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Friday, June 10, 2016

14:00 - 14:55 WS #43: Case Studies on Headache
15:05 - 16:00 WS #55: Case Studies on Headache (Repeated)
Case Studies in Headache
A Tool Box of Tips to Aid GP Survival

Dr Paul Timmings
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Headache

• Commonest acute neurological problem
  • 50% of people have had or get headaches
• Not usually an accepted reason for hospital clinic referral
• Yet
  • Some headaches are very serious
  • Others are very bothersome
  • Others cause a lot of trouble
    • To the patient
    • To the Dr

• We all have anxiety about headache diagnoses
Case 1:

• Woman age 41. Onset of migraine age 14
• Some with aura, some without. Some severe, some mild
• Triggers (self identified): menses, mid cycle, fasting, sleep deprivation
• Severe episodes start with shimmering central scotoma which evolves over 20-40 min then pulsatile H/A L>R. +/- N&V, prefers dark room.
• Early Rx helps. If Rx delayed or unsuccessful, attack will last 2-4 days
• Increased frequency & severity on COCP

• On direct enquiry:
  • Severe attacks: every 6 months.
  • Mild episodes (no aura, no vomiting, & can “carry on”): 2-3 x/month
• Other questions you might have .....
Question

Diagnosis?
• A classic migraine
• B Common migraine
• C Both
• D Tension headache
• E All of the above?
What is Migraine?

- An inherited tendency to headaches with sensory disturbance.
- An instability in the way the brain deals with incoming sensory information.
- That instability can be up or down regulated by physiological change such as sleep, exercise, hunger, hormones and emotion.

Adapted from: Prof Peter Goadsby. King’s College, London
Migraine: A definition

• Migraine with aura

At least 2 attacks fulfilling criteria B and C

B. ≥1 of the following fully reversible aura symptoms: visual; sensory; speech and/or language; motor; brainstem; retinal

C. ≥2 of the following 4 characteristics:

≥1 aura symptom spreads gradually over ≥5 min, and/or ≥2 symptoms occur in succession

Each individual aura symptom lasts 5-60 min ≥1 aura symptom is unilateral
Aura accompanied or followed in <60 min by migraine headache

TIA excluded

• Migraine without aura

At least 5 attacks fulfilling criteria A-C

A: Headache attacks lasting 4-72 h (untreated or unsuccessfullly treated)

B: Headache has ≥2 of the following:

1. unilateral location
2. pulsating quality
3. moderate or severe pain intensity
4. aggravation by, or causing avoidance of routine physical activity

C: During headache ≥1 of the following:

1. nausea and/or vomiting
2. photophobia and phonophobia

Cephalalgia. 2013:629-808
Acute Treatment: Main problem is dosing too low and too late.

<table>
<thead>
<tr>
<th>Analgesics</th>
<th>Antiemetics</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Paracetamol 1000mg</td>
<td>• Prochlorperazine 5-10mg po 2-4mg buccal, 25mg PR</td>
</tr>
<tr>
<td>• Aspirin 600-1000mg</td>
<td>• Metoclopramide 10-20mg po</td>
</tr>
<tr>
<td>• Ibuprofen 400-800mg</td>
<td>• Promethazine 25-50mg po</td>
</tr>
<tr>
<td>• Naproxen 250-750mg</td>
<td>• Ondansetron 4-8mg tabs or wafers</td>
</tr>
<tr>
<td>• Diclofenac 50mg (not CR)</td>
<td>• Beware: Triptan overuse and rebound headache</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Triptans</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>• Sumatriptan 50-200mg po. 6-12mg sub cut</td>
<td></td>
</tr>
<tr>
<td>• Rizatriptan 10-30mg (halve dose if on propranolol)</td>
<td></td>
</tr>
</tbody>
</table>
Rescue Treatments to consider in Acute Care

- Sumatriptan sub cut
- Neuroleptics
  - Chlorpromazine (0.1-1mg/kg) 12.5mg - 37.5mg iv/im with saline load
  - Prochlorperazine 5-10mg im
  - Haloperidol 5mg iv in 500ml saline
- Valproate 300-500mg iv
- Steroids. eg dexamethasone 10-24mg iv
- Metoclopramide
- Ondansetron
When to Consider Migraine Prophylaxis

