
A Case of Headache

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Critical Questions in the ED Management of HA

- What is first line therapy for the treatment of HA?
- Does a response to headache pain therapy predict the underlying etiology of the HA?
- Which patients with an acute headache require neuroimaging in the ED?
- What are the indications for a lumbar puncture in the patient with an acute headache?

ED VISIT

CC: "I have a severe migraine"

HPI: 32 year old female complained of a sudden, acute onset vertex headache radiating into her neck for 3 hours associated with nausea and lightheadedness. Similar headache 5 days prior that resolved with naprosyn.

ED VISIT

- Past history of migraines with aura: scintillating lights followed by nausea and right temporal throbbing headache
- Present headache was different in intensity, onset, and location

ED VISIT #1

PMH: Migranes Q-month
MEDS: Naprosyn PRN; BCP
LNMP: 7 Days prior
SH: No Tob / ETOH / drugs
FH: Mother - Migraines

ED VISIT

Appearance: 32 year old female, alert, cooperative but appeared uncomfortable, holding the top of her head
VSS: 118/76, 72, 16, 98.6
Head: Atraumatic
Neck: Nontender, supple
Heart: Regular, no murmurs, no clicks
Lungs: Clear
Abdomen: Soft, nontender

ED VISIT

MS: Alert; Oriented X 3
PUPILS: Not documented
CN: "Intact"
GAIT: "Normal"

A diagnosis of migraine was made. Which of the following is your drug of choice in treating acute severe migraine?

- A Opioid (Meperidine or morphine)
- B Nonsteroidal (Ketorolac)
- C Sumatriptan
- D DHE
- E Prochlorperazine

Migraine: Pathophysiology

- Common pathway for headache pain regardless of the underlying etiology
- Headache pain is transmitted via the trigeminal nerve
- Trigeminovascular axon stimulation results in a release of neurogenic peptides stored in the afferent C fibers innervating cephalic blood vessels
- Vasoactive neuropeptides mediate an inflammatory cascade, "neurogenic inflammation"
- Vasodilatation and enhanced permeability of plasma proteins result in a perivascular reaction

Migraine: Pathophysiology

- Serotonin receptors modulate neurogenic peptide release and cause vasoconstriction
- Goal of migraine therapy is to abort the neurogenic peptide release
- 5-HT_{1c} receptor is most involved in mediating headache
- Drugs working at the 5-HT receptor are the preferred therapy for headache
- Narcotics cause initial pain relief but result in vasodilatation with a high incidence of rebound

Migraine Therapy

- First line agents: Prochlorperazine 5-10 mg IV
 - Metoclopramide
 - Chlorpromazine
- Second line agents: DHE .5-1 mg IM / IV or sumatriptan 6 mg SQ
- Third line agent: Ketorolac
- Fourth line agent: Butorphanol 1 mg intranasally
- Fifth line agent: Opioids

Canadian Headache Society. Guidelines for the diagnosis and management of Migraine in clinical practice. Can Med Assoc J 1997; 156:1273-1287

US Headache Consortium. www.aan.com/public/practice_guidelines

ED VISIT

Diagnosis: Migraine
Treatment: Prochlorperazine
Disposition: Headache resolved

Does response to therapy predict the etiology of an acute severe headache?

- All headache pain is mediated by serotonin receptors
- Case series / case reports (Class III evidence)
- Seymour. Am J Emerg Med 1995. 3 patients treated with ketorolac or prochlorperazine with resolution of headache / Discharged / All with catastrophic outcomes
- Gross. Headache 1995. 3 cases of meningitis with resolution of pain with DHE and metoclopramide
- Pain response can not be used as an indicator or the underlying etiology of an acute headache.

Should this patient have received a head CT?

- Yes
- No

Should this patient have received a head CT?

- Infection
- CNS mass lesion
 - Tumor, IIH, Hydrocephalus
- Collagen vascular disease
 - Temporal arteritis, vasculitis
- Ophthalmologic etiologies
 - Glaucoma, optic neuritis
- Metabolic abnormalities
- Toxins
- Pregnancy related
 - Eclampsia, dural sinus thrombosis
- CNS vascular event
 - Subdural, epidural, SAH
- Primary headache disorder

Which patients with acute headache require neuroimaging in the ED?

- Neuroimaging is obtained to assess for treatable lesions: SAH, CVT, tumors, hydrocephalus
 - (Less tangible: Patient reassurance)
 - (Less tangible: Doctor reassurance)
- Abnormal neuro exam increases the likelihood of a positive CT 3 times (95% CI 2.3-4)
- Normal neuro exam is not predictive
- Location, vomiting, headache waking patient up, worsening with valsalva are not predictive

Which patients with acute headache require neuroimaging in the ED?

