EM:RAP C3 PROJECT
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THE C3 APPROACH TO HEADACHE

- 20 million visits to the ER every year in the United States and about 3 million have a chief complaint of headache.
- The approach to headache is simple; you only have to remember two things.

1)Treat the pain i.e. get rid of it.
2)Make sure it is nothing serious.

- About 98% of headaches in the Emergency Department are **primary headaches**. Benign, rarely evolve into something that can kill you although they can be debilitating. About 2% are **secondary headaches** caused by conditions such as subarachnoid hemorrhage, meningitis etc.
- The response to treatment is NOT related to the diagnosis. Just because the patient feels better after your treatment does not mean they don't have something serious.
- Most of the studies on pain relief in headaches include mostly migraine patients due to their prevalence. The most effective treatments for migraine are the dopamine antagonists; ex. droperidol, prochlorperazine, metoclopramide. Adverse effects? QT prolongation. Also can use DHE (dihydroergotamine), the triptans, opioids, high doses of effervescent aspirin/NSAIDs.
- In real life, knowing the details of the different types of primary headaches is less important than distinguishing between primary and secondary headaches. For the exam, it may be useful to know a few facts about primary headaches.

Primary Headaches

There are three main types. In real life, there is a great degree of overlap and definitions are artificial and constructed mainly for research purposes. There is no standard pathology for the three types of headache.

1) Migraine

- The most common. Closely linked to menstruation in females.
- Although the pathophysiology was initially thought related to vascular events, it is now thought to be related to a serotonergic brainstem abnormality that then results in the vasospasm and vasodilation.
- 80% of migraines are common i.e. without aura. 20% are classic with aura. What is an aura? Aura is a specific reversible neurologic symptom prior to the headache. The majority are visual scintillations or scotomata characterized by colorful flashing lights or dark spots, transient. Be careful with presentations that have other neurologic deficits, these may not be migraine.
- Commonly unilateral. Usually don't last longer than 72 hours unless status migrainosus. Recurrent with a gradual onset.
- Triggers: menstrual cycle, chocolate, wine, alcohol.
- **Treatment?**
  - Older therapies included **DHE** (dihydroergotamine). Contraindications? Pregnancy, breastfeeding, hypertension, heart disease, peripheral vascular disease due to the concern of vasospasm triggering stroke/ coronary event.
  - The **triptans** (sumatriptan and its analogs) have mostly replaced the use of DHE. Probably very safe but still some concern about using with coronary artery disease as they can cause chest pain. Triptans are an abortive agent to prevent headache after the onset of aura. Start as early as possible.
  - Other medications used as migraine prophylaxis include beta-blockers, calcium-channel blockers, tricyclic antidepressants, anticonvulsants such as divalproex sodium and sodium valproate.

2) Cluster headaches

- More common in men. Rare. Associated with the young, smoking and drinking.
- Occur over a short period of time (1-2 months) with many episodes of sharp, lancinating headaches centered behind one eye. May be associated with tearing and a Horner's syndrome in 30%.
- Treatment? Similar to migraine. Can use sumatriptan early on. Some classic treatments that frequently show up on exams include high-flow oxygen and intranasal lidocaine or Benadryl on the affected side. May use steroids for severe cases of both cluster and migraine headaches. There is evidence that steroids reduce recurrence.

3) Tension headaches

- Very common. More common in women, middle-aged. Part of a continuum with migraine with a great deal of overlap.
- May present similar to migraine but without aura. Bilateral. Less association with triggers. Also tend to last longer than migraines.
- Think about depression and medication associated headaches. Any patient taking frequent medications for headache may have medication overuse headaches. This is not just true for narcotics but also over-the-counter meds such as NSAIDS.