• Three or more migraine headaches per month, for >3 months
• Headaches interfere with daily activities
• Acute treatments not working or overused
• Adverse effects of acute treatments, e.g., gastritis
• Special situations:
  • Pregnant
  • Elderly
  • Paediatric
Prophylactics to Consider

• Beta blockers
  • Propranolol, Metoprolol, Nadolol

• Calcium channel blockers
  • Verapamil, Diltiazem

• Anti-Epileptic drugs
  • Sodium Valproate, Gabapentin, Topiramate

• Antidepressants
  • Amitriptyline, Nortriptyline, Doxepin,
    Fluoxetine, Venlafaxine

• Others
  • Clonidine
  • Lithium
  • Botox
  • Lisinopril
  • Candesartan
  • Trigeminal nerve stimulation
Case 2:

• 28 yr old woman presents with “stress headache”
• Previous Rx’d for anxiety and low mood, currently OK without Rx
• H/A’s date from late teens. Worse in past year, coincident with job promotion and relocation
• Description:
  • Bilateral non-pulsatile pain lasting 12-24hrs, 1-2x/wk
  • Usually preceded by tight feeling high in neck
  • Occasionally moderate to severe with some light & noise sensitivity & needing time off work on some occasions.
• Examination: Normal
• GP ordered CTB normal
Question

Likely Diagnosis
A. Migraine
B. Idiopathic Intracranial hypertension
C. Tension type headache (TTH)
D. Sinus headache
E. Hypertensive headache
Case 2: Diagnosis

- No red flags & temporal pattern not of concern
  - Stress trigger and bilateral non-pulsatile pain suggest TTH

- But the fact that the pain is moderate to severe at times and sometimes associated with photo- & phono-phobia excludes TTH.

- Correct diagnosis: **Episodic migraine without aura**

- TTH **never** reaches severe intensity & is **never** linked with photo- or phono-phobia
Tension Headache (TTH)

- 90% of patients consulting with TTH are eventually diagnosed with migraine

- **Tension Headache is defined by what it is not:**
  - Not a localised pain
  - Not throbbing
  - Not aggravated by activity
  - Not severe
  - Not associated with autonomic, neurologic or migraine features
  - No nausea, no vomiting
  - Photo-phobia and phono-phobia cannot both be present
  - Not a secondary headache
# Tension Headache Treatments

<table>
<thead>
<tr>
<th>Acute</th>
<th>Preventative</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Paracetamol</td>
<td>• Amitriptyline</td>
</tr>
<tr>
<td>• Aspirin</td>
<td>• Other TCAs</td>
</tr>
<tr>
<td>• Naproxen</td>
<td>• Venlafaxine</td>
</tr>
<tr>
<td>• Diclofenac</td>
<td>• Biofeedback (EMG)</td>
</tr>
<tr>
<td>• Other NSAIDs</td>
<td>• CBT</td>
</tr>
<tr>
<td>• Caffeine (60-200mg)</td>
<td>• Relaxation training</td>
</tr>
<tr>
<td></td>
<td>• Physio</td>
</tr>
<tr>
<td></td>
<td>• Acupuncture</td>
</tr>
</tbody>
</table>
Red Flags for Possible Secondary Headache

• First or worst ever headache
• Abrupt onset of headache
• Progression or fundamental change in headache pattern
• New headache in <5yrs or >50 yrs old patient
• New headache with cancer, immunosuppression, or pregnancy
• Headache with syncope or seizure
• Headache triggered by exertion, valsalva or sex
• Focal neurological symptoms lasting longer than 1 hour
• Abnormal findings, - on general or neurological examination
Secondary Headache

- Every Dr is concerned not to “miss” a mass lesion
- Tumour:
  - 5-30% have headache,
  - **only 1% have headache as the sole symptom**
- Headaches with tumour or raise ICP can wake from sleep.
  - But this pattern is also **common** with migraine and cluster headache

- Primary headache patterns are usually aggravated by secondary causes
  
  Put the Other Way ........

