

**DEFINITION**

1. **Migraine** is a common condition characterised by paroxysmal headaches. Although the headaches and associated symptoms may be severe, each attack almost always resolves completely.

**CLINICAL MANIFESTATIONS**

2. In about a third of cases the onset is before the age of ten. The diagnosis in children may be difficult and headache, although present, may not be dominant. In children, symptoms include fever, abdominal pain and vomiting.
3. 80% of migraine sufferers have their first attack before the age of 30 and the diagnosis should be made with caution in those where the onset is over the age of 40. Attacks tend to lessen from around the age of 50 years.
4. Migraine attacks consist of all or some of the following:
  - 4.1 Prodrome. Many patients describe a characteristic prodrome before an attack which may take the form of yawning, tiredness, depression or elation.
  - 4.2 Aura. Visual warning symptoms are common. These include teichopsiae (flashing zig-zag lights), scotomata, hemianopia or fragmentation of images. Patients may describe a shimmering heat-haze appearance. Sensory symptoms include tingling round the mouth and lips, pins and needles in the hand spreading to the arm, the face and sometimes in the legs. Hemiparesis and dysphasia may also occur. Resolution of these symptoms within 20-40 minutes is the rule.
  - 4.3 Headache, typically unilateral and on the other side of the head from the aura, follows and may be very severe. It lasts 12-48 hours, is worsened by exertion, jarring, light and noise and eased by quiet, darkness and sleep.
  - 4.4 Associated symptoms. About half the sufferers vomit during an attack. Rest lessens the pain and sleep is an integral part of the attack, heralding recovery. In some people, visual symptoms, hemiparesis and dysphasia may be prolonged, persisting after resolution of the headache. Epilepsy is associated with migraine. The incidence of epilepsy is higher in migraine patients than in controls.
5. The above describes what is known as **classic migraine**. If similar paroxysmal symptoms occur without the aura, this is known as **common migraine**. It is common for migraine sufferers to have tension headaches between the attacks of true migraine.

6. There are a number of variants of migraine. These include:
  - 6.1 **Ophthalmoplegic migraine.** Transient diplopia may be associated with classic migraine. The term ophthalmoplegic migraine is applied to recurrent headache associated with paralysis of one or more ocular nerves. The ocular palsy persists for days or weeks after the attack and may be permanent.
  - 6.2 **Hemiplegic migraine and facioplegic migraine.** Hemiplegic migraine describes the situation where the attacks are accompanied by transient hemiparesis or monoparesis, usually of an upper limb. Similarly, recurrent facial palsy in migraine attacks may occur.
  - 6.3 **Retinal migraine.** Ischaemia of the retina and thrombosis of the central retinal artery and its branches may occur and if the retinal ischaemia is recurrent bilateral optic atrophy may result.
  - 6.4 **Symptomatic migraine.** It is often assumed that in patients with a field defect, paresis or aphasia persisting after an attack there must be an underlying vascular anomaly. Investigation of such cases of complicated migraine rarely demonstrate such lesions.

## AETIOLOGY

7. It is generally agreed that the most likely explanation of migraine is that it is due to arterial spasm followed by dilatation within the distribution of the common carotid artery.
8. The basic injurious process of the condition is due to endogenous causes. Those who are predestined to migraine will start to suffer from attacks sooner or later. It is estimated that genetic factors operate in up to 70% of migraine sufferers but there is no simple Mendelian pattern.
9. Clinical attacks occur when the predisposed individual is exposed to an environmental precipitant. In any one individual there may be more than one precipitant. External factors identified as important include exercise (especially football), altered sleep pattern, bright lights, missing a meal, alcohol, specific food intolerance (especially chocolate, cheese and wine), hormonal factors (including menstruation, oral contraceptives and pregnancy), stress and relaxation after stress.
10. None of the environmental factors causes migraine. They merely trigger the symptom complex in a predisposed individual. Attacks of migraine do not worsen the basic underlying disease process.

## CONCLUSION

11. Migraine is a common disorder whose hallmark is paroxysmal headaches. It is due to endogenous causes. Heredity is important. A wide variety of external factors may trigger attacks in predisposed individuals. Such attacks do not worsen the underlying disease process.

## REFERENCES

Walton J. Migraine. In: Brain's Disease of the Nervous System. Oxford. Oxford University Press. 9<sup>th</sup> Ed. 1985. p177-183.

Pearce J M S. Headache. In: (Eds) Weatherall D J, Ledingham J G G and Warrell D A. Oxford Textbook of Medicine. Oxford. Oxford University Press. 2<sup>nd</sup> Ed. 1987. p21.28-21.30.

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