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Teaching Course 7

Acute headache treatment (Level 1)

Management in the emergency room

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Management in the emergency room

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Teaching course « Acute headache treatment » EAN OSLO 2019

Disclosure of interest

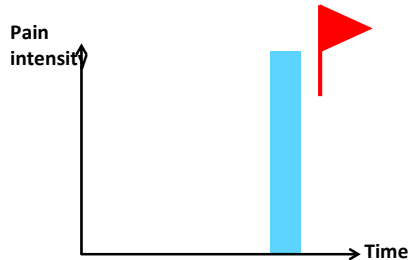
Allergan, Almirall SAS, Astellas, Grunenthal, Lilly, MSD, Novartis, Orkyn,
Pfizer, Saint-Jude, Sanofi-Aventis, Teva, Zambon



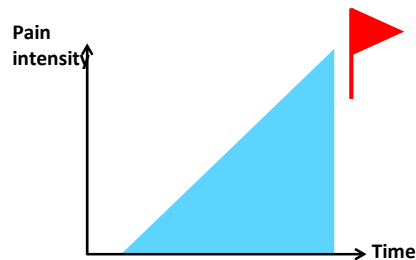
- In the majority of cases—even for patients seen in an emergency setting—headache is a **primary disorder**
- Primary headache
 - can be highly disabling but
 - does not constitute a vital risk
- What is important is **to identify secondary headache**
 - in order to institute adapted treatment
 - sometimes emergency treatment

Until evidence to the contrary, sudden-onset and/or unusual headaches should be considered as secondary disorders warranting emergency complementary exploration

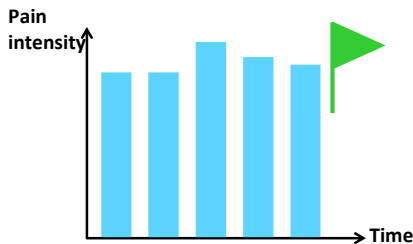
Four clinical presentations



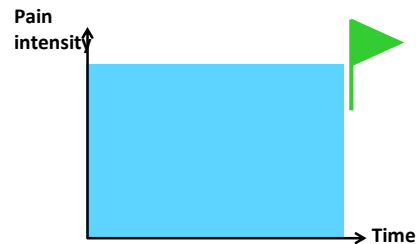
Recent sudden-onset headache



Recent progressive headache



Recurrent paroxysmal headache



Chronic daily headache

The check list



1. When did the pain start? (**recent disorder**)
2. How long did it take for the pain to reach peak intensity? (**sudden-onset disorder**)
3. Have you already had this kind of pain? (**unusual disorder**)
4. **What were you doing** when the pain started? (exercise. . .)
5. **Are there other symptoms**: fever, photo- or phonophobia, vomiting, neck pain, focal neurological deficit, slow psycho-motor response, girdle pain?
6. **Is there a particular context?**:
 - new medication, toxic substances, exposure to carbon monoxide
 - trauma
 - pregnancy or post-partum
 - cancer, systemic disease (including HIV)
 - dural injury . . .
7. **Does body position affect the pain** (relief or worsening in the supine position)?

Moisset et al. Rev Neurol (Paris). 2016 Jun-Jul;172(6-7):350-60

Physical examination



1. Impaired vigilance
2. Fever
3. Hypertension
4. Meningeal syndrome
5. Focal neurological deficit (motor or sensorial deficit, diplopia, pupil anomaly, cerebellar syndrome)
6. Disorders affecting the eyes, sinuses, ears or the oral cavity that could explain the headache

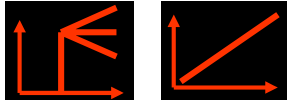
Moisset et al. Rev Neurol (Paris). 2016 Jun-Jul;172(6-7):350-60

Two key questions

- Have you already had this kind of pain?
- When did the pain start?

1. **NO!** I have never had this before and the pain started a few hours, days or weeks ago

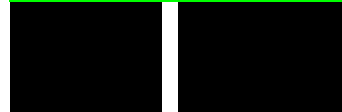
What were you doing when the pain started?



SECONDARY HEADACHE??
= complementary explorations

2. **YES!** I have the same headaches for months or years

Do you have pain between the attacks ?

