

## **Episode 14 - Headache Pearls and Pitfalls**

Prepared by Dr. Lucas Chartier

#### **Migraine**

**POUND mnemonic for diagnosis of migraine**: *P*ulsatile quality, 4-72 h*O*urs, *U*nilateral pain, *N*ausea, and *D*isabling intensity – 4 out of 5 features present gives a positive likelihood ratio of 24 for this headache to be a migraine (in a study based out of GP clinics); photophobia and phonophobia are also often present

Retinal and vitreous detachment produce flashes or floaters that are unilateral, white in color and produce a "curtain descending on the vision" phenomenon, as opposed to the migraine-associated bilateral, coloured and tunnel-vision symptoms

**SSNOOP mnemonic for red flags**: *S*ystemic signs (fever, weight loss), *S*econdary risk factors (immunocompromised status, HIV), *N*eurological signs (speech deficit, cranial nerve abnormalities), *O*nset – abrupt, *O*lder age (>40yo), *P*rogression of symptoms

To make the diagnosis of migraine, the patient really should have had prior repeated and similar symptoms that have been diagnosed as a migraine by a physician, not simply self-diagnosis

Evidence-based treatment in the ED:

Dopamine antagonist such as metoclopraminde (Maxeran©) or prochlorperazine (Stemetil©) in a mini-bag infusion over 15min (not as an injection), alongside an anticholinergic such as benzatropine (Benztropine©) or diphenhydramine (Benadryl©) to decrease the extra-pyramidal symptom of akathisia (i.e. restlessness) – NNT of 5 for these two adjuncts

Remember that the mere improvement of the headache with therapy does NOT exclude serious pathology

Steroids (eg, dexamethasone 10-15mg IV or PO) at discharge may be useful to prevent rebound headache within 72hrs by decreasing the inflammation of the blood vessels in the brain

At discharge, naproxen 500mg PO has been shown to be as useful as the 'triptan' class of drugs, which should only be prescribed in people who have had response to them in the past and who do not have hypertension or cardiovascular disease

## Subarachnoid hemorrhage (SAH)

**SUM mnemonic for diagnosis of SAH**: Sudden onset, Unlike previous headaches, Maximal at onset

Also consider risk factors of family history of cerebral aneurysm, SAH or polycystic kidney disease, or collagen vascular diseases, hypertension, and binge drinking, smoking or use of cocaine, as well as an elicited history of a recent similar headache (i.e. sentinel bleed), onset during exertion or presyncope or syncope associated with this headache

Migraine itself is not a risk factor for SAH, but remember that migraine-sufferers may have SAH as well!

ECG changes in 50-100% of patients due to neurogenic myocardial stunning and coronary vasospasm: deep, wide precordial T-wave inversion, bradycardia, and prolonged QT – beware of anticoagulating these patients on the assumption of acute coronary syndrome

Study by Perry et al. of signs of SAH:

The following are strongly and reliably associated with SAH: age >40, neck stiffness or pain, onset of headache on exertion, vomiting, witnessed loss of consciousness, and elevated BP >160/100

Also consider the following signs: stroke-like symptoms, seizure or 3<sup>rd</sup> cranial nerve palsy from mass effect, 6<sup>th</sup> cranial nerve palsy with diplopia, or subhyloid hemorrhage (i.e. dense red on fundoscopy, also called Terson syndrome – patient will eventually need referral to ophthalmology), and even meningismus

## Work-up of SAH:

CT scan of the head – sensitivity of at least 95% in first 12hrs after onset, but decreases to 85% the next day and 50% after one week

Lumbar puncture (LP) is therefore still standard of care, despite 25% risks of post-LP headache, and the small risks of neurological damage and infection

Do NOT wait to 12hrs after onset (when xantochromia becomes reliably present) to perform LP as patients with the disease would therefore be put at risk of a subsequent fatal bleed

A true SAH-positive tap may hide in a 'traumatic tap', so call a tap a 'negative tap' if and only <5 RBCs in tube #4 – a decrease in at least 25% in the number of RBCs between tubes 1 and 4 should NOT be used at all

Opening pressures should ALWAYS be done and documented, because it might be elevated in SAH (will never be elevated in traumatic tap), and may help diagnose alternate conditions, such as idiopathic intracranial hypertension or cerebral venous thrombosis

Post-LP headaches classically occur 3 days later, are worse when not supine, and are a result of CSF leak from the dura – they are minimized by using smaller (i.e. 25G – tip: use a 16G needle as a trocar to penetrate the soft tissues, then insert the 25G needle inside this), atraumatic (non-cutting) blunt tip needles; bedrest, caffeine and hydration have all been shown to NOT be effective at reducing post-LP headaches, and the definitive treatment involves an autologous blood patch inserted by an anesthetist

If a patient refuses an LP or the physician fails to obtain CSF fluid, consider doing a CT-angiogram – this will exclude aneurysms that could lead to bad outcomes in the short term, but may also lead to false positive: 2-6% of the population has cerebral aneurysms, but CT-A cannot identify whether this particular aneurysm is the culprit for the headache, or even if it has a high likelihood of rupturing in the future

