Migraine

Migraine is recurrent headache associated with visual and gastrointestinal disturbance. The borderline between migraine and tension headaches is vague. Over 12% of any population world-wide report these symptoms

The prevalence of migraine is about 20% in women and 6% in men. All varieties of migraine may begin at any age from early childhood on, although peak ages at onset are adolescence and early adulthood

The term migraine is derived from the Greek word *hemikrania.* This term was corrupted into low Latin as *hemigranea,* which eventually was accepted by the French translation as migraine..

Mechanisms

1- Precise mechanisms of migraine remain unknown.
   Genetic factors play some part - a rare form of familial migraine is associated with mutation chromosome 19.
2- The release of the neuropeptide calcitonin-gene-related peptide (CGRP) is thought to play a central role as it is a potent dilator of cerebral and dural vessels
3- The headache of migraine, often throbbing, is due to vasodilatation or oedema of blood vessels, with stimulation of nearby nerve endings.
4- Release of vasoactive substances such as nitric oxide has a role
5- Serum 5-hydroxytryptamine rises with prodromal symptoms and falls during the headache.
6- Cerebral features, such as tingling limbs, aphasia and weakness, are caused by focal depression of cortical function.

Some patients recognize precipitating factors

- week-end migraine (a time of relaxation)
- chocolate (high in phenylethylamine)
- cheese (high in tyramine)
- noise and irritating lights
- with premenstrual symptoms.

Clinical features

Migraine is common around puberty and at the menopause and sometimes increases in severity or frequency with hormonal contraceptives, in pregnancy and with the onset of hypertension. There is no reason to suppose that the development of migraine is suggestive of any serious intracranial lesion. However, since migraine is so common, an intracranial mass and migraine sometimes occur together by coincidence. Migraine sometimes follows a blow to the head - often minor.

Table 15-4 Simplified Diagnostic Criteria for Migraine

Repeated attacks of headache lasting 4–72 h in patients with a normal physical examination, no ot reasonable cause for the headache, and:

At least 2 of the following features:	Plus at least 1 of the following features:
Unilateral pain	Nausea/vomiting
Throbbing pain	Photophobia and phonophobia
Aggravation by movement	
Moderate or severe intensity	

Migraine with aura (classical migraine)

- Prodromal symptoms are usually visual and related to depression of visual cortical function or retinal function. *Unilateral patchy scotomata (retina), hemianopic symptoms (cortex), teichopsia (flashes) and fortification spectra (jagged lines resembling battlements) are common*.
- Transient aphasia sometimes occurs, with tingling, numbness, vague weakness of one side and nausea.
- The prodrome persists for a few minutes to about an hour. Headache then follows. This is occasionally hemicranial (i.e. splitting the head) but often begins locally and becomes generalized. Nausea increases and vomiting follows. The patient is irritable and prefers a darkened room. Superficial temporal arteries are engorged and pulsating. After several hours the migraine settles, sometimes with a diuresis. Deep sleep often ensues.

Migraine without aura (common migraine)

- This is the usual variety. Prodromal visual symptoms are vague. There is recurrent headache accompanied by nausea and malaise.

Rare types

- Basilar migraine
- Hemi paretic migraine
- Ophthalmoplegic migraine
- Facioplegic migraine

Differential diagnosis

- The sudden headache may resemble meningitis or SAH.
- Hemiplegic, visual and hemi sensory symptoms must be distinguished from thromboembolic T.I.As In T.I.As maximum deficit is present immediately and headache is unusual
- Unilateral tingling or numbness may resemble sensory epilepsy (partial seizures). In epilepsy, distinct march (progression) of symptoms is usual.

Management

General measures include:

- reassurance and relief of anxiety
- avoidance of dietary factors - rarely helpful.
- Patients taking hormonal contraceptives may benefit from a brand change, or trying without. Premenstrual migraine may respond to diuretics. Depot estrogens are sometimes used. Severe hemi plegic symptoms are an indication for stopping hormonal contraceptives

Treatment:

- Treatment of an acute attack consists of simple analgesia with aspirin or paracetamol, often combined with an antiemetic such as metoclopramide or domperidone.