- Secondary Headache may present as worsening of Primary Headache
Secondary Headaches: Thunderclap
Top 5 causes

- SAH
- RCVS (Reversible Cerebral Vasoconstriction Syndrome)
- Neck artery (vertebral or carotid) dissection
- Venous sinus thrombosis
- Acute low CSF pressure (Intra-cranial hypotension)

Diagnosed by the company they keep:
  History and Neurological findings
Case 3:

- 54 yr old woman presented acutely.
- Sudden onset of severe H/A during orgasmic sex
- Tourist from UK
- PHx migraine

- O/E vague & muddled. Poor vision “blurry”
- Diffuse mild limb weakness.
- Reflexes generally brisk.
Questions

• To hospital or not?
• Y/N
Questions

Red flags:
Age, gender, new H/A, worst ever, tourist.
• Came by helicopter from Whitianga

Diagnosis
A. SAH
B. RCVS
C. Dural sinus thrombosis
D. Coital cephalalgia
E. Carotid dissection
Case 3: Progress

- CTB normal. LP near normal, 10 WBC
- Developed subtotal visual loss
- MRI: patchy signal change in both occipital regions with multifocal intracranial arterial narrowing ?spasm

- More history:
  - Usually takes fluoxetine 40mg/d
  - Having a fantastic holiday, enjoyed local wine and marijuana with partner
RCVS
Reversible Cerebral Vasoconstriction Syndrome

- Increasingly recognised.
- Second commonest cause of thunderclap headache. Can be recurrent
- Commoner in 40-60yr old women
- Risk factors include: (often multiple factors concurrently present)
  - Post partum
  - Binge drinking (alcohol)
  - Marijuana use
  - SSRI use
  - Migraine
(A): SAH vs (B): Trace of cortical blood seen in 1/3 of RCVS Cases
Example of Vasospasm & Subsequent Recovery
Example of Vasospasm & Subsequent Recovery
Secondary Headaches: A Potpourri

• Not all secondary headaches are evil
• Must take history and examine, - including fundi

• Idiopathic Intracranial Hypertension:
  • Ask about wt gain, pulsatile tinnitus, TVO, diplopia. Look for papilledema

• Orthostatic H/A (headache on standing)
  • Intracranial hypotension.
    • After LP. After head or neck trauma, dehydration, multiple other rare causes
    • 15% may be thunderclap at onset
  • POTS:
    • Typically post-pubertal young women, orthostatic & non-orthostatic H/A with co-existent migraine. Usually post infectious. Assoc w fatigue, decr concentration, exercise intolerance, constitutional Syx & pre-syncope. HR increase by 30b/min after 5-30min standing is diagnostic.
    • Respond to: incr exercise, incr salt & water, B blockers, fludrocort, midodrine
Red Flags for Secondary Headache

• First or worst ever headache
• Abrupt onset of headache
• Progression or fundamental change in headache pattern
• New headache in <5yrs or >50 yrs old patient
• New headache with cancer, immunosuppression, or pregnancy
• Headache with syncope or seizure
• Headache triggered by exertion, valsalva or sex
• Focal neurological symptoms lasting longer than 1 hour
• Abnormal neurological examination findings
Case: 4

• 45yr old man presents with right frontal headache
• Daily for past 3 weeks
• Pulsatile and moderate to severe.
• Worse since recent “flu” with nasal congestion
• O/E afebrile and looks well

• Questions:
• Duration
• Radiation ? Over head ? Down face
• Time of day ? Diurnal ? Seasonal
• ESR ??
Trigeminal Autonomic Cephalalgias (TAC’s)

- Episodic moderate to severe unilateral head or face pain, 
  **with** ipsilateral cranial autonomic features such as:
  - lacrimation
  - conjunctival injection
  - rhinorrhoea
  - nasal congestion
  - eyelid oedema and ptosis

- The **striking** feature of a TAC is the **autonomic component** with each attack of pain.

