BLOCK: Mental health care and Neurology

Neurology section lec.

headache

Block staff:

Dr. Ali Mohammed Radi \neurosurgeon.

Dr. Zainab Abdui-muhsin Abood/ neurosergen.

Davidson s principles and practice of medicine

Objective

1. What is primary and 2nd headache?
2. What is migraine and how we can diagnose it from history and what is line of treatment and prophylaxis.
3. What is cluster headache and it is management?
4. How can you mange sudden sever headache ?
5. SAH ; most common cause and if brain CT is negative; what is next?
6. What is temporal arteritis?

Headache

Headaches may be **classified as primary or secondary**, depending on the underlying cause. Secondary headache may be due to structural, infective, inflammatory or vascular conditions.

7.1 Clinical characteristics of headache syndromes

	Onset	Duration/periodicity	Pain location	Associated features
Primary syndromes				
Migraine	Evolves over 30–120 min	Usually last <24 h, recurrent with weeks/months symptom-free	Classically unilateral but may be anywhere including face/neck	Aura (usually visual), nausea/vomiting, photophobia and phonophobia
Cluster headache	Rapid onset, often waking patient from sleep	30–120 min, 1–4 attacks within 24 h, clusters usually last weeks to months, with months to years of remission	Orbital/retro-orbital; always same side during cluster, may switch sides between clusters	Autonomic features, including conjunctival injection, tearing, nasal stuffiness, ptosis, miosis, agitation
Stabbing headache	Abrupt, rarely from sleep	Very brief, seconds or less	Anywhere over head	Common in migraineurs
Secondary syndromes				
Meningitis	Usually evolves over a day or two, can be abrupt	Depends on cause and treatment, usually days to weeks	Global, including neck stiffness	Fever, meningism, rash, false localising signs, signs of raised intracranial pressure
Subarachnoid haemorrhage	Abrupt, immediately maximal, rare from sleep	May be fatal at onset, usually days to weeks	Anywhere, poor localising value	20% isolated headache only; nausea/vomiting, reduced consciousness, false localising signs, III nerve palsies
Temporal arteritis	Gradual onset of temple pain and scalp tenderness	Continuous	Temple and scalp	Usually in those >55 years; unwell, jaw pain on chewing, visual symptoms, tender temporal arteries, elevated erythrocyte sedimentation rate and C-reactive protein

Tension-type headache

This is the most common type of headache and is experienced to some degree by the majority of the population.

Pathophysiology

Tension-type headache is incompletely understood, and some consider that it is simply a milder version of migraine; certainly, the original notion that it is due primarily to muscle tension.

Anxiety about the headache itself may lead to continuation of symptoms, and patients may become convinced of a serious underlying condition.

Clinical features

The pain of tension headache is characterized as '**dull**', '**tight**' or like a '**pressure**', and there may be a sensation of a **band round the head** or pressure at the vertex. It is of constant character and generalized, but often radiates forwards from the occipital region. It may be episodic or persistent, although the severity may vary, and there is no associated vomiting or photophobia

Tension-type headache is rarely disabling and patients appear well. The pain often progresses throughout the day. Tenderness may be present over the skull vault or in the occiput but is easily distinguished from the triggered pains of trigeminal neuralgia and the exquisite tenderness of temporal arteritis. Analgesics may be taken with chronic regularity, despite little effect.

Management

Most benefit is derived from a **careful assessment**, followed by discussion of likely precipitants and **reassurance** that the prognosis is good. **The concept of medication overuse headache needs careful explanation**. An important therapeutic step is to allow patients to realize that their problem has been taken seriously and rigorously assessed. **Physiotherapy** (with muscle relaxation and stress management) may help and low-dose amitriptyline can provide benefit. **Investigation is rarely required**.

Migraine

Migraine usually appears before middle age, or occasionally in later life; it affects about 20% of females and 6% of males at some point in life. **Migraine is usually readily identifiable from the history**, although unusual variants can cause uncertainty.

Pathophysiology

The cause of migraine is unknown but there is increasing evidence that the aura is due to dysfunction of ion channels causing a spreading front of cortical depolarization (excitation) followed by hyperpolarization (depression of activity). The headache phase is associated with **vasodilatation of extracranial vessels** and may be relayed by hypothalamic activity.

