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A BRIEF STUDY ABOUT MIGRAINE: REVIEW ARTICLE

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ABSTRACT

Migraine is a group of perplexing neurological disorders in which the brain and its related tissues are thought to play a vital role during an attack. Migraine was defined previously as a blood vessel problem. With the advancement evidence has led to the conclusion that migraine is a finely coordinated combination of important inputs from both the peripheral and central nervous systems. The trigeminovascular system and the cerebral nervous system are both part of the central nervous system the primary players is cortex. Premonitory, aura, headache, postdrome, and interictal are all phases of migraine, a frequent and debilitating neurological condition. In recent years, our understanding of the pathophysiology of each phase of migraine has improved.

KEYWORDS: migraine, pathophysiology, trigeminovascular pathway, aura, premonitory phase, cortical spreading depression.

INTRODUCTION

Migraine is a prevalent and debilitating neurological condition that impacts over 10% of the worldwide people. Migraine is a persistent primary headache disorder characterised by episodes lasting 4 to 72 hours, according to the International Headache Society. The headache is usually unilateral, pulsing, moderate to severe in severity, increased by regular physical activity, and preceded by nausea, photophobia, and phonophobia.[1] Migraine is one of the top 20 most disabling diseases, according to the World Health Organization.[2] Most migraine attacks begin in the brain, as evidenced by premonitory symptoms (e.g., difficulty speaking and reading, increased emotionality, sensory hypersensitivity) that can occur up to 12 hours before the attack in many patients, as well as the nature of some common migraine triggers like stress, sleep deprivation, oversleeping, hunger, and prolonged sensory stimulation.[2,3] Migraine, depression, and epilepsy are all comorbid ailments, meaning that having one of these diseases raises the likelihood of developing the others, and vice versa. Specific antiepileptic pharmaceuticals are effective in both migraine and depression and the antidepressant medication amitriptyline is commonly used in migraine prevention. Taken together, these findings point to comparable mechanisms underlying migraine, epilepsy, and depression, most likely shared genetic causes.[4] The International Classification of Headache Disorders (ICHD) utilises the term chronic migraine to characterise patients who have regular headaches that are thought to be biologically migrainous. Over the last two decades, the definition of the word 'chronic migraine' has changed as it has gradually supplanted prior terminology such as 'chronic daily headache' and 'transformed migraine.[5] The aura is a fascinatingly complicated and changeable aspect of migraine that has important consequences for pathophysiology, comorbidity, and treatment.[6] The aura is typically a serrated arc of dazzling, sparkling, crenellated shapes that begins close to central vision and spreads peripherally over 5–20 minutes, frequently followed by headache After the same retinotopic progression from centre to peripheral vision areas.[7]

TYPES OF MIGRAINE

The International Classification of Headache Disorders divides migraine into following subtypes[A].

- Migraine without aura
- Migraine with aura
- Childhood periodic syndromes that are commonly precursors of migraine
- Retinal migraine
- Complications of migraine
- Probable migraine

PATHOPJYSIOLOGY

Although the brain processes that initiate a migraine episode are still poorly understood, much more is known about the components that contribute to migraine headache pain pathogenesis. The brain has little sensory innervation, and the capsule structures (meninges), like other viscera, which are the most major pain-producing intracranial tissues.[8] Three separate phases of migraine

can be identified based on clinical features: a starting trigger, an aura, and finally, the headache. Although there is still a lack of knowledge concerning the trigger phase.[9] Migraine is a genetic disorder with a high genetic component. There is a lot of evidence that migraine is a hereditary condition with a family history. Migraine runs in families, and migraineurs with early onset illness or severe disease are more likely to have first-degree relatives who are affected.[10] Specific pathogenic genes have been identified for several unusual kinds of migraine, such as familial hemiplegic migraine (FHM). Cav2.1 calcium channels are expressed presynaptically throughout the brain and in the peripheral nervous system at the neuromuscular junction, and mutations in FHM1 impair their function.[11] FHM1 mutant channels open at lower negative voltages and have a higher chance of opening than normal channels. Greater Ca2+ influx occurs from this "gain-of-function" effect, implying increased neurotransmission.[12] The Na+,K+ pumps, which are largely expressed in neurons and glial cells, are affected by FHM2 mutations in the ATP1A2 gene. 45 Astrocytic Na+,K+ pumps are required for neurotransmitter and potassium removal from the synaptic cleft. FHM2 mutations cause "loss-of-function," resulting in decreased ion and neurotransmitter absorption from the synaptic cleft and an increased risk of cortical spreading depression (CSD).[13]