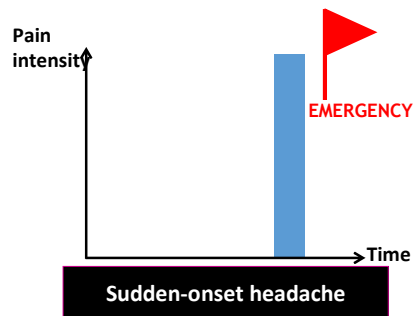


PRIMARY HEADACHE
= clinical diagnosis and specific treatment

EMERGENCY CARE is recommended for all patients presenting:

- **Sudden-onset or thunderclap headache** (peak intensity within one minute)
- **Recent-onset or recently worsening** (< 7 days) unusual headache
- Headache associated with **fever** (without other obvious general cause of fever such as a flu-like syndrome in an outbreak context)
- **Headache associated with neurological signs**
- **Headache suggestive of intoxication** (particularly carbon monoxide)
- **Headache in a context of immune deficiency**

I. Sudden-onset headaches



Sudden-onset headache

- SECONDARY HEADACHE**
- Headache is the **only** clinical sign in one-third of patients with subarachnoid hemorrhage
 - Until evidence to the contrary, all patients complaining of headache who fulfill one criterion of the Ottawa clinical decision rule should be suspected of having **subarachnoid hemorrhage**

Perry et al., 2013

The Ottawa clinical decision rule

The rule

- 1- Age \geq 40 years
- 2- Neck pain or stiffness
- 3- Witnessed loss of consciousness
- 4- Onset during exertion
- 5- Thunderclap headache (intensity $>$ 7/10 in less than 1 min)
- 6- Limited neck flexion

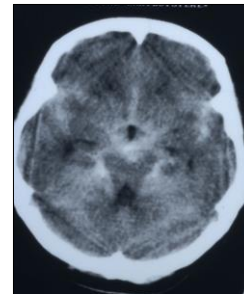
The conditions

- Exploration to search for subarachnoid hemorrhage is warranted in all patients presenting **at least one of the six criteria**
- This rule is:
 - for patients aged over 15 years
 - who present with severe non-traumatic headache
 - that reached peak intensity in less than one hour

The rule has an excellent negative predictive value, but poor specificity

Sudden onset headache: complementary explorations

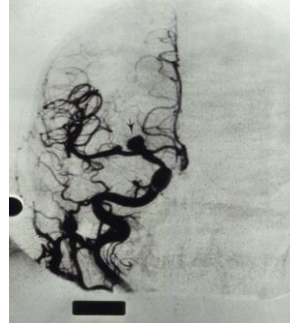
- A patient who presents with **sudden-onset headache** or **headache associated with a neurological deficit** should have an **emergency CT scan**
- **Positive diagnosis of subarachnoid hemorrhage** is confirmed by the cerebral CT scan without contrast injection
 - 98% sensitivity when performed within **12 h of symptom onset**
 - 93% after **24 h**
 - 85% at **5 days**
 - 50% at **7 days**



Edlow et al., 2008

Sudden onset headache: complementary explorations

- CT scan without contrast injection is indispensable, but **not sufficient**
- Cerebral CT angiography
 - should also be performed in all patients
 - in order to explore the **arterial and the venous networks**
- MRI with MR angiography can be the first-line exploration if therapeutic management is not delayed



Edlow et al., 2008

Sudden onset headache: complementary explorations

- If the CT angiography (or MR angiography) does not provide evidence for the diagnosis, a **lumbar puncture** should always be performed, even if the headache has subsided
- A lumbar puncture using an atraumatic 25-gauge needle is recommended in order to reduce the risk of postlumbar puncture headache and limit further induced costs



Edlow et al., 2008; Steiner et al., 2013; Stewart et al., 2014; Davis et al., 2014; Lavi et al., 2006; Struppet al., 2001; Tung et al., 2012

Sudden onset headache: complementary explorations

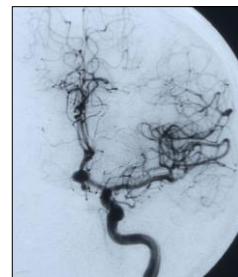
- If no diagnosis is established after the lumbar puncture, the initial imaging will be completed by **imaging of the supra-aortic trunks**
- Thunderclap headache can also be the inaugural sign of:



- cervical artery dissection
- cerebral venous thrombosis
- reversible cerebral vasoconstriction syndrome (RCVS)
- pituitary apoplexy, ...

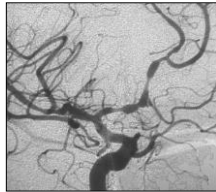
Diagnosis strategy: patients with *proved* subarachnoid hemorrhage

- CT or MR angiography
- Arteriography may be discussed on a case-by-case basis



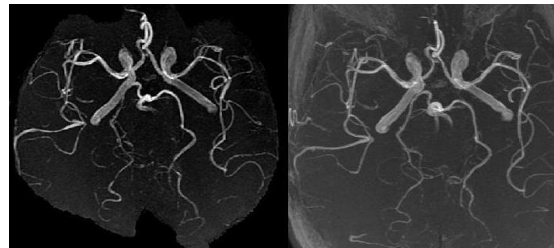
Sudden onset headache: reversible cerebral vasoconstriction syndrome (RCVS)

