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A REVIEW ON DIVERGENT AND MORBID OF MIGRAINE

***Ms. Gayatri Mahadeo Hajare**

Maharashtra Institute of Pharmacy (B.Pharm), Betala, Bramhapuri.

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***Corresponding Author**

Ms. Gayatri Mahadeo Hajare

Maharashtra Institute of Pharmacy (B.Pharm), Betala, Bramhapuri.

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ABSTRACT

Migraine is a prevalent, chronic neurological disorder characterized by recurrent episodes of moderate to severe headache, often associated with nausea, vomiting, photophobia, and phonophobia. It is a multifactorial condition influenced by genetic, environmental, and hormonal factors, and can present with or without aura. The global burden of migraine is significant, affecting approximately 15–18% of the population and contributing substantially to disability and reduced quality of life. The pathophysiology of migraine involves complex neurovascular mechanisms, particularly the activation of the trigeminovascular system, leading to the release of neuropeptides such as calcitonin gene-related peptide (CGRP), substance P, and neurokinin A. Migraine progression typically occurs in four phases: premonitory, aura, headache, and postdromal phases, each associated with distinct clinical features. Various triggers, including stress, dietary factors, hormonal fluctuations, sleep disturbances, and environmental stimuli, play a crucial role in initiating migraine attacks. Diagnosis is primarily clinical and based on criteria established by the International Headache Society. Management strategies aim to reduce the frequency, severity, and duration of migraine attacks and improve patient quality of life. Current treatment approaches include pharmacological therapies such as nonsteroidal anti-inflammatory drugs (NSAIDs), triptans, anti-emetics, and CGRP antagonists. In addition, alternative approaches including Ayurveda, homeopathy, yoga, and lifestyle modifications have been explored for their supportive role in migraine management. In conclusion, migraine remains an underdiagnosed and undertreated condition despite its high prevalence and impact. A comprehensive understanding of its pathophysiology and triggers, along with an integrated therapeutic approach, is essential for effective management and improved patient outcomes.

1. INTRODUCTION

Migraine is the most common multidisciplinary and multifactorial neurologic disorder that is characterized by recurrent attacks of headache. Migraine occurs either in episodic or in chronic form, with or without aura. It is a neurological disorder that was regarded as a "hypoglycaemic headache" in the early years of the twentieth century. A plethora of transient motor and somatosensory disturbances are common in this condition.[1]

Migraine is defined as severe throbbing plus unilateral headache related to nausea, photophobia, phonophobia and vomiting.[2] The term migraine is of Greek origin and comes from the word hemicrania's, which means "half of the head". This is a striking feature of the disease, as most individuals feel pain in one half of the head. However, bilateral pain is also common and occurs at the front and back of the head. The nature of the pain is throbbing, which worsens with exertion or movement. Migraine attacks are mainly moderate or severe. Research addressing the underlying mechanism of migraine has reported that activation of the trigeminovascular system is the main factor. Both genetic and environmental factors play a significant role in the development of migraine. It has been postulated that the pathogenesis of migraine is associated with energy deficit syndrome.

The worldwide prevalence of migraine affecting both males and females is estimated to be 15-18%. Migraine is disabling condition that is ranked eighth in the world and fourth in women in terms of burden. According to a recent report, approximately 44.5 million adults in the USA, comprising 18% of women and 6% of men, have experienced migraine.[3]

The economic burden imposed by migraine on society is substantial. In the USA, the estimated direct costs related to migraine accounts to more than US 17 billion. Nearly half of the affected cases have 50% reduced productivity or ability to work during attacks. These individuals are usually absent from school or work once every 3 months, on average.

Migraine is classified into two forms: - migraine with aura (MA) and migraine without aura (MO). Migraine can also be classified into chronic and episodic migraine. Hemiplegic migraine is another type of MA and is a severe and rare condition that affects one side of the body and causes temporary numbness.[4]

Episodic migraine is defined whether the headache days per month are less than 15, and Chronic migraine, whether headache days are equal or more than 15 for a period of more than

three months. Moreover, migraine appears to occur more in women than in men. In addition, it is estimated that about 30% of patients experience an aura that consists in a short period of visual, sensory, or motor disturbances.