- Syndromes vary in duration and frequency
  - Cluster headache (CH) attacks are longest and least frequent,
  - Short-lasting Unilateral Neuralgiform headache Attacks (SUNA) are the most frequent and shortest attacks
# Trigeminal Autonomic Cephalalgias (TAC’s) Clinical Features:

<table>
<thead>
<tr>
<th>feature</th>
<th>Cluster headache</th>
<th>Paroxysmal hemicrania</th>
<th>SUNCT</th>
<th>Idiopathic Stabbing headache</th>
<th>Trigeminal neuralgia</th>
<th>Hemicrania continua</th>
<th>Hypnic Headache</th>
</tr>
</thead>
<tbody>
<tr>
<td>gender (M:F)</td>
<td>9:1</td>
<td>1:3</td>
<td>8:1</td>
<td>F &gt; M</td>
<td>F &gt; M</td>
<td>1:1.8</td>
<td>5:3</td>
</tr>
<tr>
<td>Pain type</td>
<td>boring</td>
<td>boring</td>
<td>stabbing moderate orbital</td>
<td>stabbing severe orbital</td>
<td>stabbing very V2/V3 &gt; V1</td>
<td>steady moderate unilateral</td>
<td>throbbing moderate generalised</td>
</tr>
<tr>
<td>severity</td>
<td>very orbital</td>
<td>orbital</td>
<td>orbital</td>
<td>any</td>
<td>&lt; 1 s</td>
<td>variable</td>
<td>15-30 mins</td>
</tr>
<tr>
<td>location</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1-3/night</td>
</tr>
<tr>
<td>duration</td>
<td>15-180 m</td>
<td>2-45 m</td>
<td>15-120 s</td>
<td>1/d-30/hr</td>
<td>&lt; 5 s</td>
<td>&lt; 1 s</td>
<td>lithium</td>
</tr>
<tr>
<td>frequency</td>
<td>1-8/d</td>
<td>1-40/d</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>autonomic</td>
<td>+</td>
<td>+</td>
<td>±</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>alcohol</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>indomethacin</td>
<td>- (? )</td>
<td></td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
</tbody>
</table>
Beware TAC Mimics

Posterior fossa or pituitary lesions may mimic TAC’s. SUNCT and SUNA are disproportionally over-represented in headaches due to pituitary micro- or macro-adenomata.

Pathophysiology of TACs

Functional imaging shows activation of the posterior hypothalamus in CH, SUNCT and PH. In migraine activation is in the dorsal pons, locus ceruleus and periaqueductal grey matter.

Hemicrania Continua shares clinical features of migraine and TACs, and shows activation in both hypothalamus and brainstem structures.
## Table 2. Treatment Options for TACs

<table>
<thead>
<tr>
<th></th>
<th>CH</th>
<th>PH</th>
<th>SUNCT/SUNA</th>
<th>HC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abortive therapies</td>
<td>Oxygen</td>
<td>Sumatriptan sc</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sumatriptan in</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Short-term preventive</td>
<td>Prednisolone</td>
<td>—</td>
<td>Intravenous lidocaine</td>
<td>—</td>
</tr>
<tr>
<td>Preventive therapies</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indomethacin</td>
<td>—</td>
<td>+++</td>
<td>—</td>
<td>+++</td>
</tr>
<tr>
<td>Verapamil</td>
<td>+++</td>
<td>+</td>
<td>—</td>
<td>+/-</td>
</tr>
<tr>
<td>Other calcium channel antagonists</td>
<td>—</td>
<td>+</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Topiramate</td>
<td>++</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Lithium</td>
<td>++</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Lamotrigine</td>
<td>—</td>
<td>—</td>
<td>+++</td>
<td>—</td>
</tr>
<tr>
<td>Gabapentin</td>
<td>—</td>
<td>—</td>
<td>++</td>
<td>+</td>
</tr>
<tr>
<td>Amitriptyline</td>
<td>—</td>
<td>—</td>
<td>++</td>
<td>+</td>
</tr>
<tr>
<td>Non-pharmacological</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GON blockade</td>
<td>++</td>
<td>+/-</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>ONS</td>
<td>++</td>
<td>+</td>
<td>++</td>
<td>++</td>
</tr>
</tbody>
</table>
Thank You

Questions and Discussion

Dr Paul Timmings
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Hamilton
# Features of Common Thunderclap Headache Syndromes