- Severe, acute, recurrent **thunderclap headache**
- **Short lasting 1 -3hours**
- **No vascular malformation**
- Often triggered by **sexual activity or Valsava maneuvers**
- **Transient reversible abnormal regulation of cerebral arterial tone**
- Precipitants:
 - illicit drugs (cannabis, cocaine, ecstasy, amphetamines, LSD)
 - antidepressants (serotonin or serotonin and noradrenalin; reuptake inhibitors)
 - nasal decongestants
 - triptans and ergotamine



RCVS

- Association with hemorrhage, ischemia or cerebral artery dissection
- **Diagnosis requires demonstration of typical arterial anomalies on the CT or MRI angiogram**
- The first imaging exploration may be **normal** if performed early during the first 4–5 days after symptom onset
- Anomalies reach a maximum **2–3 weeks** after the first symptoms



Diffuse and multifocal vasoconstriction and vasodilatation

RCVS: treatment

- Symptomatic management
- Rest (even in the purely cephalalgic forms)
- Avoid sexual activity, physical exertion, Valsalva manoeuvres, and other headache triggers
- Any vasoactive drugs should be stopped and avoided even after disease resolution
- Analgesics, antiepileptic drugs for seizures
- Admission to intensive-care units in severe cases
- Monitoring of blood pressure
 - treatment of hypertension according to the guidelines
 - hypotension in the setting of cerebral vasoconstriction is potentially more dangerous

Ducros A. Lancet Neurol 2012; 11: 906–17

RCVS: treatment

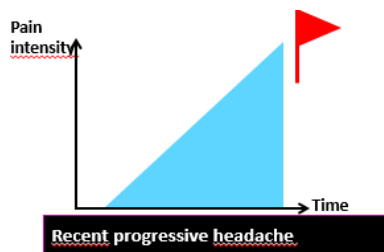
- Oral or IV nimodipine prescribed immediately for suspected RCVS
- Dose used for the prevention of vasospasm in aneurysmal subarachnoid haemorrhage
- Initial treatment started at 30 mg every 8–12 h per day (median, 1.5 mg/kg/day)
- Dose of nimodipine unchanged for 1–3 months
- Avoid glucocorticoids (risk of deterioration of the clinical course)

Ducros A. Lancet Neurol 2012; 11: 906–17

The most common etiologies of sudden-onset headache

	Potentially associated clinical features	Complementary explorations
Subarachnoid hemorrhage	Meningeal syndrome Isolated third nerve palsy Loss of consciousness	Brain MRI + brain arterial angiogram sequences Brain CT ± CT brain angiogram ± lumbar puncture Arteriography
Other intracranial bleeding	Focal signs that can be discrete in certain localizations (cerebellum)	Brain MRI ± brain CT ± brain CT angiogram
Brain infarction	Focal signs that can be discrete in certain localizations (cerebellum)	Brain MRI + brain arterial angiogram sequences Brain CT ± CT brain angiogram
Reversible cerebral vasoconstriction syndrome (RCVS)	Repeated episodes occurring spontaneously or induced by exercise, Valsalva maneuver, Before or with orgasm Possible focal signs or epilepsy	Brain MRI + brain arterial angiogram sequences Brain CT ± CT brain angiogram ± lumbar puncture ± arteriography
Cerebral venous thrombus	Neurological deficit Epileptic seizure	Brain MRI + brain arterial angiogram sequences Brain CT ± CT venous angiogram
Cervical arterial dissection	Focal signs, intracranial hypertension Neck pain, Claude-Bernard-Horner sign, pulsating tinnitus	Brain CT + CT angiogram of the superior aortic trunks (SAT), Ultrasound-Doppler of the SAT, Brain MRI + MRI angiogram of the head and neck Lumbar puncture
Meningitis ± encephalitis	Fever, meningeal syndrome, cranial nerve involvement	
Hypertensive encephalopathy and eclampsia, posterior reversible encephalopathy syndrome (PRES)	Headache followed by perturbed consciousness, focal deficit, seizures High blood pressure, 240/120 mmHg (lower if eclampsia)	Ocular fundus: papillary edema Brain MRI
Pituitary necrosis	Vision disorders, oculomotor disorders	Brain MRI
Temporal arteritis	(sudden-onset rare) Age > 50 yr Impaired general condition	C-reactive protein: elevated Temporal artery biopsy

II. Progressive unusual headaches



Progressive unusual headache (onset or worsening within the last 7 days)

History taking and physical examination (with ocular fundus):

- **signs of intracranial hypertension**
 - headache particularly intense at awakening in the morning
 - vomiting, visual blurring, papillary edema at the ocular fundus)
- **neck pain** suggestive of dissection of the cervical arteries
- **orthostatic headache**



Progressive unusual headache (onset or worsening within the last 7 days)