- Severe sudden onset headache:
 - Lledo Headache 1994, prospective study: 9 of 27 had SAH (only 4 had a positive CT)
 - Mills Ann Emerg Med 1986, prospective study 42 patients: 29% with worst headache had a positive CT
- Headache in the HIV patient:
 - Lipton Headache 1991, prospective 49 patients: 35% had mass lesion
 - Rothman Acad Emerg Med 1999, prospective 110 pts: 24% had a focal lesion

Which patients with acute headache require neuroimaging in the ED?

- Patients presenting with an acute HA and an abnormal neurologic exam should have an emergent head CT
- Patients presenting with a sudden severe HA should have an emergent head CT
- HIV patients with a new type of headache should have an urgent head CT
- Patients over the age of 50 with a new type of headache should have an urgent neuroimaging study

Should this patient have had a head CT?

- History:
 - HA was sudden and severe in onset
 - HA was different from past headaches
- Physical:
 - No neurologic exam documented:
 - In the HA patient, the neuro exam focuses on pupil, fundoscopy, and cranial nerves III, IV, VI

ED Visit #2

- Patient returned 24 hours later with worsening headache
- Positive findings on the physical examination:
 - Papilledema
 - Left 6th cranial nerve palsy on far lateral gaze
- A noncontrast head CT was normal

What are the indications for LP in acute HA?

- Suspected SAH in a patient with a normal head CT
 - CT is 90 – 98% sensitive for acute SAH
 - Sensitivity decreases over time
- Suspected meningitis
 - LP without CT in patients with normal neuro exam including normal mental status and normal fundoscopic exam
- Suspected idiopathic intracranial hypertension
 - Headache with papilledema
 - Normal CT

ED Visit #2

- Lumbar puncture:
 - Opening pressure 280 mm Hg;
 - CSF: No cells, Normal protein and glucose
- Diagnosis of idiopathic intracranial hypertension was made

**Idiopathic Intracerebral Hypertension:
Diagnostc Criteria**

- Symptoms reflect only ICP or papilledema
 - HA (70-98%)
 - Visual symptoms (57-72%)
 - Pulsatile noises /tinnitus (to 60%)
- Signs only of elevated ICP
 - Papilledema (virtually 100%)
 - Blind spot, field defect or 6th palsy

Friedman. Neurology 2002;59:1492-1495
Ball. Lancet Neurology; 5: 433-42

**Idiopathic Intracerebral Hypertension:
Diagnostc Criteria**

- ICP elevated above 20cm H₂O (25cm in obese)
- CSF is normal
- No structural lesion on enhanced CT or MRI
- No other cause ICP
 - Metabolic
 - Toxic
 - Venous obstruction

Friedman. Neurology 2002;59:1492-1495
Ball. Lancet Neurology; 5: 433-42

**Idiopathic Intracerebral Hypertension:
Epidemiology**

- .03- 2 cases per 100,000
- Most common 20-40 y.o.
- 4 -15 to 1 female to male
- 20 cases per 100,000 in obese women of childbearing age.

**Idiopathic Intracerebral Hypertension:
Etiology**

- Reduced absorption of CSF ?
- Increased CSF production?
- Increased cerebral venous pressure?
- Increased brain water content?

**Idiopathic Intracranial Hypertension:
Clinical Findings**

- | | |
|----------------------------------|------|
| • Papilledema | 100% |
| • Headache | 94% |
| • Visual disturbance | 80% |
| • Transient visual obscuration | 68% |
| • VI CN palsy (False localizing) | 38% |
| • Decreased visual acuity | 30% |
| • Pulsatile noises | 30% |
| • Blindness | 10% |

Giuseffi. Neurology 1991; 41:239-244

**Idiopathic Intracranial Hypertension:
Treatment**

- Weight loss
- Serial lumbar punctures
- Acetazolamide, 1-4 gms / day
- Corticosteroids, 40-60 mg / day
- Surgery
 - Optic nerve sheath decompression
 - Lumboperitoneal shunt
 - Bariatric

Radhakrishnan. Mayo Clin Pro 1994; 69:169-180

**Idiopathic Intracerebral Hypertension:
Treatment**

- 51 studies identified concerning IHH
- 7 concerned treatment
- 2 retrospective, none with control groups
- No Studies met inclusion criteria

- “ There is insufficient evidence to recommend or reject any of the treatments currently available for IHH”

Cochrane Database of Systematic Reviews 2005

CONCLUSIONS

- Errors in management
 - No fundoscopic exam: Ophthalmoscope was not working
 - No CT: symptoms resolved and CT backed-up

CONCLUSIONS

- **Lessons learned**
 - **Patients with headache require a comprehensive neurologic exam**
 - **First line therapy for headache are drugs that work at serotonin receptors**
 - **Response to therapy does not predict etiology**
 - **Patients with sudden severe headache require a CT; if negative followed by an lumbar puncture**

Thank you.

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