<table>
<thead>
<tr>
<th>Cause</th>
<th>Clinical Features</th>
<th>Brain CT</th>
<th>Lumbar Puncture</th>
<th>Angiography</th>
<th>Brain MRI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aneurysmal subarachnoid hemorrhage</td>
<td>Altered consciousness, seizures, meningismus</td>
<td>Subarachnoid blood in basilar cisterns and sylvian fissures</td>
<td>Elevated red blood cells, xanthochromia</td>
<td>Ruptured aneurysm, vasospasm</td>
<td>Subarachnoid blood in basilar cisterns and sylvian fissures</td>
</tr>
<tr>
<td>Reversible cerebral vasoconstriction syndrome</td>
<td>Recurrent thunderclap headaches</td>
<td>Normal, subarachnoid blood along cortical surface/sulci</td>
<td>Normal, mild white blood cell elevation, mild protein elevation</td>
<td>Multifocal multivessel vasoconstriction</td>
<td>Normal, subarachnoid blood along cortical surface/sulci, ischemic stroke, cerebral edema, intracerebral hemorrhage</td>
</tr>
<tr>
<td>Carotid and vertebral artery dissection</td>
<td>Neck pain, symptoms related to cerebral ischemia, Horner syndrome (carotid dissection)</td>
<td>Normal, ischemic stroke</td>
<td>Normal</td>
<td>Dissected artery, multifocal, segmental vasoconstriction if associated with reversible cerebral vasoconstriction syndrome</td>
<td>Normal, ischemic stroke</td>
</tr>
<tr>
<td>Cerebral venous sinus thrombosis</td>
<td>Focal neurologic deficits, altered mental status, visual changes</td>
<td>Dense triangle sign (clot inside the sinus), cord sign (thrombosed cortical or deep vein), venous hemorrhages</td>
<td>Elevated opening pressure, high protein</td>
<td>Venous sinus thrombosis</td>
<td>Normal, venous infarctions with hemorrhage; MRI evidence of intraluminal thrombus on T1, T2, and susceptibility-weighted imaging sequences</td>
</tr>
<tr>
<td>Spontaneous intracranial hypotension</td>
<td>Orthostatic headache, auditory muffling</td>
<td>Normal, subdural collections</td>
<td>Low opening pressure</td>
<td>Normal</td>
<td>Pachymeningeal enhancement, sagging brain, subdural collections</td>
</tr>
</tbody>
</table>

CT = computed tomography; MRI = magnetic resonance imaging.
Causes of Thunderclap Headache

Most Common Causes of Thunderclap Headache
- Reversible cerebral vasoconstriction syndrome
- Subarachnoid hemorrhage

Less Common Causes of Thunderclap Headache
- Cerebral infection
- Cerebral venous sinus thrombosis
- Cervical artery dissection
- Complicated sinusitis
- Hypertensive crisis
- Intracerebral hemorrhage
- Ischemic stroke
- Spontaneous intracranial hypotension
- Subdural hematoma

Uncommon Causes of Thunderclap Headache
- Aqueductal stenosis
- Brain tumor
- Cardiac cephalgia
- Giant cell arteritis
- Pituitary apoplexy
- Pheochromocytoma
- Retroclival hematoma
- Spontaneous spinal epidural hematoma
- Third ventricle colloid cyst

Possible Causes of Thunderclap Headache
- Primary or idiopathic thunderclap headache
- Unruptured intracranial aneurysm

Schwedt T. Continuum. 2015:1058-1071
### Table 1: Differential Diagnoses of the TACs

<table>
<thead>
<tr>
<th>Headache Syndrome</th>
<th>Differential Diagnoses</th>
<th>Distinguishing Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>CH</td>
<td>Migraine with prominent autonomic features</td>
<td>Agitation usually present in CH; also circadian and circannual periodicity</td>
</tr>
<tr>
<td>PH</td>
<td>CH</td>
<td>PH responds absolutely to indomethacin</td>
</tr>
<tr>
<td>SUNCT/SUNA</td>
<td>1) Trigeminal Neuralgia (TN)</td>
<td>Autonomic features and agitation are more prominent in SUNCT/SUNA, plus no refractory period between attacks as in TN</td>
</tr>
<tr>
<td></td>
<td>2) CH or PH (groups of stabs of SUNCT/SUNA)</td>
<td>Cutaneous triggering more common in SUNCT/SUNA; also characterisation of the attack- stab/group of stabs/sawtooth</td>
</tr>
<tr>
<td>HC</td>
<td>1) CH with background pain</td>
<td>HC responds absolutely to indomethacin</td>
</tr>
<tr>
<td></td>
<td>2) Migraine with chronic background pain</td>
<td></td>
</tr>
</tbody>
</table>