertension, poorly controlled
- Medications
  - Anticoagulants
  - Adrenergic (amphetamines, pseudoephedrine and phenylpropanolamine)

SAH = subarachnoid hemorrhage

Subarachnoid hemorrhage (SAH) often presents as an abrupt onset of severe pain – presenting with the classic "worst headache of my life" complaint, often with a "thunderclap" onset. The pain is usually lateralized in 30% of patients to the side of the bleed.[1] The headache may radiate into the neck and shoulders, causing meningismus.[3] Patients often complain of nausea with or without vomiting and may rapidly decline with mental status change or have a seizure; however, the signs can be subtle and 25% of patients with SAH are initially misdiagnosed especially in the face of sentinel bleeds.[4]

The majority of SAH are due to an aneurysm. Risk factors for SAH include a family history of a first- or second-degree relative with SAH. The probability of a SAH is significantly higher with a positive family history.[4]

The work up for suspected ICB includes a non-contrast computed tomography (CT) scan or contrast-enhanced magnetic resonance imaging (MRI) with or without a lumbar puncture (LP). An LP is indicated when there is suspicion of an acute SAH and neuroimaging is negative.[4] Few urgent care clinics can complete this evaluation and transfer to an ED is indicated.

The treatment for aneurysmal SAH may include various surgical interventions to stop active bleeding and decrease ICP and is considered to be the only effective treatment and it needs to be done within 24 to 72 hours; therefore, arranging prompt transfer to an ED is indicated.[5]

Sentinel headache, an unruptured intracranial aneurysm, is likely caused by leaking of blood into the subarachnoid space. Patients with these headaches often have the same presenting symptoms as with SAH, but the headache improves and may resolve within hours to days. It is estimated that 20% to 50% of those with a SAH report a sentinel or 'warning' headache days to weeks prior to an aneurysm rupture.[4]

Hypertensive vasculopathy is an important cause of nontraumatic ICB. Poorly controlled hypertension is the common chronic risk factor. There are many acute factors associated with hypertensive ICB, including hemorrhagic infarction, septic emboli, brain tumor, bleeding disorders including anticoagulant therapy, central nervous system infections, and vasculitis. Drug exposures associated with hypertensive ICG are adrenergic drugs (amphetamines and phenylpropanolamine) and pseudoephedrine, which directly stimulates alpha-adrenergic receptors causing vasoconstriction, and beta-adrenergic receptors, which cause increased heart rate and contractility, often increasing vascular strain.[6,7]

The clinical presentation differs slightly from SAH in that the neurological symptoms do not begin abruptly and are not maximal at the onset. Headache onset is often within minutes to a few hours and is frequently associated with symptoms of increased ICP and traction of the meningeal pain fibers, causing nausea with or without vomiting. Focal neurological findings are specific to the location of the bleed (that is, cerebellar bleeds present with inability to walk due to imbalance).

The etiology of the vasculopathy associated with this type of ICB is chronic hypertension leading to vessel wall damage, which in turn causes "pseudo aneurysms." The primary risk factor is hypertension, which is thought to cause "pseudoaneurysms" by way of damage to the intimal layer of the vessel wall, causing weakened areas and increasing the risk of a massive hemorrhage. Secondary risk factors, for vascular disease, include older age, exposure to antithrombic therapy, high alcohol intake, African-American ethnicity, lower cholesterol, lower LDL cholesterol, and low triglycerides.[8]

The diagnostic studies of choice include non-contrast CT scan or contrast-enhanced MRI.[9]

Treatments for hypertensive vasculopathy include both medical and surgical interventions tailored to the underlying etiology and severity of the ICB.[10]

Warfarin – associated ICB: These headaches present similarly to SAH and symptoms may include focal neurological signs such as hemiparesis, aphasia, and ataxia. These events usually occur in the older population, with a mean age of 70.[11] The suspicion rises with a history of head-related trauma, which may be trivial. The diagnosis of warfarin-associated ICH is met when neurological signs are present, the international normalized ratio (INR) is >1.4 and there is evidence of ICB on non-

contrast CT scan or MRI. The treatment includes infusions of vitamin K and fresh frozen plasma.[12]

Warfarin is the anticoagulant most represented in the literature; however, very similar complications have been associated with all currently available Novel Oral Anticoagulants (NOAC), including dabigatran, rivaroxaban, aoixaban, and edoxaba. In terms of bleeding-associated risks, these medications should be considered as warfarin-equivalent.