### ED treatment of SAH:

To prevent re-bleeding, treat hypertension <u>only if</u> the mean arterial pressure is persistently over 100-110 for a few hours, and consider involving your consultant neurosurgeon on the target and method to do this – labetalol 20mg IV bolus followed by an infusion may be appropriate

To prevent vasospasm and resultant cerebral infarct, nimodipine (calcium-channel blocker) 60mg PO/NG q4-6hrs needs to be started within 24hrs of presentation

To prevent seizures, which will occur in 5-20% of patients with SAH, consider starting anti-epileptics

#### Spontaneous cervical artery dissection

Often present after a trivial trauma such as hyperextension of the neck as a result of shaving, checking one's blind spot while driving, chiropractic manipulation, roller coaster ride, boxing, or even coughing or vomiting, especially in the setting of connective tissue disease

Carotid artery dissection presents with unilateral facial, neck or head pain with a partial Horner's syndrome (myosis and ptosis, but not anhydrosis), and  $1/3^{rd}$  of patients will have retinal or cerebral TIA within one week (neurological symptoms lag behind because it takes time to have a thrombus formed and thrown from the site of dissection)

Vertebral artery dissection presents with posterior neck or occiput pain and posterior circulation symptoms – ataxia, vertigo, dysarthria, diplopia and dysphagia

Diagnosis is made by CT-A of the head and neck (carotid Doppler may be used if CT-A not available, but it is not as good and therefore a CT-A is still eventually necessary)

Treatment includes antiplatelet or anticoagulation therapy, except in the presence of large infarct with mass effect, hemorrhagic transformation of an infarct, or intracranial extension of the dissection, but consultants should weigh in before treatment is initiated

# Cerebral venous thrombosis (CVT)ii

May present as three different entities: headache (from thunderclap to subacute), stroke-like symptoms, or seizures

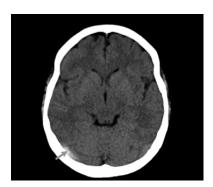
Clinical features associated with CVT are risk factors associated with thromboembolic disease, as well as papilledema, younger patients (<40yo), orbital chemosis and proptosis in cavernous CVT, dilated scalp veins and scalp edema in sagital CVT, and CNS or ENT infections such as sinusitis

D-dimers are not reliable in the diagnosis of CVT, even in low-risk patients (if you are considering the diagnosis, you need to fully investigate it with neuro-imaging)

Given that the optimal test MR-V is not readily available, diagnosis can be made with plain CT head in only 30% of cases (delta sign, hemorrhagic infarct at gray-white junction, or hyperdense cortical vein or dural sinus – image on the left) so CT-venogram must be done as well if plain CT is negative (CT-V signs: empty delta sign – image on the right)

If LP is performed, opening pressure may be elevated; treatment includes unfractionated heparin or LMWH (despite the risk of hemorrhagic transformation, anticoagulation has been shown to reduce death and dependency)

CVT is on the same spectrum as idiopathic intracranial hypertension (IIH), which presents as refractory headache with blurry vision and visual field defects in young, obese women on oral contraceptive pills; signs include papilledema and VERY high opening LP pressure in the face of normal CT scan; it is treated with diuretics, not anticoagulation



## Extracranial causes of headache

CO poisoning (think in the setting of multiple patients or wood-bruning stove), acute glaucoma (photophobia – do an eye exam!), temporal arteritis (systemic signs, and associated with polymyalgia rheumatica, jaw claudication blurry vision or retinal ischemia – check the ESR!), and hypertensive encephalopathy (altered mental status with papilledema and end-organ damage in the setting of severe hypertension)

#### 10 serious causes of headache

- Lesion on CT scan (blood, pus or tumor):
  - Blood Subarachnoid hemorrhage, subdural hemorrhage, or stroke hemorrhagic or not
  - Pus meningitis or encephalitis
  - Tumor tumor 1<sup>ry</sup> or 2<sup>ry</sup>, benign or malignant
- Other diagnoses in the head:
  - o Cervical artery dissection carotid or vertebral
  - Hypertensive encephalopathy
  - Pre-eclampsia (or eclampsia)
  - o Cerebral venous thrombosis or idiopathic intracranial hypertension
  - o Glaucoma
  - Temporal arteritis
- Thinking 'outside the box':
  - o Carbon monoxide (CO) poisoning

<sup>&</sup>lt;sup>1</sup> Perry JJ, Stiell IG, Sivilotti MLA, et al. High risk clinical characteristics for subarachnoid haemorrhage in patients with acute headache: prospective cohort study. *BMJ* 2010;341:c5204

<sup>&</sup>lt;sup>ii</sup> Saposnik G, Barinagarrementeria F, Brown RD, et al. Diagnosis and Management of Cerebral Venous Thrombosis: A Statement for Healthcare Professionals From the American Heart Association/American Stroke Association. *Stroke* 2011;42;1158-1192