- A genetic contribution is implied by the frequently positive family history.
- The female preponderance and the frequency of migraine attacks at certain points in the menstrual cycle also suggest hormonal influences.
- Oestrogen-containing oral contraception sometimes exacerbates migraine and increases the very small risk of stroke in patients who suffer from migraine with aura.

- Dietary precipitants such as cheese, chocolate or red wine.
- When psychological factors contribute, the migraine attack often occurs after a period of stress, being more likely on Friday evening at the end of the working week or at the beginning of a holiday.

Clinical features

- Some patients report a **prodrome** of malaise, irritability or behavioral change for some hours or days.
- Around **20% patients experience an aura** and are said to have migraine with aura (previously known as classical migraine). The aura may manifest as almost any neurological symptom but is most often visual, consisting of fortification spectra, which are usually positive phenomena such as shimmering, silvery zigzag lines marching across the visual fields for up to 40 minutes, sometimes leaving a trail of temporary visual field loss (scotoma). Sensory symptoms characteristically spreading over 20–30 minutes, from one part of the body to another, are more common than motor ones, and language function can be affected, leading to similarities with TIA/stroke.
- Isolated aura may occur (i.e. the neurological symptoms are not followed by headache).
- The 80% of patients with characteristic headache but no 'aura' are said to have migraine without aura (previously called 'common' migraine).

- Migraine **headache** is usually **severe and throbbing**, with photophobia, phonophobia and vomiting lasting from 4 to 72 hours. Movement makes the pain worse and patients prefer to lie in a quiet, dark room.

- In a small number of patients the aura may persist, leaving more permanent neurological disturbance. This persistent migrainous aura may occur with or without evidence of brain infarction.

Management

1. **Avoidance of identified triggers** or exacerbating factors (such as the combined contraceptive pill) may prevent attacks.

2. Treatment of an acute attack consists of simple analgesia with aspirin, paracetamol or non-steroidal anti-inflammatory agents.

✓ Nausea may require an antiemetic such as metoclopramide or domperidone.

✓ Severe attacks can be aborted by one of the 'triptans' (e.g. sumatriptan), which are potent 5 hydroxytryptamine(5-HT, serotonin) agonists. These can be administered via the oral, subcutaneous or nasal route. Caution is needed with ergotamine preparations because they may lead to dependence.

Overuse of any analgesia, including triptans, may contribute to medication overuse headache.

3. If attacks are frequent (more than two per month**), prophylaxis** should be considered. Many drugs can be chosen but the most frequently used are vasoactive drugs (β-blockers), antidepressants (amitriptyline, dosulepin) and antiepileptic drugs (valproate, topiramate). Women with aura should avoid oestrogen treatment for either oral contraception or hormone replacement, although the increased risk of ischaemic stroke is minimal.

Cluster headache

Cluster headaches (also known as migraineurs neuralgia) are much less common than migraine. Unusually for headache syndromes, there is a significant **male** predominance and onset is usually in the **third decade**.

Pathophysiology

The cause is unknown but this type of headache differs from migraine in many ways, suggesting a different pathophysiological basis. Functional imaging studies have suggested abnormal hypothalamic activity. Patients are more often smokers with a higher than average alcohol consumption.

Clinical features

Cluster headache is **strikingly periodic**, featuring runs of **identical headaches** beginning at **the same time for weeks** at a stretch (the 'cluster'). Patients may experience either one or several attacks within a 24-hour period, and typically are awoken from sleep by symptoms ('alarm clock headache'). Cluster headache causes severe, unilateral periorbital pain with autonomic features, such as ipsilateral tearing, nasal congestion and conjunctival injection (occasionally with the other features of a Horner's syndrome). The pain, though severe, is characteristically brief (30–90 minutes). In contrast to the behavior of those with migraine, patients are highly agitated during the headache phase.

The cluster period is typically a few weeks, followed by remission for months to years, but a small proportion do not experience remission.

Management

Acute attacks can usually be halted by subcutaneous injections of sumatriptan or inhalation of 100% oxygen. The brevity of the attack probably prevents other migraine therapies from being effective. Migraine prophylaxis is often ineffective too but attacks can be prevented in some patients by verapamil, sodium valproate, or short courses of oral glucocorticoids. Patients with severe debilitating clusters can be helped with lithium therapy, although this requires monitoring.