- Severe attacks can be treated with one of the 'triptans' (e.g. sumatriptan), 5-HT agonists that are potent vasoconstrictors of the extracranial arteries. These can be administered orally, sublingually, by subcutaneous injection or by nasal spray.
- Ergotamine preparations should be avoided since they easily lead to dependence. This is less likely to happen with the triptans, but it can occur. Women should be warned that the small risk of ischaemic stroke attributable to taking oral contraception is increased if they have migraine (), especially if they also smoke.
- Triptans should be avoided when there is vascular disease, and not overused. A recent study has shown that an i.v. CGRP antagonist was effective in treating severe attacks.

Prophylaxis:

- The following are used when attacks are frequent:[more than 3-4 attacks per month]
- pizotifen (antihistamine and 5-HT antagonist) 0.5 mg at night for several days, increasing to 1.5 mg (common side-effects: weight gain and drowsiness)
- propranolol 10 mg three times daily, increasing to 40-80 mg three times daily
- amitriptyline: 10 mg (or more) at night.
- Sodium valproate, methysergide, SSRIs, verapamil, topiramate, nifedipine and naproxen are also used

Complications of Migraine

- Include chronic migraine, migraine-triggered seizures, migrainous infarction (stroke with migraine), and persistent aura (eg, 30-60 minutes) without infarction.
- Ischemic stroke may occur as a rare but serious complication of migraine.
- In migraines with aura, the risk for hemorrhagic stroke may be possible, but rare.
- Risk factors for stroke include migraine with aura, female sex, cigarette smoking, and estrogen use.

Cluster headache (migrainous neuralgia)

This is some less common than migraine. There is a 5:1 predominance of males and onset is usually in the third decade. **The characteristic syndrome** comprises

periodic, severe, unilateral peri-orbital pain accompanied by unilateral lacrimation, nasal congestion and conjunctiva injection, often with the other features of Horner's syndrome.

The pain, whilst being very severe, is characteristically brief (30-90 minutes). Typically, the patient develops these symptoms at a particular time of day (often in the early hours of the morning). The syndrome may occur repeatedly for a number of weeks, followed by a respite for a number of months before another cluster occurs

Despite intense pain there are no serious squeal. Attacks recur at intervals over several years but tend to disappear after the age of 55. Analgesics are unhelpful. Subcutaneous sumatriptan is the drug of choice. Alternatively oxygen inhalation 7.12 L/min may abort an attack. Most prophylactic migraine drugs are unhelpful. Verapamil, topiramate and lithium carbonate sometimes prevent 'cluster'

Trigeminal neuralgia

- Trigeminal neuralgia (*tic douloureux*) is of unknown cause, seen most commonly in old age.
- It is almost always unilateral. Symptoms Severe paroxysms of knife-like or electric shock-like pain, lasting seconds, occur in the distribution of the fifth nerve.
- Spasms occur many times a day. Each paroxysm is stereotyped, brought on by stimulation of one or more trigger zones in the face. Washing, shaving, a cold wind or eating are examples of trivial stimuli that provoke pain. The face may be screwed up in agony (hence the term *tic).*
- Pain characteristically does not occur at night. Spontaneous remissions last months or years before recurrence, which is almost inevitable.
- Signs There are no signs of trigeminal nerve dysfunction. The corneal reflex is preserved. The history alone make the diagnosis.
- Treatment The anticonvulsant carbamazepine 600-1200 mg daily reduces severity of attacks in the majority. Phenytoin, gabapentin and clonazepam are used, but are less effective**. If drug therapy fails,** surgical procedures (radiofrequency extirpation of the ganglion, neurovascular decompression or sectioning of the sensory root) are useful. Alcohol injection into the trigeminal ganglion or peripheral fifth nerve branches can also be carried out. *Thank you,,,*