

Resident and Fellow Section

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TEACHING CASE—HEADACHE IN THE EMERGENCY ROOM: A REVIEW

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A 37-year-old man presented to the emergency room (ER) at 3 AM. The prior evening he had had the sudden onset of “the worst headache of my life” with nausea, vomiting, and diarrhea. As he had a prior history of migraine, he did not seek immediate medical attention. Matters did not improve, so he eventually went to the ER. He had a history of (treated) hypertension, meningitis “many years ago,” and a history of craniofacial trauma in 1996 with left maxillary sinus fracture, nasal ethmoid vomer fracture, and left orbital fracture. At 4 AM, he was drowsy and noted to have a fever of 39.9°C pr (103.8°F). He subsequently became unresponsive. Computerized tomography (CT) of the brain without contrast was normal. Lumbar puncture revealed 15,290 white blood cells (WBCs)/mL (86% PMNs), 205 red blood cells/mL, protein of 516 mg% (normal <46 mg%), glucose 3 mg%. Cerebrospinal fluid (CSF) Gram stain revealed Gram+diplococci. Peripheral WBC was 17,000 and chest x-ray revealed a left lower lobe pneumonia. Rapid treatment with antibiotics resulted in a complete cure and he left the hospital after 6 days.

Broadly speaking, the first order of business in diagnosing headache is to determine if the headache is primary (eg, migraine, tension-type, cluster) or secondary and therefore due to an underlying cause. The next step is to make a specific diagnosis as headache is a symptom rather than a specific diagnosis. Treatment is then directed against the underlying cause if one is found, or against the pain and associated symptoms of the primary headache.

Assessing the patient with headache in the ER is a special circumstance as the patients are often not known to the physician and may not be seen again. Rapid, accurate diagnosis is essential as is making appropriate plans for follow-up. Most patients, approximately two-thirds,¹ will have primary headache, usually migraine or variants of migraine. However, having a history of migraine in no way “inoculates” the patient from also developing a secondary headache, as illustrated by our case. One must work effi-

ciently to make a diagnosis, and keep an open mind. History is often crucial to making the correct diagnosis and a thorough examination will also help in the selection of any testing.

Several caveats may be useful to remember:

1. Primary headaches are not associated with fever, so in the febrile patient an explanation must be sought.
2. Pain from a headache may elevate blood pressure, but a rising blood pressure and a falling pulse (Cushing’s reflex) suggest an expanding intracranial mass.
3. Signs of elevated intracranial pressure include papilledema (which may take hours to develop or may never develop), bilateral sixth nerve palsies, and diminished upgaze.
4. Meningeal irritation manifested as Brudzinski’s and/or Kernig’s signs may be seen in meningitis or subarachnoid hemorrhage (SAH) but are often lost in deep coma.
5. Additionally, any new or worsening headache in a patient ≥ 50 years old should arouse at least suspicion of giant cell arteritis.

There are several common scenarios that lead patients into the ER. The clinician should ask why has the patient come to the ER now, has the patient had similar headaches before, are there new or unusual features accompanying this headache, has there been any recent head trauma or other new illness/new medication, and note if the patient is improving or worsening under observation. The exact nature of the onset of the current headache should be ascertained (sudden or gradual onset), as well as the setting of onset (ie, during exertion, at orgasm, was there a loss of consciousness?, etc) and the presence of nausea or vomiting should be established. A general and neurologic examination including vital signs should be performed and any abnormality, including the mental status examination, should arouse suspicion that a secondary cause may be present.²

We will address several of the types of presentations of headache in the ER, including headache with fever, thunderclap headache, headache with transient neurologic deficit, and headache with deteriorating neurologic status.

HEADACHE WITH FEVER

Fever is not an expected feature of primary headaches. When encountered, it should lead to a search for a systemic

Table 1.—Causes of (Secondary) Thunderclap Headache

Subarachnoid hemorrhage
Cerebral venous sinus thrombosis
Arterial dissection
Pituitary apoplexy
Hypertensive encephalopathy
Intracerebral hemorrhage
Retroclival hematoma
Obstructive hydrocephalus
Intracranial hypotension
“Crash migraine”
Sphenoid sinusitis
Reversible cerebral vasoconstriction syndrome
Cardiac cephalgia

or central nervous system (CNS) infection or intracranial hemorrhage. Headache is a frequent consequence of systemic infection, presumably due to the effects of interleukin-1 and a nitric oxide mechanism. If there is any abnormality on the neurologic examination, prompt neuroimaging to exclude a CNS mass lesion such as an abscess should occur and, if negative, a lumbar puncture with opening pressure should then be performed to evaluate for signs of infection and/or hemorrhage. CT imaging is generally readily available and will identify hemorrhage (which can cause a low-grade fever) and some infectious etiologies such as an abscess. However, magnetic resonance imaging (MRI) is indicated in some situations as it is much more sensitive with regard to identifying the changes that occur with some infections (eg, herpes simplex encephalitis).