Subarachnoid haemorrhage

Subarachnoid haemorrhage (SAH) is less common than ischaemic stroke or intracerebral haemorrhage). **Women are affected more** commonly than men and the condition usually presents **before the age of 65**. The immediate mortality of aneurysmal SAH is about 30%; survivors have a recurrence (or rebleed) rate of about 40% in the first 4 weeks and 3% annually thereafter.

Some 85% of cases of SAH are caused by saccular or 'berry' aneurysms arising from the bifurcation of cerebral arteries ,particularly in the region of the circle of Willis. There is an increased risk in first-degree relatives of those with saccular aneurysms, and in patients with polycystic kidney disease and congenital connective tissue defects such as Ehlers–Danlos syndrome .

In about 10% of cases, SAHs are non-aneurysmal haemorrhages (so-called peri-mesencephalic haemorrhages), which have a very characteristic appearance on
CT and a benign outcome in terms of mortality and recurrence. Around 5% of SAHs are due to arteriovenous malformations and vertebral artery dissection.

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Clinical features

SAH typically presents with a sudden, severe, 'thunderclap' headache (often occipital), which lasts for hours or even days, often accompanied by vomiting, raised blood pressure and neck stiffness or pain. It commonly occurs on physical exertion, straining and sexual excitement. There may be loss of consciousness at the onset, so SAH should be considered if a patient is found comatose. On examination, the patient is usually distressed and irritable, with photophobia. There may be neck stiffness due to subarachnoid blood but this may take some hours to develop.

Focal hemisphere signs, such as hemiparesis or aphasia, may be present at onset if there is an associated intracerebral haematoma.

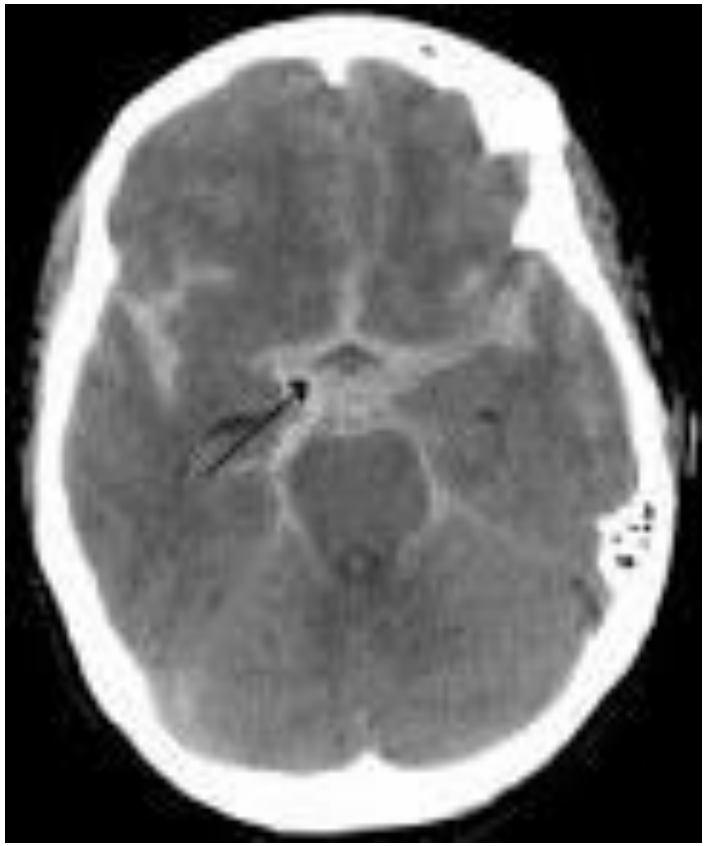
A third nerve palsy may be present due to local pressure from an aneurysm of the posterior communicating artery, but this is rare. Fundoscopy may reveal a subhyaloid haemorrhage, which represents blood tracking along the subarachnoid space around the optic nerve.

Investigations

CT brain scanning and **lumbar puncture** are required. The diagnosis of SAH can be made by CT but a negative result does not completely exclude it, since small amounts of blood in the subarachnoid space cannot be detected by CT. Lumbar puncture should be performed 12 hours after symptom onset if possible, to allow detection of xanthochromia. If either of these tests is positive, **cerebral angiography** is required to determine the optimal approach to prevent recurrent bleeding.