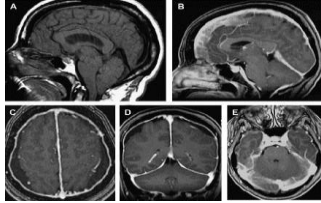
SECONDARY HEADACHE

- Brain MRI with T1, T2, FLAIR, and T1 injected sequences
 - contrast uptake
 - signs of venous thrombosis
- T2* sequence
 - identify potential bleeding
 - search for venous thrombosis
- The supra-aortic trunks will be explored to search for dissection, warranting a fat-saturation sequence but also MR angiography of the supra-aortic trunks
- If MRI is easily accessible, CT is not needed

Progressive unusual headache (onset or worsening within the last 7 days)

PRESSURE ↓

- Orthostatic headache
- Cerebral MRI



- Lumbar puncture should not be made if spontaneous intracranial hypotension is suspected

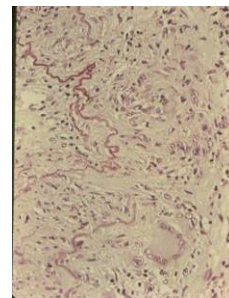
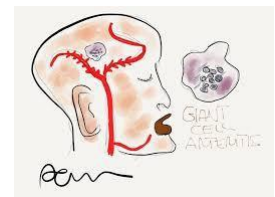
PRESSURE ↑

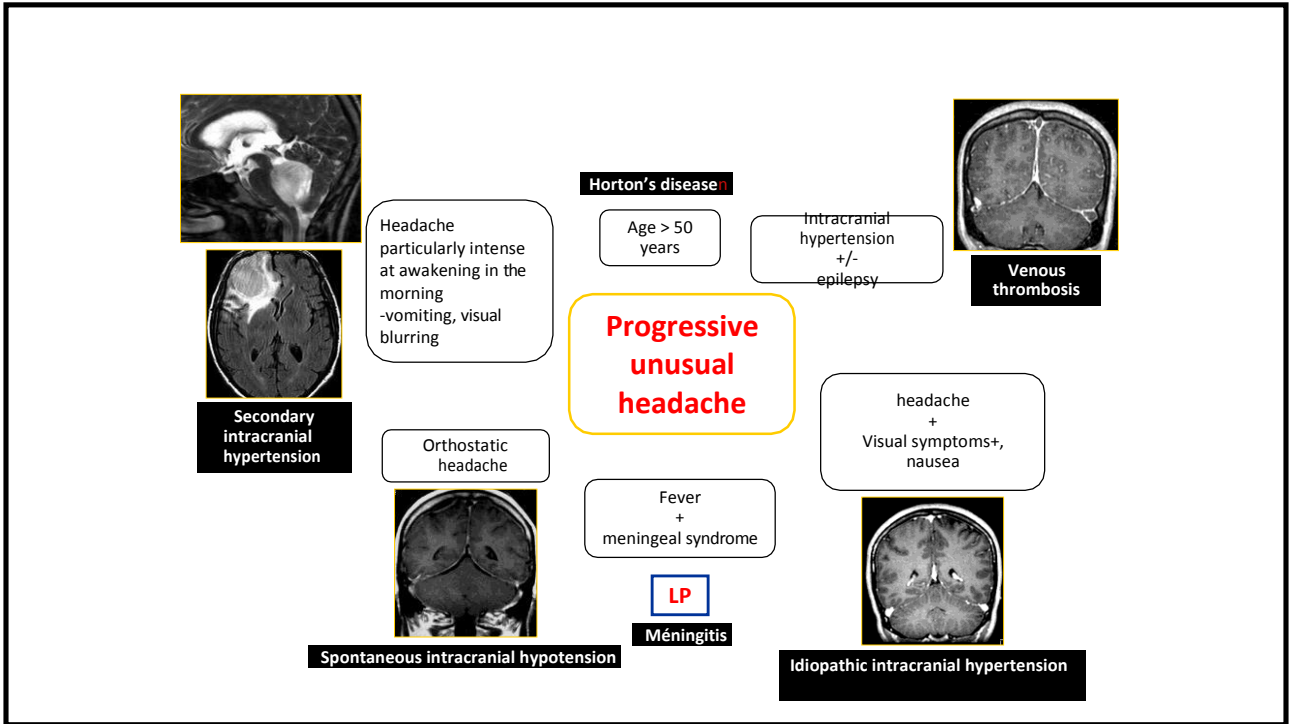
- Brain imaging
- If intracranial hypertension is suspected and imaging does not provide the diagnosis, CSF analysis (with pressure) is needed
- Ocular fundus