The genesis of pain occurs by activation of the trigeminovascular system (TGVS). This system is composed of the cranial vasculature, the trigeminal nerve and the trigeminal nucleus caudalis (TNC). TGVS plays an important role as a major control centre in regulating the cerebral blood flow and it is believed as a key conduit for pain transmission. The activation of trigeminal sensory nerve endings induces the release of vasoactive agents, such as calcitonin gene-related peptide (CGRP), substance P and neurokinin A, resulting in vasodilation and dural plasma extravasation, leading to neurogenic inflammation.[5]

Fig. 1: Migraine Headache.

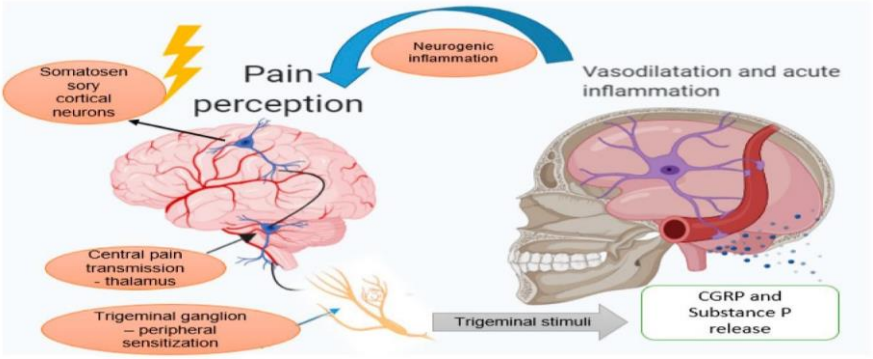

Fig. 2: Pain perception in Migraine.

2. SYMPTOMS

- Some of the common symptoms of Migraine are as follows.

2.1.Headache

Headache, also known as cephalalgia, is the symptom of pain in the face, head, or neck. It can occur as a migraine, tension-type headache, or cluster headache. There is an increased risk of depression in those with severe headaches.

2.2.Nausea

Nausea is a diffuse sensation of unease and discomfort, sometimes perceived as an urge to vomit. While not painful, it can be a debilitating symptom if prolonged and has been described as placing discomfort on the chest, abdomen, or back of the throat.[6]

Fig. 3: Common symptoms of Migraine.

2.3. Sensitivity to light

Photophobia is a medical symptom of abnormal intolerance to visual perception of light. As a medical symptom photophobia is not a morbid fear or phobia, but an experience of discomfort or pain to the eyes due to light exposure or by presence of actual physical sensitivity of the eyes, though the term is sometimes additionally applied to abnormal or irrational fear of light such as heliophobia.

2.4. Sound

Hyperacusis is the increased sensitivity to sound and a low tolerance for environmental noise. Definitions of hyperacusis can vary significantly, it can refer to normal noises being perceived as: loud, annoying, painful, fear-inducing, or a combination of those, and is often categorized into four subtypes: loudness, pain, annoyance, and fear.[6]

2.5. Smell

Osmophobia or Olfactophobia refers to a fear, aversion, or psychological hypersensitivity to odours. The phobia generally occurs in chronic migraine sufferers who may have odours, but the hypersensitivity may extend to all odours. One study found as many as 25% of migraine sufferers had some degree of osmophobia. The condition may also be present in individuals in substance withdrawal, specifically opoid withdrawal syndrome, where it is usually associated with nausea and/or vomiting.[6]

3. CAUSES OF MIGRAINE

3.1. Drinks

These include alcohol, especially wine, and too much caffeine, such as coffee.

3.2. Stress

Stress at home or work can cause migraine. Stress includes feeling overwhelmed at home or work. But you can also become stressed by exercising too much or not getting enough sleep. Biological changes and hormonal fluctuations during stress can make migraine patients more sensitive to other triggers.

3.3. Sensory Stimuli

Bright or flashing lights can induce migraines, as can loud sounds. Strong smells – such as perfume, paint thinner, second-hand smoke, and others – trigger migraines in some people.[7]

3.4. Sleep changes

Missing sleep or getting too much sleep can trigger migraines in some people. Adults need to get 7-8 hours of sleep each night and children and teens at least 9 hours. Migraine patients who get 6 hours of sleep or less have more frequent headaches. If there are numerous changes in sleep hours and bedtime routine, sleeping in on the weekend or even taking an afternoon nap can trigger a headache in migraine sufferers.[7]

