



## **New Onset Headache: Diagnosis and Management**

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#### **ED Visit One**

A 20 year old Hispanic woman presented to the ED with a complaint of a severe headache present for three days. The headache woke her. Since that time, it has been intermittent, throbbing, and unresponsive to over the counter medications. The headache began at the vertex of her head and moved to the occiput. It also radiates down her neck. It seems to get slightly worse if she touches her scalp. She has never had a headache before. She denies photophobia, fevers, trauma, visual changes, or upper respiratory track symptoms. She vomited once because of the pain. In the ED, she is afebrile, with a blood pressure of 114/68 and a HR= 76. She is in NAD. Her general physical examination is normal, PERRLA, and her fundi are normal. Her neck is supple. Her neurologic exam is normal. She is given droperidol, 2.5mg IM and her headache is relieved completely at 30 minutes. She does complain of some increased neck stiffness, which is thought to be a dystonic reaction to the droperidol. Her symptoms are relieved with diphenhydramine, 50 mg IM. She is diagnosed with a benign headache, tension vs migraine vs other vascular. She is sent home with ibuprofen and no specific follow up care.

#### **ED Visit Two**

One week later, paramedics are called to her home. Because the paramedics do not speak Spanish, they have trouble getting a history from family members. However, her husband speaks a little English and relates that the patient woke this morning with a headache, and became progressively more lethargic until he could no longer rouse her. The paramedics have been called to this house before for drug related problems, but the husband adamantly states that the patient has not used illicit drugs for several days. Paramedics find her unresponsive, with shallow respirations. Her BP = 110/60, HR= 110. RR= 12, shallow. She has no signs of trauma. Her blood sugar is 115. An IV is started and she is given naloxone by standard protocol for unresponsive patients in the field. She seems to become slightly more responsive after naloxone, so the paramedics think she has overdosed, and call the hospital with that information. She is given bag – mask ventilation and transported to the Stabilization Room.

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### **Diagnosis**

#### *Headaches*

According to the International Headache Society, the first episode of a severe headache cannot be classified as migraine or tension – type; these diagnoses require multiple episodes of pain with specific characteristics (i.e. nine episodes for the diagnosis of tension headaches and four for migraine). The first headache therefore requires specific evaluation, at the discretion of the primary physician. If the first headache is particularly severe or has neurologic findings associated with it, most physicians will proceed with an extensive evaluation to rule out life threatening or structural causes of headache. Changes in established headache patterns also require evaluation.

#### *Differential Diagnosis- New Onset Headache*

Primary headache disorders are eventually established as the cause of headaches in most patients. In most circumstances, these are annoying but not life threatening. Benign headache types include migraine, other vascular, or tension headache. Since the emergency physician cannot follow the longitudinal pattern or frequency of the patient's headaches, it is impossible to establish these diagnoses when the patient presents to the ED with a first time headache. The responsibility of the EP is to rule out life threatening causes of headache, provide prompt effective pain management and other indicated treatment, appropriately use resources in the evaluation of the headache, and arrange early and easy follow up.

A review of the medical literature has described the final diagnoses of undifferentiated headaches presenting to the ED as non-intracranial infection (39%), tension headache (19%), miscellaneous causes (15%), post-traumatic (9%), hypertension related (5%), vascular/ migraine (4%), no diagnoses (6%), subarachnoid hemorrhage (0.9%) and meningitis (0.6%) (1).

#### *Differential Diagnosis- Serious Causes of Headache*

It is often difficult to differentiate serious from benign headaches when the patient is experiencing a severe first time headache. Certain characteristics will help risk - stratify the patient and can help direct the ED work up and treatment of these patients. However, caution should be exercised when considering risk stratifying patients; no established clinical pathway is fool proof, and physician complacency is perhaps the highest risk to the patient.

High risk characteristics for first time headaches include (but are not limited to) headaches occurring in patients over 40 years old, associated with exertion, female gender, hypertension, cigarette smoking, presenting with memory impairment, ataxia, drowsiness, sensory loss or signs of meningeal irritation. Other high-risk characteristics include fever, photophobia, tender pulsatile temporal arteries, progressive visual changes, confusion, weakness, loss of coordination, papillary asymmetry.

*Differential Diagnosis – Sudden Onset Worst – Ever Headache*

SAH are thought to be responsible for 12-33% of headaches in patients with normal neurologic exams but who describe the headache as the “worst headache of their lives” (2). If the neurologic exam is abnormal, this increases to at least 25%. The presentation and neurologic findings may be confusing or subtle. For instance, 34% of SAH occur during exertion; 12% occur during sleep (3). The headache may be mild, in any location, and relieved by non-narcotic analgesia. However, about 85% of patients with a SAH report an excruciating headache, and almost 50% of patients with SAH present with at least a brief period of alteration of consciousness, including syncope, confusion, seizure, or coma. Nausea and vomiting are also prominent features. Neck stiffness is described in 15% of patients.