Secondary Headaches

1) Subarachnoid headaches

- Don't forget that these are considered strokes. 10% of all strokes are said to be subarachnoid hemorrhage.
- Causes include berry aneurysms. Likelihood of developing berry aneurysms increases with age especially with chronic hypertension. Often in patients in their 40-60s. Other associations with berry aneurysms include autosomal dominant polycystic kidney disease, coarctation of the aorta, Marfan's syndrome, Ehlers-Danlos syndrome.
- Subarachnoid hemorrhage is very uncommon in children and is more likely due to congenital AVM than aneurysm.
- Presentations often associated with exertion although may also occur at rest. Fundoscopic examination may show a subhyaloid hemorrhage (a layering of dark red blood behind the eye). May have sentinel headache (about 30-40%); sudden onset, maximal at onset, unlike prior headaches.
- Hunt and Hess classification of subarachnoid headaches. Not important to know the details except for a Grade I is essentially a sentinel leak and a Grade V is the comatose patient with dilated pupils and has a very poor prognosis.

Grade	Signs and Symptoms
I	Asymptomatic or minimal headache and slight neck stiffness
II	Moderate to severe headache; neck stiffness; no neurologic deficit except cranial nerve palsy
III	Drowsy; minimal neurologic deficit
IV	Stuporous; moderate to severe hemiparesis; possible early decerebrate rigidity and vegetative disturbances
V	Deep coma; decerebrate rigidity; moribund

Hunt, Hess **Surgical risk as related to time of intervention in the repair of intracranial aneurysms.** J neurosurg. 1968 Jan; 28(1):14-20

- **A patient presents with a headache and a dilated pupil on one side.** If this is chronic, they may have an aneurysm of their posterior communicating artery with pressure on the third cranial nerve. This is not necessarily ruptured and a lumbar puncture may be negative. These patients still require emergent referral to a neurosurgeon. These aneurysms tend to be larger and at increased risk of rupture.
- **How do you interpret the lumbar puncture?** In real life, this is controversial. For the exam, keep it simple: a positive LP is one with red blood cells that do not clear. 10,000 RBC in tube #1 and 4,000 RBC in tube #4 will be considered a subarachnoid hemorrhage. Even in the setting of a traumatic tap, if the cells do not clear to 0 you can't rule out a subarachnoid. You must repeat at a different interspace or obtain CSF in another manner. You can also make the diagnosis with **xanthochromia**; the supernatant of a spun sample will appear colored or xanthochromatic due to the byproducts of red cell breakdown. Subarachnoid hemorrhage is ruled out by lumbar puncture when there are no red blood cells (or close to no red blood cells i.e. <100 in the last tube) and no xanthochromia.
- The CT alone can't rule out subarachnoid hemorrhage.

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Critical Headache Diagnoses for the Emergency Physician

Diagnosis	Critical Clinical Features	Critical Diagnostic Tests	Critical Interventions	Comments
Subarachnoid Hemorrhage	Sudden onset Maximal at onset Different than previous headaches	CT head LP	Neurosurgery consultation Blood pressure control (nimodipine) Ventriculostomy	CT head and other imaging are insufficient to rule out SAH
Occult Trauma	Signs of abuse/neglect Anticoagulation/coagulopathy	CT head	Neurosurgery consultation Admission	At-risk patients may not volunteer a history of trauma
Bacterial Meningitis	Fever Meningeal irritation Immune compromise Head/neck infection/ instrumentation	CT head LP	Antibiotics Corticosteroids Isolation	With high suspicion, initiate treatment prior to confirmation of diagnosis. Give steroids before/with the first dose of antibiotics
Temporal Arteritis	Jaw claudication Superficial temporal artery tenderness/nodularity Visual symptoms Associated with polymyalgia rheumatica (proximal weakness)	Temporal artery biopsy	Systemic corticosteroids	ESR is an adequate screening test without high risk feature. Empiric treatment with high risk features/ findings, increased ESR
CO Toxicity	Symptomatic cohabitants Flu-like and worse each morning Potentially toxic environment (ex. home furnace in winter)	Arterial co-oximetry	Hyperbaric oxygen therapy (HBOT)	HBOT indicated for neuro/cardiovascular signs, pregnant and beyond certain arbitrary cutoffs
Acute Glaucoma	Red eye Midrange fixed pupil Cloudy cornea	Intraocular pressure	Topical ocular therapy Systemic osmotic agents (Start with timolol/ diamox then pilocarpine) Ophtho consultation	Should identify with cursory exam prior to neuroimaging to prevent delay in consult/treatment
Cervical Artery Dissection	SAH like onset Facial (carotid), neck (vertebral) pain Cranial nerve abnormalities	Angiography	Neurology/ neurosurgery consultation Anticoagulation	In the absence of hemorrhage, start anticoagulation to reduce risk of thrombus/ embolization (otherwise may have delayed presentation of stroke symptoms)