Brain Tumor Headache

Tumor-related headache is considered the great masquerader. It mimics common, non-emergent types of headaches including tension (77%), migraine (9%), and other types (14%).[13] The classic "brain tumor triad" of nocturnal or early-morning headache, nausea/vomiting and severe nature is now thought to be inaccurate based on current studies.[13]Although headache is a common symptom, it is rarely severe or the only symptom (**Table 2**). Nausea with or without vomiting is another common complaint occurring in 40% of patients.[13] The tumor-related headache is worse with bending forward (or other Valsalva-type maneuvers) and is thought to be more sensitive and specific then the nocturnal or early morning headache. The headaches are characterized as generalized, dull, constant, and may be throbbing.[13] Brain tumors are often associated with other neurological symptoms, such as seizure or focal weakness.[14]

Isolated headaches of more than 10 weeks' duration are rarely caused by a brain tumor.[15]

Risk factors for brain tumor include new onset of headaches in patients older than age 50 and comorbid malignancy, particularly of the lung, breast, melanoma, and gastrointestinal cancers.

The gold standard diagnostic test is MRI with and without intravenous contrast.

Treatment is specific to the tumor location, size, type and the patient's overall health and can include surgical interventions and palliative care when appropriate.

Idiopathic intracranial hypertension

Idiopathic Intracranial Hypertension (IIH), also known as pseudotumor cerebri, was previously known as benign intracranial hypertension but it is a serious disorder. IIH is caused by elevated ICP due to overproduction of cerebral spinal fluid (CSF) and often causes significant disability. Although this type of headache is not life-threatening, it can cause significant impairment if not identified and treated promptly. Without prompt diagnosis and treatment, patients may suffer intractable disabling headaches and are at risk of blindness.[16]

Table 2. Pearls on Brain Tumor-Related Headache

Headache:
- Alone is uncommon
- Often generalized
- Often associated with nausea/vomiting
- Worse with Valsalva or bending forward
- Less likely if present >10 wks

Table 3. Pearls on Idiopathic Intracranial Hypertension-Related Headache

- Headache and transient visual loss are common
- Papilledema is common
- Elevated opening pressure on LP
- Female > Male
- <50 years, obese
- Recent use of tretinoin or doxycycline

LP = lumbar puncture

The most common symptoms of IIH are headache (92%), transient visual loss (72%), pulsatile tinnitus (60%), photopsia (54%), retrobulbar pain (44%), diplopia (38%), and sustained visual loss (25%) (**Table 3**).[16] The headache is often lateralized, pulsatile, and worse in the supine position. The unique quality of IIH is retrobulbar pain and pain with eye movement or globe compression. Another IIH-specific symptom is dysacusis, which are often perceived as rushing water or wind and are pulse-synchronous.[17] Visual disturbances, such as transient loss, are caused by papilledema whereas diplopia is caused by sixth nerve palsy from increased ICP.[18]

Physical exam findings characteristic of IIH are papilledema (bilateral and symmetrical), which is the hallmark sign, visual field loss, and sixth nerve palsy.

Risk factors for IIH are being female, obese, and using tretinoin and/or doxycycline. Most cases occur in patients younger than age 50.

The diagnostics of choice for IIH are MRI: to exclude secondary cause of elevated IPC, lumbar puncture to reveal elevated opening pressure, and visual field testing to reveal an enlarged blind spot, generalized constriction, and inferonasal vision loss.

The goals of treatment for IIH are recognition and appropriate referral. Initial referral will likely be to an ED unless urgent coordinated care with a neurologist is readily available. Treatment is aimed at resolution of symptoms and preservation of vision. Options for treatment are weight loss, carbonic anhydrase inhibitors, loop diuretics, serial lumbar punctures, and surgical

Table 4. Pearls on Giant Cell Arteritis-Associated Headache

- Temporal headache: often new or different
- Scalp tenderness and jaw claudication
- Age >50 years
- Associated with PMR
- CRP and ESR elevated
- Treatment: glucocorticoids and aspirin

CRP = C-reactive protein; ESR = erythrocyte sedimentation rate; PMR = polymyalgia rheumatica

intervention.[20,21] Carbonic anhydrase inhibitors are thought to reduce the production of CSF. First-line treatment is acetazolamide 500 mg twice daily. Caution should be used in any patient with a sulfa allergy. Furosemide (20 mg to 40 mg per day) may be helpful as an adjunct to carbonic anhydrase inhibitors. This combination has been shown to normalize CSF pressure within 6 weeks in many patients. Serial lumbar punctures are generally *not* recommended, but can be considered in patients who do not tolerate medication therapy or in pregnant women.