3.5.Hormonal changes in women

Women may experience migraines related to their menstrual cycles, to menopause, or to using hormonal birth control or hormone replacement therapy. Some women have migraine due to hormonal changes (mainly estrogen) during their monthly menstrual cycle. Fluctuations in estrogen, such as before or during menstrual periods, pregnancy and menopause, seem to trigger headaches in many women. Hormonal medications, such as contraceptives, also can

worsen migraines. Some women, however, find that their migraines occur less often when taking these medications.[7]

3.6.Foods

Certain food may cause migraine. Dehydration and dieting or skipping meals trigger migraines. Aged cheeses and salty and processed foods might trigger migraines. So might skipping meals. Some foods and drinks (alcohol, caffeine, vitamin waters, energy drinks) can trigger a migraine attack. Many food additives, preservatives and colour can also trigger migraines. There is monosodium glutamate (MSG) in Chinese food, seasonings, sauces, parmesan cheese and meat tenderizers, sodium nitrite (sausage, hot dogs, deli meat, canned and packaged meat, smoked and dehydrated food products); FD&C yellow (tartrazine) in soft drinks, cosmetics, many medicines and ice cream.[7]

3.7.Weather changes

A change of weather or baroreceptor pressure can prompt a migraine. Any changes in temperature, barometric pressure and humidity can trigger migraine. Food additives: These include the sweetener aspartame and the preservative monosodium glutamate (MSG), found in many foods.

3.8.Senses

Loud sounds, bright lights (such as paint fumes or some perfumes). Sight (sensitivity to bright or flickering lights, certain colours, pattern glare, fluorescent lights), sound (loud noises), smell (certain chemicals in fumes, perfumes, smoke, pollution, automobile exhaust and odours) can cause migraine.[7]

Fig. 4: Migraine Triggers.

4. TYPES OF MIGRAINE

The class cation of migraine is closely related to the symptoms they cause. There are symptoms specific to each type and are usually known by the body part they affect. The two most common type of migraine are migraine with aura [classic migraine], and migraine

without aura [common migraine].[8]

4.1. Migraine with aura [classic migraine]

Aura is a visual disturbance which occurs in approximately 20% of migraine attacks. It is known as a neurological disturbance that presents with random flashes of light. Hallucination, blind spots, zigzag figures of light, and photophobia, which means sensitivity to light. Generally, it starts 5-30 min before the actual one set of the migraine headache. It makes the person see a glow around objects or at the margin of the vision area. In addition, it is reported that patients have blurred vision, and difficulty focusing.

4.2. Migraine without aura [common migraine]

As the name suggests, migraine without aura, presents itself with symptoms similar to that of migraine with aura, such as vomiting, nausea, sensitivity to light and noise, but does not present with aura .it often occurs unilaterally or bilaterally.[8]

Fig. 5: Migraine without Aura.

4.3. Hemiplegic migraine

There is generally a family history of hemiplegic migraine in patient of these type of migraine. These is a rare but extreme type of migraine with a presentation aura. Hemipelagic migraine usually starts with short-term numbness on one side of the body. These symptoms are followed by the headache itself when the headache presents itself, the early neurological symptoms may recede.[9]

Fig. 6: Hemiplegic Migraine.

4.4 Ophthalmoplegic migraine

An ophthalmoplegic migraine causes pain around the eyeballs and the paralysis of the optical muscles and also results in the dropping of eyelids. This type of migraine, although very rare, can last from a few days to a few months in some bad cases.

4.5. Basilar artery migraine [Bickerstaff's syndrome]

It is are prevalent amongst young adult women. In this type of migraine, the circulation of blood at the back of the brain or neck is affected. Other associated symptoms include dizziness, loss of balance, disorientation, slurred speech, vertigo, double vision, vomiting, nausea such migraines may last for a few hours to a few days.[9]

Fig. 7: Basilar artery migraine.

4.6. Abdominal migraine

This type of migraine is generally observed in children who have a family history of migraine. As the pain is felt in the abdomen the recognition of migraine is difficult. This pain is not caused by gastrointestinal problems.[9]

Fig. 8: Abdominal migraine.

5. PATHOPHYSIOLOGY OF MIGRAINE

Headache has been known for almost 600 years. The modern concept of chronic migraine was known at the beginning of the 17th century. In the early days, the pathophysiology of migraine was principally based on neurological or vascular mechanisms.

Migraine is divided into four phases including (Premonitory, Aura, Headache and Postdromal). These phases can occur sequentially or may show significant overlap. Meningeal vasodilation together with inflammation is caused by activation of vascular networks, resulting in headache[10,11] et.al.