It is estimated that there are 33,000 cases of SAH per year, with overall incidence of 13/100,00. The causes of non-traumatic SAH are Aneurysm (51%), hypertension (15%), arteriovenous malformation (AVM) in 6% and other miscellaneous causes. The mortality of untreated SAH is over 50% with up to 20% of deaths occurring in the first day. The initial bleed can be fatal, and is described as a “thunderclap” headache in 20-50% of patients with SAH. Many patients (between 15-40%) have warning headaches (“sentinel bleed”), occurring days to weeks before the index episode of bleeding. This is thought to be due to a limited leakage of blood from an aneurysm (4).

The thunderclap headache is described as an acute headache with peak intensity at its onset. It develops in seconds, achieves maximum intensity within a few minutes, and may last for hours to days (5). Acute expansion, dissection or thrombosis of an unruptured aneurysm, and cerebral venous sinus thrombosis can also cause a thunderclap headache.

**Evaluation**

Clues to establishing the cause of the headache include a careful physical examination, the age of the patient, co-morbid factors, the location of the headache, the onset ( abrupt vs gradual), pain characteristics, associated symptoms, the duration, prior medical history, and appropriately chosen diagnostic tests.

*Diagnostic Evaluation – CT scanning*

The first diagnostic test for establishing the diagnosis of SAH is non-contrast thin cut ( 3 mm ) computed axial tomography ( CT ) scanning. Magnetic resonance imaging can detect aneurysms, but CT scans are better at detecting acute hemorrhage, and are usually more available, quicker, less costly and more convenient when monitoring potentially unstable patients. The ability to detect SAH on CT depends on a number of factors, such as the type of scanner, the time since bleeding, the patient’s hemoglobin concentration, the viewing physician’s experience (neuroradiologist, radiologist, EP) and the size of the bleed.

One prospective study of outpatients found a sensitivity of third generation CT scanning of 98% (117 of 119 patients with SAH) if scanning is done within the first 12 hours after the bleed. This decreases to 93% if the scan is performed within the first 24 hours (6, 7).

### *Lumbar Puncture*

Obviously if the CT scan is positive for blood, the ED work up is complete. However, a diagnostic dilemma occasionally arises when the index of suspicion is high but the CT scan is negative. In general, the literature supports the performance of a lumbar puncture (LP) in these cases, but in practice, this recommendation is not always followed. Because the CT scan may miss a number of SAHs, the stroke council of the American Heart Association advises the performance of an LP in high-risk cases where the CT scan is negative, equivocal, or technically inadequate (8). The safety of an LP first in suspicious cases has not been prospectively studied. However, mathematical modeling has predicted that LPs can safely be performed without a CT in selected individuals presenting within 12 hours of the bleed, thus reducing the time to definitive diagnosis and the cost of the work up (9).

*Traumatic taps:* Blood in the CSF can occur as a result of traumatic taps, which may occur in up to 20 % of cases. Methods to detect this clinically (i.e. clinician impression, decreasing number of RBCs in successive tubes of CSF, creanated RBCs ) are not fool proof in identifying a traumatic tap ( 10 ). Options to rule out a traumatic tap include a second tap at a higher interspace or after a few hours, a repeat CT after a few hours, or an angiogram.

*Xanthochromia:* Blood cells released into the subarachnoid space gradually lyse and released hemoglobin is metabolized to oxyhemoglobin and bilirubin , resulting in xanthochromia. Oxyhemoglobin (pink) is detectable within hours, whereas bilirubin (yellow) takes up to 12 hours to be detectable. Timing is therefore important in interpreting the results of the CSF analysis. Xanthochromia is considered by most experts to be a criterion for the diagnosis of SAH in patients with negative CT scans (10) if the detection is done by spectrophotometry. Visual inspection of the CSF reveals RBCs but may miss discoloration due to xanthochromia.

## **Management**

### *Emergency Department*

Misdiagnosis of SAH is usually due to failure to appreciate the spectrum of presentation, failure to understand the limitations of the CT and failure to perform and correctly interpret the results of a lumbar puncture.

In the ED, the patient should undergo stabilization, attending to the ABCs as for all critical patients. If the patient is extremely hypertensive, blood pressure should be carefully lowered and controlled, but to levels no lower than 140-160 systolic. Approximately one third of patients with SAH will develop delayed ischemic deficit within the first few hours and up to 4 days after the bleed due to vasospasm. Nimodipine 60 mg, should be given as soon as feasible to reduce the incidence of this complication.

*Surgical*

Angiography will assist in determining the location, source and the extent of the bleeding site. In addition, it allows assessment for other unruptured lesions. Early definitive surgery to repair ruptured aneurysms reduces short-term complications (i.e. recurrent bleeding and vasospasm) and improves patient outcome.