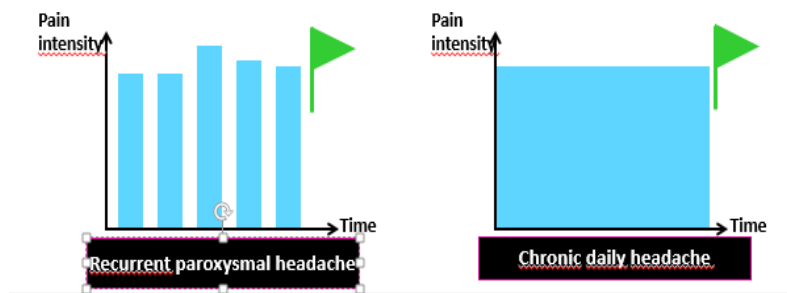
Progressive unusual headache (onset or worsening within the last 7 days)

- In patients aged over 50 years, blood tests will include:
 - cell counts
 - electrolytes
 - liver tests
 - C-reactive protein
- An inflammatory syndrome is suggestive of giant-cell arteritis (Horton's disease)





III. Recurrent paroxysmal headaches



Clinical features for the diagnosis of primary headache

Table 1 – Main clinical features for the diagnosis of migraine, tension-type headache and cluster headache.

	Migraine	Tension-type headache	Cluster headache
Duration	4-72 h	30 min-7 days	15 min-3 h
Site	Generally unilateral	Bilateral	Orbito-temporal unilateral
Intensity	Moderate to severe	Mild to moderate	Very severe
Type	Often pulsating	Compression, band-like pressure	Boring, squeezing
Accompanying signs	Nausea, vomiting, photo- and phono-phobia	No nausea or vomiting, Photo- or phono-phobia possible	Homolateral autonomic signs, agitated
Impact of exercise	Aggravation	No change	No change
Number of prior attacks	≥ 5	≥ 10	≥ 5