The pathophysiology of migraine involves modulating pain originating in disrupted neural networks in the head. Studies have shown that brain stem and diencephalic nuclei control the trigeminovascular system, which comprises efferent neurons supplying vascular networks and afferent neurons that feed information to the trigeminal nucleus caudalis.

Head pain is perceived as meningeal inflammation and vasodilation due to activation of these networks. Neurotransmitters, such as serotonin, also play critical roles in the pathophysiology and the treatment of migraine. Serotonin initiates an intracellular network cascade that causes inhibitory or excitatory neurotransmission. Receptors of serotonin are dispersed in the brain, including those used in pain-signalling circuits and cranial blood vessels. Therapies for treating migraine have been tailored to modulate serotonin receptors. The modulation is directed toward amplifying the serotonin signal, leading to pain relief via vasoconstriction of blood vessels and inhibition of peptides, for example substance P[21]and calcitonin gene-related peptide (CGRP)[12-13] et.al.

The International Classification of Headache Disorders (ICHD) is a standardized tool that helps in the identification of primary and secondary headache. The diagnosis of headache disorders is based mainly on the clinical manifestations of the different phases. The aura and headache phases are the main focus, as these mainly require medical attention.[14]

5.1. Premonitory phase

This phase starts before the typical migraine headache. The symptoms precede the headache phase by about 72 h. The symptoms include irritability, food cravings, mood swings, fatigue, stiff neck, and phonophobia. These symptoms persist during the aura and even during the headache phase, and indicate the association between the premonitory phase and the hypothalamic origin. Imaging studies have reported increased blood flow in the hypothalamic region of the brain, indicating the role of the hypothalamus in the early stages of a migraine attack. Hunger, bright light, or sleep deprivation can trigger migraine in migraineurs, or they can indicate premonitory symptoms.[15]

5.2. Aura phase

This phase is seen in one-third of migraine patients. Depolarization of the cortex and creation of a transient wave are the main pathological mechanisms associated with the aura phase, which is also known as cortical spreading depression (CSD). The retinotopic propagation in the visual cortex implies a potential role of CSD in migraine. This is the main characteristic feature of the aura phase, as supported by imaging studies.[16]

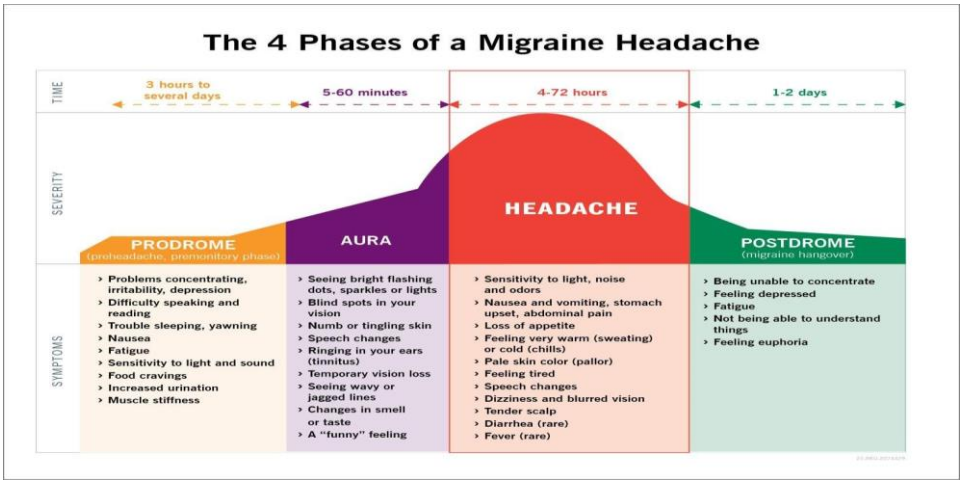

Fig. 9: Phases of Pathophysiology of Migraine.