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### **Annotated Bibliography**

1. **Leicht MJ. Non-traumatic headaches in the emergency department. *Ann Emerg Med* 1980;9:404-409.**
2. **Frishberg BM. The utility of neuroimaging in the evaluation of headache in patients with normal neurologic examinations. *Neurology*, 1994; 44; 1191-1197.**
3. **Mayer PL, Awad IA, Todor R, et.al. Misdiagnosis of symptomatic cerebral aneurysms; prevalence and correlation with outcomes at four institutions. *Stroke* 1996; 27; 1558-1563.**

Of 217 patients with SAH, 25 % of SAH were misdiagnosed, most often as viral meningitis. Most misses failed to consider the diagnosis or perform the appropriate diagnostic tests.

4. **Jakobson K, Saveland H, Hillman J, et. al. Warning leak and management outcome in aneurysmal SAH. *J Neurosurg*, 1996; 85; 995-999.**

20 % of 422 patients with SAH had episodes likely to be a sentinel bleed, with episode occurring a mean of 11 days prior to the SAH.

5. **Harling DW, Peatfield RC, Van Hille PT, Abbott RJ. Thunderclap headache: is it migraine? *Cephalgia* 1989; 9:87-90.**
6. **Morgenstern LB, Luna-Gonzales H, Hunter JC, et. al. Worst headache and SAH; Prospective modern CT and CSF analysis. *Ann Emer Med* 1998; 32:297-304.**

This study CT scanned 107 HA patients and performed LPs on 79; concluded that with current technology. CT alone is sufficient to exclude SAH in HA patients.

7. **Sames TA, Storrow AB, Finkelstein JA, Magoon MR. Sensitivity of new generation CT in SAH. *Acad Emerg Med* 1996;3: 16-20.**
8. **Mayberg MR, Batjer HH, Dacey R, et. al. Guidelines for the management of aneurysmal SAH : a statement for healthcare professional from a special writing group of the Stroke Council, AHA. *Stroke* 1994;25:2231-2238.**

9. **Schull MJ. LP first: an alternative model for the investigation of lone acute sudden headache. *Acad Emerg Med* 1999; 6: 131-136.**

This study uses mathematical modeling to evaluate an LP first ( or alone ) approach to SAH diagnosis, when applied at least 12 hours after the bleed. This would result in 9 additional LPs for every 100 patients with sudden onset HA, but 81 fewer CT scans, with 100% pick up rate for SAH. . If no signs of increased ICP are present, the risk of an LP in these circumstances is probably minimal.

10. **Vermeulen M. SAH; diagnosis and treatment. *J Neurol* 1996;243:496-501.**

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### **Case Outcome**

On presentation to the ED, the patient was unresponsive, BP= , 120/70, HR= 110, agonal respirations. Rectal temperature was 99.0.

There were no signs of external trauma, PERRLA, no rash, MAE with painful stimuli. No spontaneous movement.

Because of the paramedics' suspicions and the patient's possible response to naloxone, it was initially assumed that the patient's unresponsiveness was due to an overdose or a complication of an illicit drug. She underwent rapid sequence induction and was intubated. Gastric lavage was performed. She received more naloxone but no response was observed.

The family was questioned with the help of a Spanish medical interpreter and it was discovered that the patient had headaches off and on all week, but the headache on the day of presentation was the worst. She had no fevers, trauma, or any other symptoms. Once it was established that this patient had a history of headaches, and woke today with the worst ever headache, the diagnosis of Subarachnoid Hemorrhage ( SAH) was entertained.

An emergency non-contrast CT scan was performed and showed a significant amount of SAH surrounding the brain stem, and surrounding the suprasellar cistern surrounding the circle of Willis. Blood was also seen in the ventricles, the intrahemispheric fissure, the Sylvian fissure, and ventricular enlargement (early acute hydrocephalus). Given the location of the blood on CT the suspicion was a left posterior communicating artery aneurysm rupture. An angiogram confirmed the location and found a second 2 cm aneurysm at the bifurcation of the left carotid terminus.

The patient went directly to the OR after the CT scan and had the ruptured aneurysm clipped. At day two post op, she was withdrawing only to pain. No improvement occurred after another 6 days and she was ventilator dependent. At this time, the family decided to withdraw support and she died shortly afterward.

### *Lessons Learned*

The first presentation of HA in this patient may have represented a sentinel bleed. The language barrier, the young age of the patient, the mild symptomatology and the positive response to pain medication may have confused the picture, or lulled the clinician into complacency. It also was assumed that the neck stiffness which worsened in the ED was a dystonic reaction to the medication given to her. It may also have been meningeal irritation.

The second event was minimized by the paramedics again because of the language barrier. In addition, the history of previous calls to the same home for drug related problems, and the patient's possible response to naloxone may have biased the paramedics

in their impression of the case. These assumptions were initially transferred to the ED. Unless the appropriate history had been obtained through the use of interpreters, the final diagnosis may have been delayed.