The trigeminovascular pathway is activated by nociceptive neurons that innervate the dura mater, which release vasoactive neuropeptides like calcitonin gene-related peptide (CGRP) and pituitary adenylate cyclase-activating polypeptide-38, causing signalling along the trigeminovascular pathway the extent to which arterial vasodilation, mast cell degranulation, and plasma extravasation.[15]

DIAGNOSES

To begin, a headache history is the most important tool in diagnosing primary headache disorders, including migraine, while neuroimaging is only required if history or physical examination are suggestive of secondary headache.[16] The International Headache Society criteria are quite useful in migraine diagnosis.[17]

FOLLOWING INFORMATION TO BE COLLECTED FROM PATIENT.

History of headache	***Medical history***	***Physical examination***
Beginning age	*Any other disease*	*Blood pressure, heartrate, BMI*
Severity and position of pain	*Any other condition*	
Signs and symptoms earlier, during and between headache	*Pregnancy, breastfeeding, menopause*	
Aura symptoms		
Time span of attacks		
Migraine triggers		
Prevalence of attacks		
Risk factors		
Follow up		

TREATMENT OF MIGRAINE

There are three types of migraine treatment: pharmacological, non-pharmacological, and neurostimulation.[18]

- **PHARMACOLOGICAL TREATMENT**

Non-prescription non-steroidal anti-inflammatory medicines (NSAIDs) such as aspirin, acetaminophen products, and caffeine-containing combination analgesics are utilised as first-line pharmacological treatment for mild to moderate migraines when used alone**.** These are considered as first line therapies because they have low cost, effective and easily available.[19] Triptans are a class of medicines that work by binding to serotonin receptors in the brain and are 5-HT1B/1D receptor agonists with some affinity for the 5-HT1F.[20] Another type of drug examined in Lasmiditan is ditas which are 5-HT1F agonists.[21] NSAIDs and intravenous (IV) antiemetics are effective treatments in emergency rooms, and they can be given with or without IV dihydroergotamine. These medications help to relieve immediate pain, but they aren't often used to treat long-term chronic migraines. Because of the risks associated with opiates, they are not recommended for the treatment of migraines.[22]

- **NON-PHARMACOLOGICAL TREATMENT**

Nonpharmacological treatments such as cognitive behavioural therapy (CBT) and relaxation training have been linked to the prevention and improvement of migraine symptoms. For chronic migraines, these remedies should be used in conjunction with pharmaceutical therapy.[23] CBT's conventional focus is on stress management, whereas behavioural therapies, mindfulness, and meditation are used to address comorbidities.[24] Biofeedback is also a self-regulatory behavioural strategy for migraine treatment that aims to assist patients gain voluntary control over specific physiological functions.[25]

PREVENTIVE THERAPY

Preventive therapy is indicated for patients who experience attacks that are refractory to acute-attack medicines and cause significant disability. Preventive therapy should be investigated if attacks occur three to four times per month; if the patient has five or more

attacks per month, preventive therapy should be seriously examined.[26]

Drug	Dose
b-Adrenergic–receptor antagonists	
Propranolol	40-120mg Bd
Metoprolol	100-200mg
Amitriptyline	25-75mg
Valporate	400-600mg
Widely used	
Verapamil	160-320mg
Selective serotonin-reuptake inhibitors	

CONCLUSION

This review has compiled the most up-to-date information on migraines and how to treat them. Migraine is a hereditary neurological condition marked by heightened reactivity of cortical and subcortical networks that magnify the intensity of sensory stimuli. Migraine is a condition that affects about 16 percent of the population in the United States and about 1–2 percent of the world's population. Migraine is a sickness that affects many people all over the world and makes it difficult for them to go about their regular lives. Migraine episodes are progressive, with a premonitory, headache pain, and postdromal phase, as well as reversible visual, sensory, and linguistic symptoms in around one-third of cases (aura phase). Currently, it is treated with a range of medicines, and patients are counselled on a regular basis.

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