5.3. Headache phase

The headache phase is marked by unilateral, pulsating, pain of moderate to extreme severity.

his pain can be explained by the neurovascular theory where the activation of trigemino-vascular system is initiated by the earlier activity of higher intracranial centers such as the hypothalamus and thalamus. Consequently, nociceptive fibre's, innervating the vascular supply of the dura matter and originating from the trigeminal ganglion, are sensitized and release inflammatory mediators such as calcitonin gene-related peptide (CGRP) substance P, and Vaso-inhibitory peptide (VIP). These mediators initiate signals along the trigemino-vascular pathway.[17]

5.4. Thalamo-cortical circuits, thalamic circuits and migraine

Changes in brain functionality have been reported during the premonitory phase. Electrophysiological studies have reported increased blood flow, particularly in circuits connecting the thalamus-cortex. Differences in thalamic and thalamocortical activity have also been reported by functional and structural imaging studies. Changes in brain functionality in migraine patients have also been reported in electrophysiological studies.[18]

5.5. Postdromal phase

This phase is the least studied in the literature. It is mostly ignored and unreported by the patients. However, it is sometimes a distinct phase of the disease or a continuation of the same pathology. Patients might report symptoms like tiredness, muscle weakness, mood changes, difficulty in concentration and reduced appetite. A possible explanation of the postdromal phase might be the persistent activation of the brainstem and diencephalic while and after processing the pain stimuli.[19]

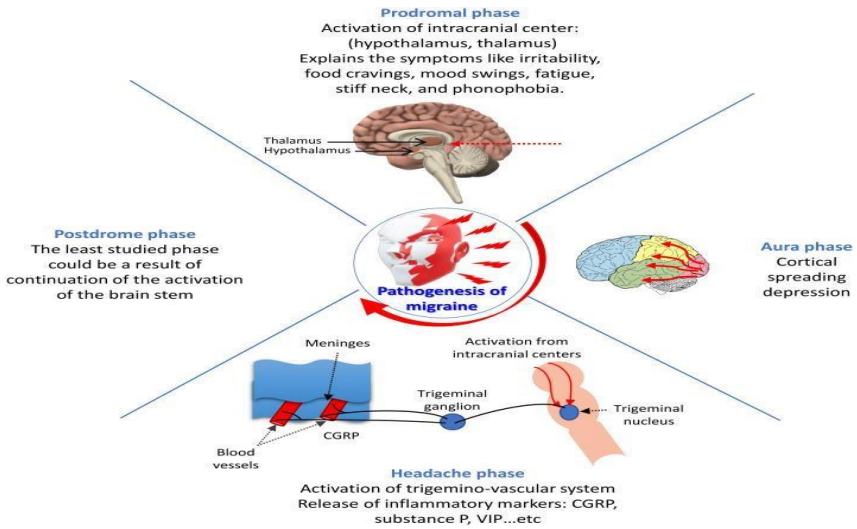

Fig. 10: The mechanisms of the different phases of migraine. An overlap of the different phases is possible.[1]

6. DIAGNOSIS OF MIGRAINE

The guidelines for diagnosing migraine were formulated by the International Headache Society.

There is a difference in the diagnostic criteria for MA (Migraine with aura) and MO (Migraine without aura). The features of MA involve a minimum of five headaches in 4–72 h. Also, MA demonstrates either pulsation, unilateral location, intense pain, or exacerbation of headache with routine activities. These features are accompanied by vomiting, nausea and phonophobia or photophobia. Notably, the symptoms and headache should not be attributed to other diseases.[5]

The diagnostic features for migraine with the typical aura include a minimum of five headaches, among which at least two episodes must be accompanied by an aura. The headache should begin or occur within 60 min of the aura. The aura must consist of reversible dysphasic speech or unilateral sensory or homonymous visual symptoms. There must be at least one symptom that gradually increases with time, with each symptom ranging from 5 to 60 min.[5]

Table No. 1: The diagnostic criteria for migraine headache as formulated by the International Headache Society.[5]

Without Aura	With Aura
Minimum of five headaches within 4-72 hrs. Pulsation. Unilateral location Intense pain Exacerbation of headache with routine activities. These features are accompanied by vomiting or nausea and phonophobia or photophobia.	Minimum of five headaches, among which at least two episodes must be accompanied by an aura. The headache should begin with, or be within 60 min of, the aura. The aura must consist of reversible dysphasia speech. Unilateral sensory. Homonymous visual symptoms.