Critical Headache Diagnoses for the Emergency Physician (Con't)

Diagnosis	Critical Clinical Features	Critical Diagnostic Tests	Critical Interventions	Comments
Cerebral/Dural Venous Sinus Thrombosis	Hypercoagulable state (pregnancy/ puerperum, OCPs, malignancy) Head/neck infection Proptosis (cavernous sinus thrombosis)	MR head Venography	Neurosurgery consultation Systemic anticoagulation	May have falsely negative D-dimer CT can show empty delta sign (see a triangular filling defect at confluence of sinuses)
Space Occupying Lesion	Progressively worse with time New onset in patient >50y History of malignancy Worse in morning/ head down position	CT head	Neurosurgery consultation ICP lowering therapy Lesion specific therapies.	ICP lowering therapy: elevate head of bed, restrict IV fluids, mannitol, hyperventilation Lesion specific therapy: steroids, surgery, neuroradiology procedure, antibiotics
Cerebellar Infarction	Headache with dizziness Cerebellar signs Cranial nerve abnormalities	CT head	Neurology/ neurosurgery consultation	CT head is insensitive for infarct but helpful to rule out hemorrhage, edema, mass effect
Idiopathic Intracranial Hypertension	Obese, young female patient Cranial nerve 6 palsy (false localizing sign due to globally increased ICP) Papilledema	LP	CSF drainage Neurology referral	After negative imaging, LP will have markedly elevated opening pressure and provide temporary relief
Pituitary Apoplexy	Thunderclap headache Vomiting Visual acuity, field deficits Ocular palsies	CT head MR head	Neurosurgery consultation	Many are not visible on CT, MR is the imaging of choice
Pre-eclampsia	Post-partum (up to four weeks)	CBC Chem panel with LFTs Coagulation studies	IV magnesium OB consultation	Up to half of all patients present in the post-partum period, the majority with headache

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Cases

1) 56 year old man with a headache that started during sexual intercourse. Different from prior headaches. Vital signs normal except for slightly hypertensive. No findings on exam. Negative CT. -> **Subarachnoid hemorrhage.**

2) 16 year old brought in by deputies with headache. He has just been arrested and says he is fine. Asking for aspirin. On exam, signs of fighting (scratches, bruises) but normal neuro exam. -> **Occult trauma.**

3) 35 year old man with chronic sinusitis is two weeks post-op from a procedure and complaining of headache for two days and rigors. -> **Bacterial meningitis.** Note: Classic case = college kid in dorm (meningococcus).

4) 80 year old female from Australia with chronic weakness presents with a headache and new visual changes. Tenderness over temporal artery -> **Temporal arteritis.**

5) 15 year old male presents with several days of headache and flu-like symptoms that are worse in the morning. Family members and dog are also ill. -> **CO toxicity.**

6) 50 year old man with sudden onset of headache after leaving a movie theater. He won't let you examine his eye. -> **Acute glaucoma.**

7) 47 year old man doing yoga presents with sudden onset of headache, the worst of his life. Normal neuro exam. Negative CT/LP. -> **Cervical artery dissection.**

8) 42 year old pregnant female who is about ready to pop presents with a new onset headache that is gradually worsening and not better with pain medications. Urinalysis has no proteinuria and her blood pressure is low. -> **Cerebral/dural venous sinus thrombosis.**