Management

Admission to ICU , observation of vital sign and conscious level and serum electrolyte, avoid dehydration , antiepileptic medication , cardiac monitoring because of risk of arrythmias, Nimodipine tab, avoid anxiety and constipation and surgical management of aneurysm with in first 24h.



Temporal arteritis

Temporal arteritis is a form of vasculitis ,also known as giant cell arteritis or Horton's arteritis, the blood vessels near the temples, which supply blood from the heart to the scalp, are inflamed (swollen) and constricted (narrowed). The vasculitis that causes temporal arteritis can involve other blood vessels, such as the posterior ciliary arteries (leading to blindness), or large blood vessels like the aorta and its branches, which can also lead to serious health problems.

If not diagnosed and treated quickly, temporal arteritis can cause:

- sudden blindness in one or both eyes.
- Damage to blood vessels, such as an aneurysm.
- Other disorders, including stroke or transient ischemic attacks.

Temporal arteritis is one of the most common vascular disorders, but is a relatively rare condition. It usually occurs in people who are over 50 years old, and affects women more often than men.

Clinical presentation

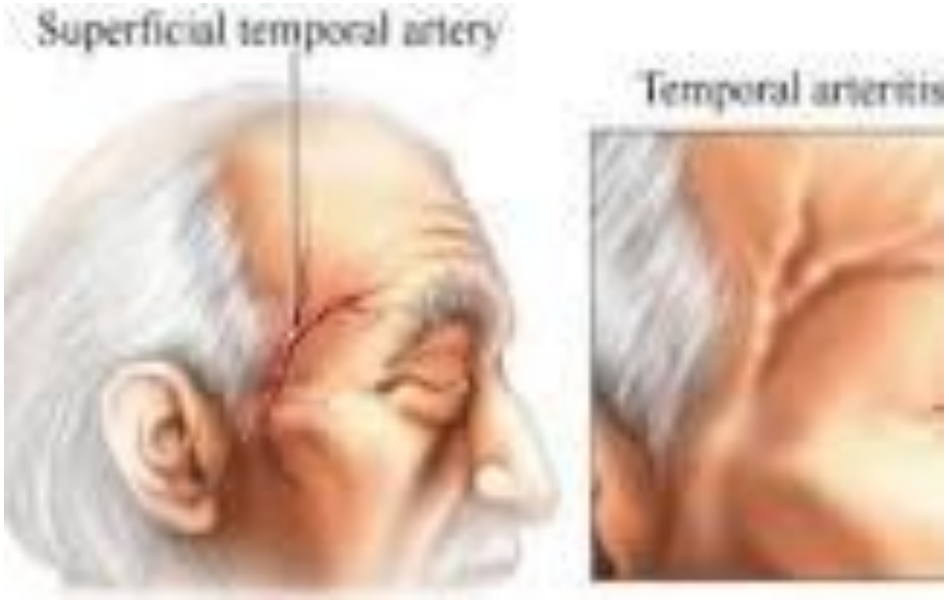
The most common symptom of temporal arteritis is a throbbing, continuous headache on one or both sides of the forehead. Other symptoms may include:

- Fatigue.
- Fever.
- Jaw pain that may become worse after chewing.
- Tenderness at the scalp or temples.
- Vision problems, such as double vision, blurry vision, or transient (brief) vision loss; if this is not treated, it could be followed by permanent, irreversible vision loss.
- Muscle aches in the upper arms or shoulders, hips, upper thighs, lower back, and buttocks.
- Loss of appetite or weight loss.

Often, temporal arteritis can be associated with an entity called polymyalgia rheumatica (PMR), which is an inflammatory condition affecting the shoulders, hip girdle and neck. This leads to significant stiffness and pain. PMR is far more common than temporal arteritis, but up to 30 percent of temporal arteritis patients have PMR.

Investigations

- Elevated ESR and CRP.
- Doppler us of temporal artery
- Biopsy of temporal artery.



Management

The mainstay of therapy for temporal arteritis is glucocorticoids, such as prednisolone. Patients sometimes need to take glucorticoids for up to two years, sometimes longer; the dosage is gradually reduced over this period. Treatment should start even before biopsy to avoid risk of blindness.
Immunosuppressant agent can be used as second line.

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