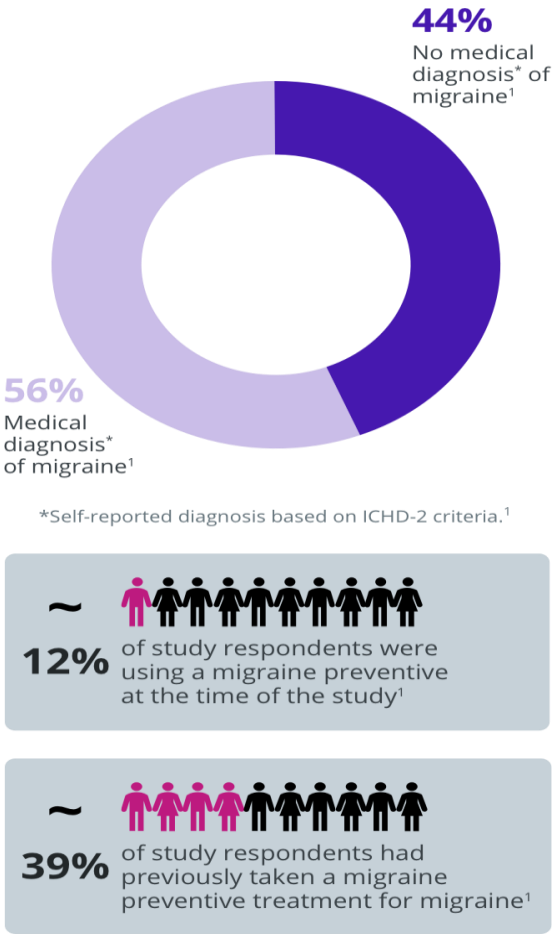

Fig. 11: Diagnosis based on ICHD-2 Criteria.

7. TREATMENT OF MIGRAINE

The main goal of treatment for migraine is to reduce the severity and duration of the migraine attack. Other objectives include restoring functioning ability, reducing the use of rescue medications and promoting overall management with no or minimal side effects. The current therapies for migraine include acetaminophen, triptans (sumatriptan, rizatriptan, almotriptan, frovatriptan, naratriptan and zolmitriptan). Nonsteroidal Anti-Inflammatory Drugs (NSAIDs) (naproxen sodium, acetylsalicylic acid (ASA), ibuprofen, and diclofenac potassium, dihydroergotamine, non-opioid analgesics (ASA, acetaminophen, and caffeine), NSAID-triptan combinations, and anti-emetics (chlorpromazine, metoclopramide and prochlorperazine).[20-22]

Drugs such as acetaminophen, butorphanol and tramadol show some efficacy; however, the disadvantages of NSAIDs surpass their benefits and hence they are less recommended for use. Opioids should be avoided due to their addiction risk. Opioids can reduce the efficacy of triptans and promote sensitization to medications. Chronic migraine patients always require

prophylactic treatment, while migraine patients with low frequency of symptoms can be managed with effective acute therapy.[22]

On a botulinumtoxin A has been approved for treating chronic migraine in European Union, since it was in use countries like Italy. CGRP receptor antagonists was reported for decreasing migraine frequency. The most important risk due to the overuse of symptomatic medication should be considered during migraine progression. A key component of migraine therapy involves over-the-counter medications, which are considered the first-line therapy by most people suffering from migraine. Medications such as naproxen, ibuprofen, acetaminophen and aspirin form the first line of treatment for a migraine attack. These medications have fewer side effects and a favourable administration route, in addition to low cost and high efficacy.[23]

7.1. ALLOPATHIC TREATMENT

7.1.1 Triptans

In acute attacks of moderate to severe Migraine (with or without aura), Triptans are the most efficient "First-line therapy", especially in patients whose pain is not relieved by analgesics/NSAIDS.

Triptans are selective Serotonin-agonists. Triptan binding to the Serotonin receptors (5-HT1B) in intracranial vessels (which are dilated during a migraine attack), produces vasoconstriction.[24]

Triptan binding to the neurogenic and central Serotonin receptors (5-HT1D) inhibits Substance P and CGRP release and blocks pain signals to the brain by inhibiting nociceptors. In this way, Triptans can potentially reverse various steps of trigeminovascular activation in Migraine.[24]

As compared to Ergot alkaloids which are non-selective Serotonin agonists, Triptans have more favourable risk profile. There are seven Triptans commercially available. Although the pharmacology of the triptans is very similar, individual patient responses can vary remarkably to different triptans because of the role of genetic factors in Migraine.