Surgery should be considered only in patients whose IIH has failed to respond to all other measures and who have intractable pain and loss of visual acuity loss. The procedures include ventriculoperitoneal shunt or lumboperitoneal shunt and / or optic nerve sheath fenestration.

Giant Cell (Temporal) Arteritis Headache

Giant Cell Arteritis (GCA) is a chronic vasculitis of medium and large vessels and can cause permanent vision loss, which occurs in 15% to 20% of patients.[22] Although GCA rarely requires an ED evaluation, prompt assessment, treatment, and referrals are critical in reducing the risk of significant and permanent impairment, namely vision loss.

The most common symptom of GCA is new onset or new type of headache that is often temporally located, but can be more generalized (**Table 4**). Headache is the most common complaint, with the critical feature being new onset (or new type) of headache. There are often associated symptoms with GCA, including systemic complaints such as low-grade fever, anorexia, weight loss, malaise, fatigue, paresthesias, joint pain, dizziness, hoarseness and dysphagia. The most common associated symptom is jaw claudication. It occurs in more than 50% of patients with GCA and, with profound fatigue upon mastication, is the most specific symptom correlated to a confirmed diagnosis of GCA.[23] The headache is often burning with or without episodes of lancinating pain. Scalp tenderness is seen in about 50% of patients, who may describe pain with brushing their hair.[24] The headache often progressively worsens over time until treatment is initiated. Visual symptoms are not uncommon with GCA and may present as amaurosis fugax. A non-productive cough is associated with GCA in 10% of patients and is a result of vasculitis near the cough receptors.[25]

The characteristic physical examination qualities may include tender and enlarged temporal artery and the pulse may be absent.

Risk factors for GCA include advanced age. The disorder is almost never seen in patients younger than age 50, with a mean age of 72 years at diagnosis.[26] Comorbid polymyalgia rheumatica is closely associated with GCA, occurring in one-half of patients.[27]

The gold standard of diagnosis is temporal artery biopsy, which should be performed within 3 days of presentation. However, biopsy only carries an 87% sensitivity, making adjunct diagnostics studies valuable.[28] In addition to biopsy, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP) and complete blood count (CBC) are recommended. The ESR is elevated in 85% of patients at a value >50 mm/hour.[29,30] CRP has a sensitivity of 97.5% to 100% for active GCA and is more sensitive than biopsy with less risk. It is less influenced by factors such as age than is ESR and recognition of it as an important diagnostic test is growing.[29,30] ESR does show age-related elevation after age 40 years, whereas CRP is also affected by age, but to a much lesser extent.[31] The characteristic abnormality seen on CBC with GCA is thrombocytosis.[29,30]

Treatment for GCA should not be delayed while awaiting biopsy results and should be initiated as soon as the diagnosis is entertained. The recommended treatment is glucocorticoids and the initial dose should be equivalent to 40 mg to 60 mg of prednisone as a single dose per day until symptoms improve.[32] In addition to glucocorticoid therapy, low-dose aspirin (80 mg-100 mg/day) is recommended to reduce the risk of blindness.[33] Because of the increased risk of gastroduodenal complications with steroids and NSAIDs, especially when combined, and given the duration of treatment, beginning a proton pump inhibitor is highly recommended with the above therapy.

The complications of GCA include blindness. Estimates indicate that up to 50% of patients who present with unilateral visual loss or diplopia may have progression to bilateral blindness within 1 to 2 months if GCA is left untreated.[34]

Conclusion

Rapid recognition of an emergent type of headache is essen-

tial to coordinate a patient's care urgently. Identifying key components of an emergent type of headache, such as onset and severity (sudden and severe); exacerbating factors (movement), associated symptoms (vomiting, focal neurological and / or visual disturbances) is paramount. The complaint of a new or different type of headache should raise concern and warrants a detailed evaluation. For patients whose headaches have a life-threatening cause, transfer to an appropriate ED is required. Prompt recognition of a potentially disabling cause of headache is equally important to reduce the risk of permanent functional loss. ■

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