ICHD-3 criteria

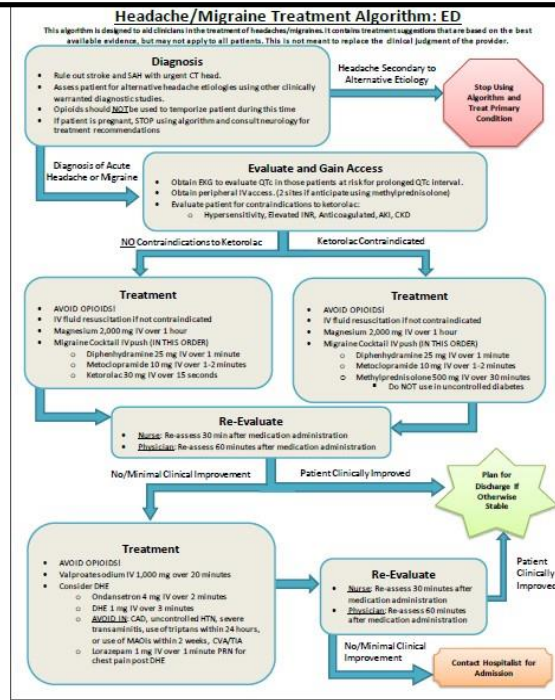
Migraine

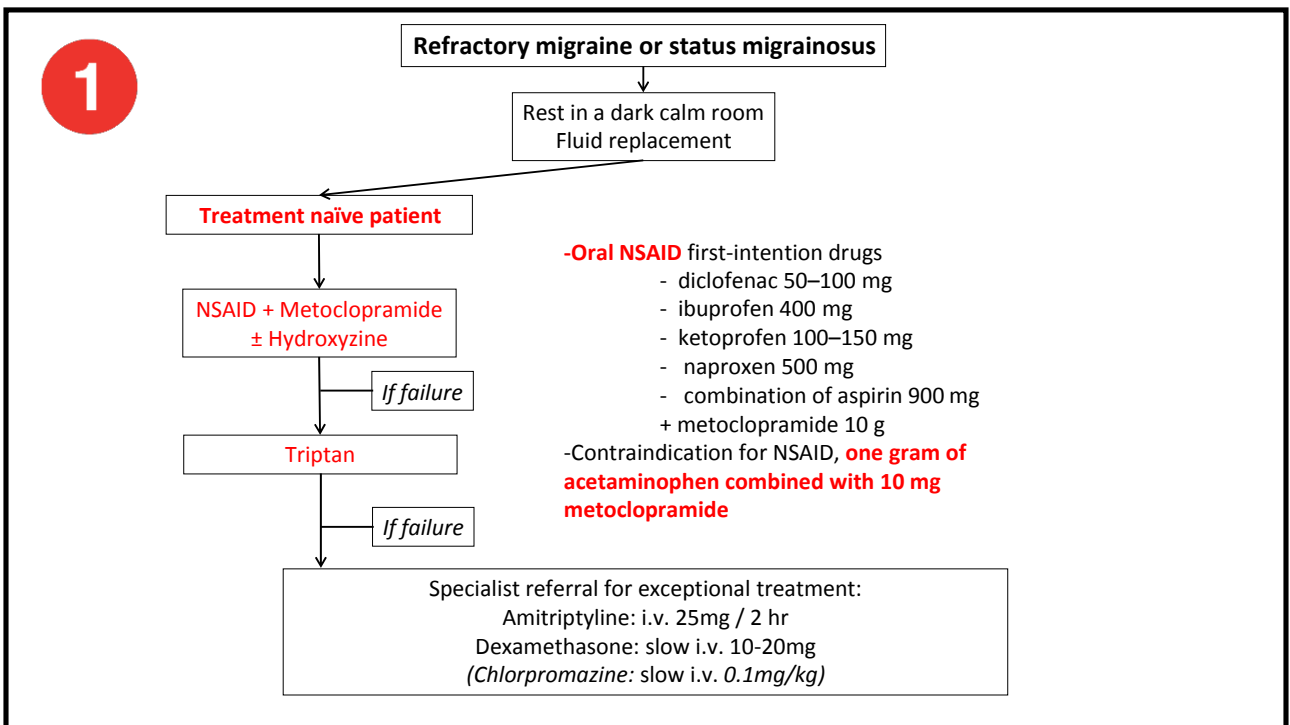
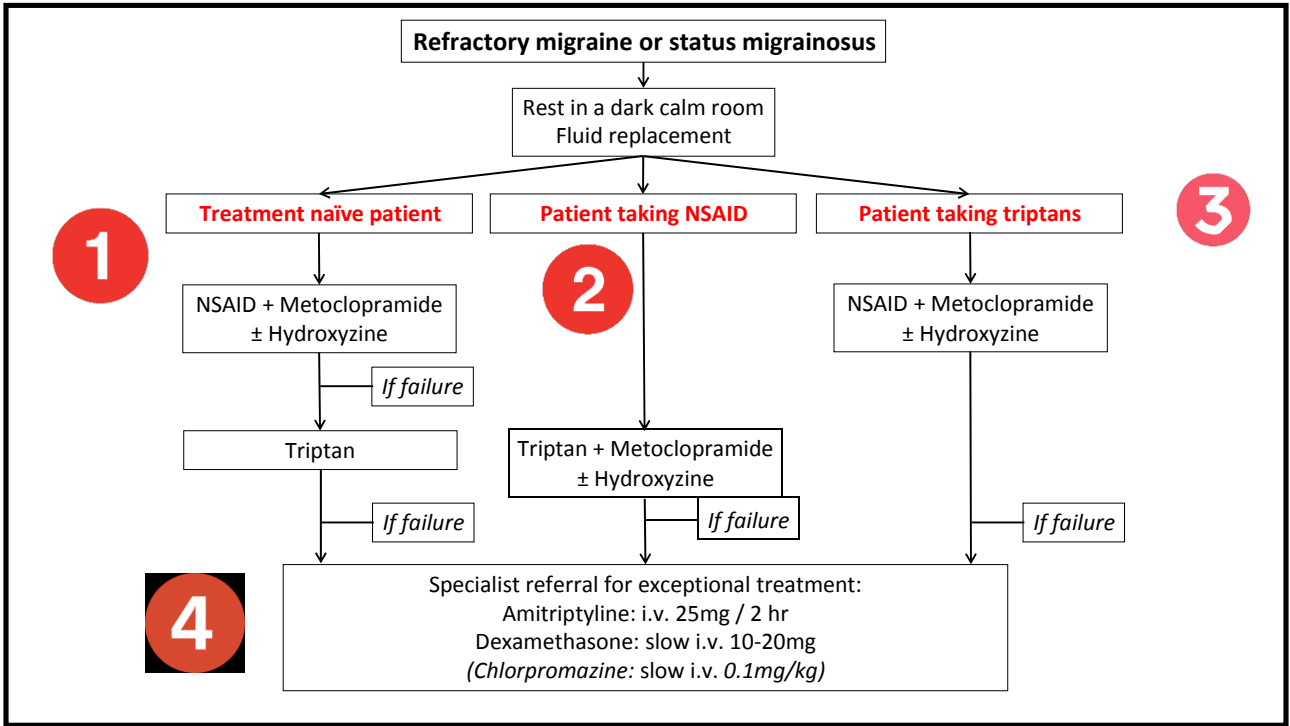
2 rules:

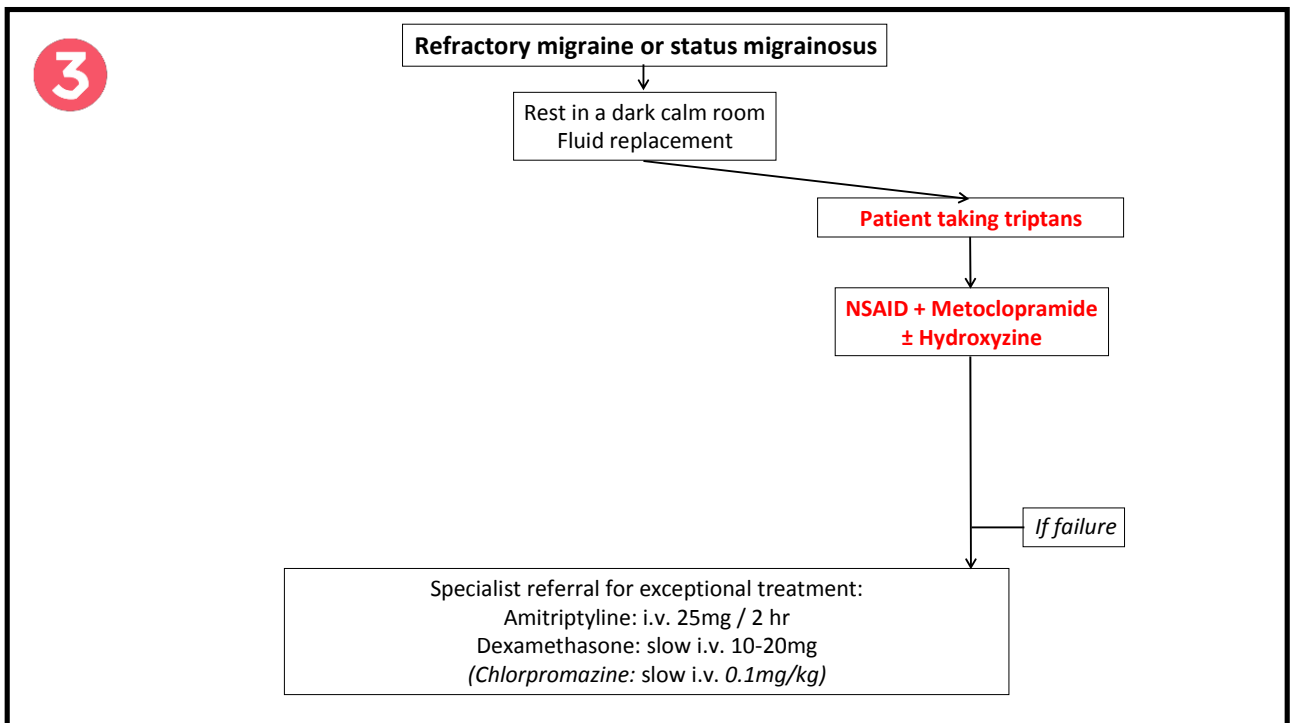
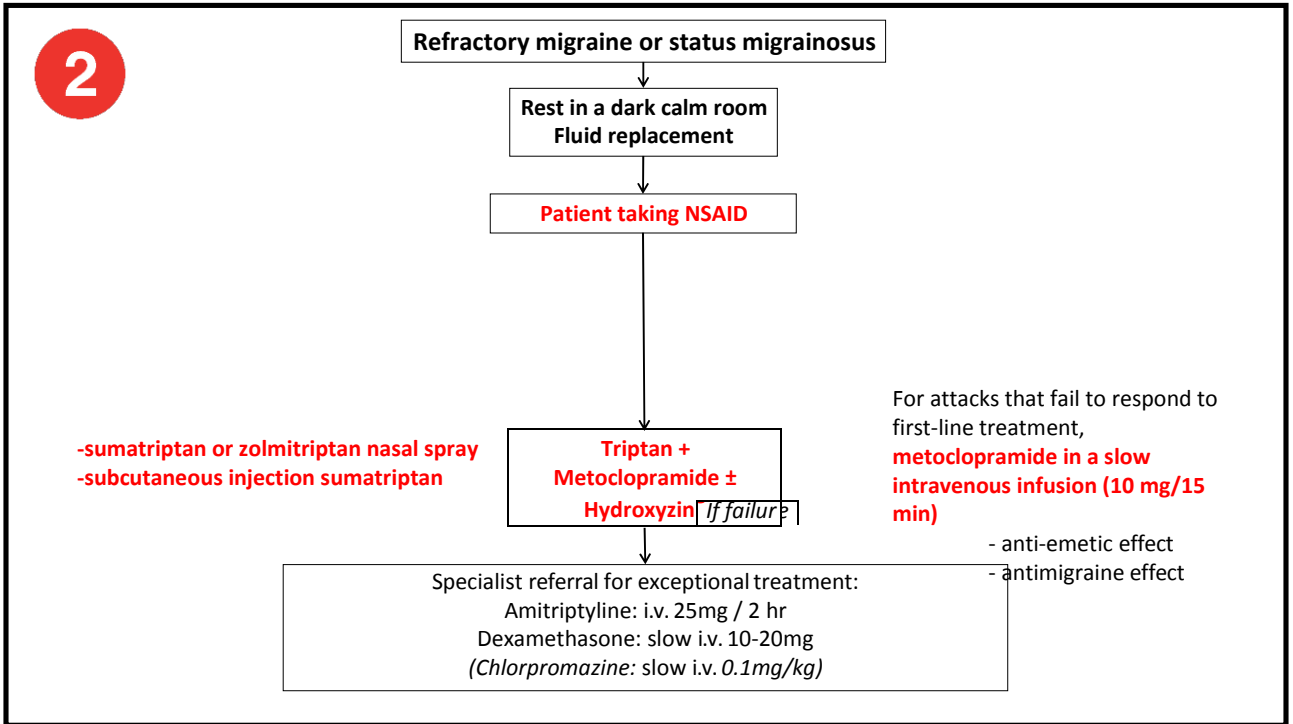
-Response to analgesic treatment is not indicative of a primary cause and should not be considered reassuring