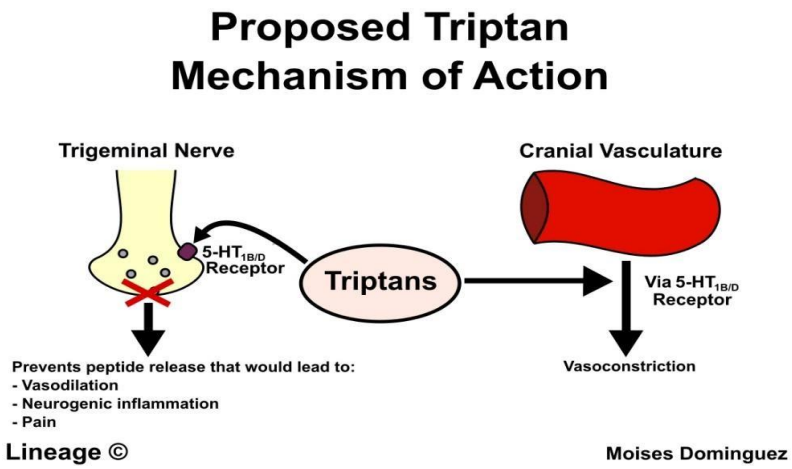

Fig. 12: Mechanism of action of Triptans.

7.1.1.1. Sumatriptan

Sumatriptan is the first triptan introduced in 1991. It is the most extensively studied triptan and considered as the "Gold standard" till today. It is available in a variety of dosage forms (tablet, nasal spray, and subcutaneous injection). The subcutaneous formulation has the fastest onset of action; therefore, it is preferred in patients with severe headache. Also, in patients who cannot tolerate oral tablets due to excessive nausea/vomiting, subcutaneous preparation is the drug of choice.

Because of its selectivity for cranial vasculature, it may produce minimal cardiovascular adverse effects. Due to its low oral bioavailability and short half-life, “second-generation” triptans (frovatriptan, zolmitriptan, eletriptan, almotriptan, naratriptan, rizatriptan) having a better pharmacokinetic profile have been formulated.[25]

Table No. 2: Dosage of Sumatriptan.

Drug	Formulation	Strengths
Sumatriptan	Nasal spray	5.2.mg
	Nasal Powder	11 ml/nosepiece
	Oral tablet	25,50,100, mg
	SC injection	3,4,6 mg/0.5 mL

Fig. 13: Sumatriptan tablets.

7.1.2. Ergotamine

Ergotamine were the first class of drugs used specifically for migraines. These drugs help in causing blood vessels around the brain to contract and relieve migraine within few minutes. Ergotamines are available in tablets that dissolve under tongue, pills, suppositories, nasal spray and injections. Ergotamines are generally taken at a first sing of headache symptoms, and some have the option to take additional doses every 30 minutes if the headache continues.[8]

7.2. AYURVEDIC TREATMENT

There are a number of ways that Ayurveda can bring balance to life and treat the root cause of headache and migraine in particular. In addition to specific treatment option for each type of headache, we can use other modalities to treat migraine headaches.

- **Vata-type headache treatment:**

According to Vaghabhata's Astanga Hrdayam, for treatment of vata-type headache, ghee should be applied to the head and should also be ingested with warm water. Warm milk boiled with dashmula and other drugs that reduce vata dosha can be poured on the head. Herbs of varanadi gana are boiled in milk and water (50:50) until only milk remains. The butter of this mixture is taken out by churning and herbs of sweet taste are cooked in this butter (ghee). This ghee can be used as nasya. This ghee can also be taken by adding it to milk and some sugar. The herbs (karpasamajja, tvak, musta and buds sumana) can be steeped in hot water and instilled in the nose.[26]

- **Pitta-type headache treatment**

Pitta sirasula chikitsa (pitta-type headache treatment): In pitta-induced headaches, oleation and then venesection can be done. The patient can apply cold compress to the face and wash the head (not with hot water). Purifying enema therapy can be beneficial. Medicated milk or ghee with jivaniya gana herbs are helpful either internally or nasally.[26]

- **Kapha-type headache treatment**

Kaphaja sirasula chikitsa (Kapha-type headache treatment): According to Vagbhata's Ashtanga Hrdayam, oleation therapy can be done by drinking old ghee and then inducing vomiting. Nasal application of herbs with dry, hot and penetrating qualities and avoiding food (fasting) are beneficial. Fomentation (sweating) and external application of herbal pastes are also recommended.[26]

7.2.1. Diet modification

- **Dosha specific nutrition**

Depending on the dosha[s] involving in headache and also the patient constitution, the patient must be instructed to eat a diet that pacifies the aggravated dosha.