9) 28 year old African American female who is obese with a history of hypertension and polycystic ovarian disease presents with daily headaches that are worse every morning. Exam shows left sixth cranial nerve palsy. -> **Idiopathic intracranial hypertension.**

10) 55 year old female with a history of breast cancer presents with headaches that are daily, progressive and worse in the mornings. -> **Space occupying lesion.**

11) 78 year old woman with diabetes, hypertension presents with acute new headache, dizziness and vomiting. No specific findings on neuro exam. -> **Cerebellar infarction.**

12) 55 year old with a history of an endocrine tumor presents with a thunderclap headache. -> **Pituitary apoplexy.**

13) 32 year old post-partum female, headache on day 3, gradual onset, intense, not sure if different than previous headache. -> **Pre-eclampsia.**

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RHABDOMYOLOSIS

- **Pathophysiology?** Occurs with injury/necrosis to the muscle fibers. Contents of muscle fibers (myoglobin, CK, aldolase etc) leak into circulation and result in the complications of rhabdomyolysis. Consider this an end-stage of badness similar to ATN, DIC, ARDS, etc.
- Rare in children.
- **A wide variety of causes in adults.** Alcohol, caffeine, illicit drugs (especially cocaine which causes vasoconstriction leading to ischemia of the muscle beds, opiates may result in coma/found down). Medications such as statins. Note: Patients taking statins may present with myalgias but normal CK/enzymes. It is possible to have a statin-induced myalgia that is considered by some to be equivalent to rhabdomyolysis. Electrolyte abnormalities such as hypokalemia (Why? Potassium is a vasodilator in the muscle bed, falling levels will result in vasoconstriction much like with cocaine), hypophosphatemia. Traumatic injuries. Crush injuries. Compartment syndrome from running. Viral infections; classically influenza A and B.
- **Note:** Don't confuse the electrolyte abnormalities that cause rhabdomyolysis (hypokalemia, hypophosphatemia) with the electrolyte abnormalities that result from rhabdomyolysis (hyperkalemia).
- **Presentation?** Found down/coma. Muscle pain/weakness, malaise. On medications known to cause rhabdomyolysis. Low grade fevers. Urine looks dark/brown.
- **How do you make the diagnosis?** Textbooks say at least a fivefold increase above the upper limit of normal in CK level. In the US, this is approximately >1000 U/L of CK. Be careful; a CK level of 600 U/L may not technically meet the definition of rhabdomyolysis but could be rising. Why do we follow CK rather than myoglobin? Myoglobin is probably more responsible for the renal failure of rhabdomyolysis but it has a short half-life and unreliable presence in the urine. CK is more reliable. **Classic findings on the urinalysis?** Dipstick positive for blood (because it does not differentiate between myoglobin, hemoglobin, etc) but no red blood cells on micro. On exams, this urine finding is more commonly due to rhabdomyolysis but remember that intravascular hemolysis (ex. blood transfusion reaction, DIC, HUS, TTP) may have similar findings due to circulating free hemoglobin.
- **Complications?** Acute renal failure. Compartment syndrome secondary to exercise, trauma, pressure/crush injuries may need surgical intervention (ex. patient found down developed rhabdomyolysis and renal failure as a result of gluteal compartment syndrome). Electrolyte abnormalities including hyperkalemia, hypercalcemia (late finding)/hypocalcemia, hyperphosphatemia/hypophosphatemia (late finding), hyperuricemia. Remember that when phosphate increases, calcium decreases (to prevent precipitation of crystals in the body). Also DIC.
- **Note:** If you have a patient with rhabdomyolysis due to an unknown etiology, make sure you do a very careful physical exam to rule out any muscle injury that may have triggered it.
- **Treatment?** Early and aggressive IV fluids. Force these patients to produce a lot of urine (at least several cc/kg/h). What about bicarbonate? Controversial. Theory that myoglobin is more nephrotoxic in an acidic environment so bicarbonate given to alkalinize the urine. No studies have shown benefit to this but may want to consider in patient in rhabdomyolysis with a low urinary pH.

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