THUNDERCLAP HEADACHE

Headache of very sudden onset, also known as “thunderclap headache,” is a common ER headache presentation. The differential is broad, and includes the benign primary form. However, the International Classification of Headache Disorders, second edition states that the evidence supporting primary thunderclap headache is poor (ie, most if not all thunderclap headache is secondary). Many of these etiologies are life-threatening and testing is essentially always necessary (Table 1). SAH is one of the major considerations,³ but other causes include: cerebral venous sinus thrombosis, vertebral or carotid arterial dissection, intracerebral hemorrhage, hypertensive encephalopathy, obstructive hydrocephalus, intracranial hypotension, “crash” migraine, sphenoid sinusitis, cerebral vasculitis, reversible cerebral vasoconstrictive syndrome,⁴ and cardiac cephalgia.

HEADACHE WITH TRANSIENT NEUROLOGICAL DEFICIT

Headache with a concomitant transient neurologic deficit may cause diagnostic confusion in the emergency

department setting. Migraine aura may manifest with a reversible neurologic deficit including visual changes. Less often described are sensory and/or motor deficits, or even aphasia. These symptoms usually last less than 1 hour and may overlap with the time course of a transient ischemic attack (TIA). Confusion arises most often in the middle-aged or older patient with who may or may not have a headache history but does have cardiovascular risk factors for TIA and stroke. Fisher called these events “late life migraine accompaniments.”⁵ In middle-aged or elderly patients with new-onset transient deficits associated with headache, it may be most prudent to approach the situation as a TIA given the debility that a missed stroke may incur. Various clinical features of the attacks may help identify the diagnosis but there is a fair amount of overlap and a conservative approach is recommended (Table 2). Headache and TIA symptoms may also occur in the setting of arterial dissection and this constellation of symptoms should prompt questions about head or neck trauma, particularly in a younger patient. Epilepsy also may enter into the differential diagnosis in some situations.

HEADACHE WITH NEUROLOGICAL DETERIORATION

It should be noted that observing the patient over time to look for improvement or worsening can sometimes be misleading. The patient with a “sentinel hemorrhage” from a leaking aneurysm may have a sudden and severe headache with subsequent improvement. The same may occur in a patient with an epidural hematoma; these patients sustain a head injury often significant enough to cause a concussion. They may then improve and regain consciousness in the so-called “lucid interval” only to subsequently deteriorate a second time as the hematoma expands. In both situations, history is crucial and should lead to further investigation.

Lastly, we should mention testing. Testing is chosen based on the history and examination. CT scanning is widely available and can often be obtained rapidly. On the day of the ictus, it will detect 90-95% of SAHs but becomes less sensitive as time passes. Once a CNS mass lesion is excluded, lumbar puncture is an important test and, when performed, should include an opening pressure. It can reveal evidence of infection, hemorrhage, and high or low intracranial pressure. Further testing depends on what modalities are available, but may include computed tomographic angiography/venography and magnetic resonance imaging/angiography/venography when clinically appropriate.

However, it should be commented that in cases of suspected bacterial meningitis when CT imaging is not available within a short window of time, the practitioner should consider moving forward with a lumbar puncture followed by antibiotics (or straight to antibiotics) when the clinical suspicion of meningitis is high. This is justified given the morbidity and mortality associated with fulminant bacterial meningitis.

Table 2.—Features to Help Differentiate Migraine Aura From TIA or Seizure

Migraine	TIA	Epilepsy
Gradual onset of neurological symptoms usually precedes headache and the headache is generally the primary complaint, may have clear precipitant	Sudden onset of symptoms which are maximal at onset and then resolve slowly within (generally) 1 hour Headache is not usually primary complaint	Onset may be sudden Headache is common postictally
May have march of symptoms from 1 part of a body to another over minutes (cheiro-oral ascent of paresthesias) Hemi-body symptoms uncommon	Symptoms should not march from one body part to another and usually reflect dysfunction in a single vascular territory	May have march of symptoms (more rapid than for migraine aura)—“Jacksonian spread”
Visual symptoms (usually positive) which last minutes: scintillating scotoma, sparkles, or shimmering. Fortification spectra or lightening bolts	Loss of function (negative symptoms): weakness/heaviness that is 1-sided, visual loss in 1 or both eyes, dysarthria, receptive/expressive aphasia	Positive symptoms more likely than negative Todd’s paralysis may be present postictally but is uncommon
Loss of consciousness unusual Associated nausea, vomiting, photo/phonophobia generally lasting hours	Loss of consciousness unusual	Loss of consciousness common Associated gastric aura/tastes/or odors last seconds Photo/phonophobia uncommon

TIA = transient ischemic attack.

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