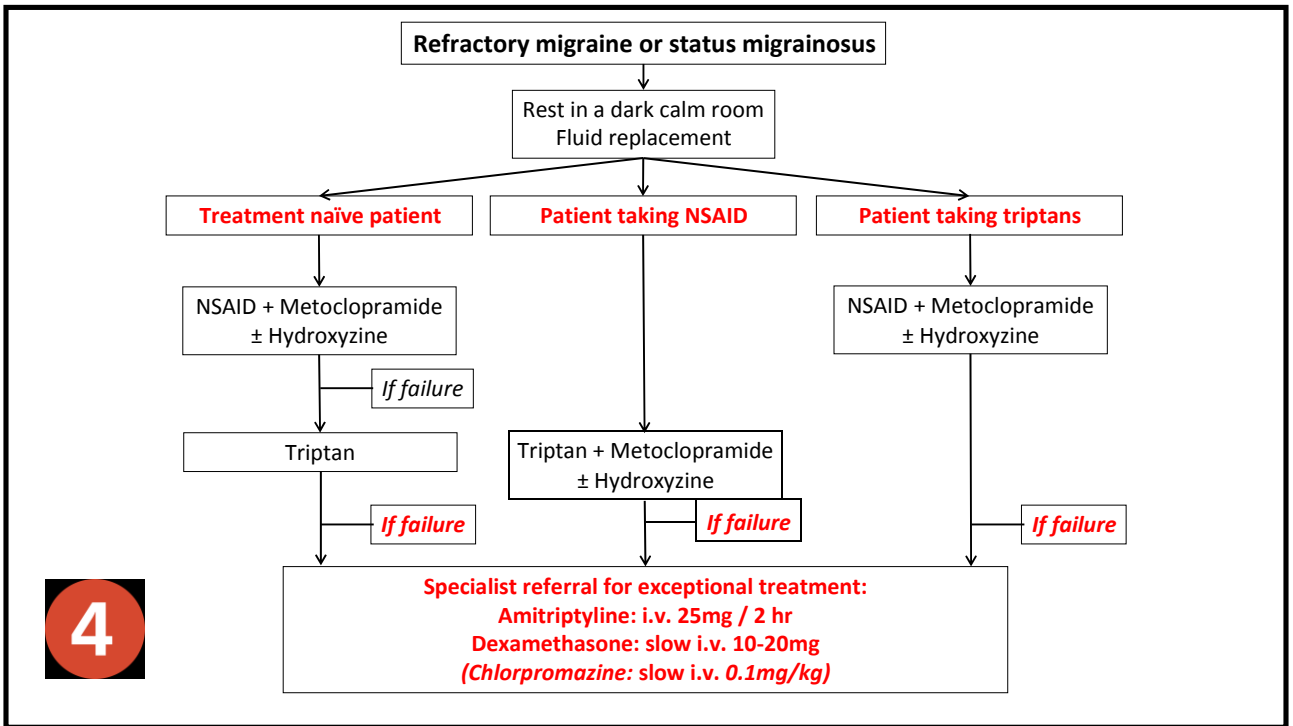
- AVOID OPIOIDS!

increased risk of revisit, hospital admission, and increased ED length of stay









Tension type headache

- First-line treatment relies on use of an analgesic drug (acetaminophen 1 g) or a NSAID (ibuprofen 400 mg or ketoprofen 100 mg)
- As a last resort, intravenous infusion amitriptyline

Cluster headache

- SC sumatriptan 6 mg
- Cxygen (12–15 L/min) using a high-concentration mask for 15–20 min

To conclude...

“WHICH Headache to Investigate, WHEN, and HOW?”

- Less than 10% headaches in practice belong to the category of secondary headaches
- Fear of missing a treatable serious secondary headache disorder