- **Avoiding or minimizing migraine trigger foods**

Migraine trigger foods can be different in every individual and finding these trigger foods can help to minimize migraine headaches. This confirms the Ayurvedic perspective of dosha specific diet for each individual.[8]

7.2.2. Lifestyle modification

Migraine and other type headaches can be prevented by schedule, eating routing and working habits and avoiding migraine triggers can reduce the frequency and severity of migraine headache.

Fig. 14: Ayurvedic treatment.

7.2.3. Meditation

Stress is a major contributor to both tension and migraine headaches'-OM Breath meditation can reduce stress. In this meditation, sit in comfortable position. There are many types of types of meditations are avail.[8]

7.2.4. Pranayama (Breathing exercise)

Different types of breathing exercises have different effects of the body, mind and spirit. Breathing practices purify nadis. If the headache is due to vata vitiation, one can benefit from alternate nostril breathing. If the headache is due to pitta vitiation, doing lunar and shitali pranayama can help and if due to kapha dosha vitiation, solar and kapalabhati pranayama can be beneficial.

Fig. 15: Pranayama for Migraine.

7.2.5. Soothing nose drops

Putting about 5 drops of Brahmi ghee in each nostril can alleviate the pain of migraine headache. In vata -type headaches, placing 3-5 drops of warm ghee in each nostril can help to calm down the headache.[8]

7.2.6. Herbal pastes for headaches

A mixture of herbal essential oils can help to alleviate headache Peppermint essential oil in massage oils and balm can with help with migraine. Combine the following essential oils in a base oil (almond, coconut or sesame oil) for quick relief of headache:5 parts eucalyptus oil, 1 part menthol crystals, 1part camphor. Dr Lad in the book The Complete Book of Ayurvedic Remedies, suggests making a paste by adding 1/4 teaspoon nutmeg powder to some water and applying it to the forehead and leaving it for 30 minutes. For pittatype headache a cooling paste can be made by mixing sandalwood powder with water. One can apply this paste to forehead and temples and leave it on the skin for half to one hour before rinsing.[8]

7.2.7. Aromatherapy

Essential oils can enter the body through the skin, nasal passages, bronchioles, lungs and gastrointestinal tract.

7.3. HOMEOPATHIC TREATMENT

There are lots of drugs which helps for the treatment of migraine. The selection of similimum depends on the totality which is varies one person to another so we must stress on the individuality. The well-known drug is basically used on totality but rare medicine used when totality is not properly formulated. Paucity of symptoms in these remedies compels to use

them on the basis of few indicated symptoms. This information is the base for uses this medicine. One should add some other symptoms which will be more useful to gather more information about this medicine.[27]

7.3.1. Belladonna

Headache in plethoric and healthy individuals. The motive is some disturbance of the circulatory gadget. The headache is violent, pounding, and throbbing. Throbbing and bursting headache in temples with fiery crimson and warm face. Eyes bloodshot and crimson. Face flushed. The pains disappear as all of a sudden as they appear. Solar headache with full bounding pulse.[27]

Fig. 16: Belladonna plant.

7.3.2. Hypericum

Headache due to indulging in alcohol and different stimulants. It has aching pain as though overwhelmed; tensive; heavy; sinking, and bruised with associated symptoms of nausea and vomiting of sour and nasty remember. Ineffectual urging for stool. Headaches are higher in the night time.[27]

7.3.3. Chamomilla

Headache with peevishness. The affected person is cross and indignant. This headache happens generally in sufferers who use alcohol in excess and take opium to counteract its effect or who take sturdy espresso to quieten the nerves.

7.3.4. Cocculus

Migraine with vertigo and nausea, occipital pain is characteristic. Sick headache from riding in a carriage, boat train or cars. headache at each menstrual period with nausea and inclination to vomit. Headache from loss of sleep.[27]

Fig. 17: Homeopathic treatment.

7.3.5. Gelsemium

Pains starting within the nape of the neck and shift over to move, inflicting a bursting sensation inside the brow and eyeballs. Feels as though the head is full and huge. Unconsciousness. As though there is a band the pinnacle.

7.3.6. Glonine

Headache because of running below gaslight, in the solar while warmness falls on the top. Head feels tremendously big, Sunstroke, and sun headache without unconsciousness.[27]

7.4. YOGA

7.4.1. Yoga for headache

Stress is an important factor in creating both migraine and tension headaches, yoga can without doubt help in preventing these types of headaches. Yoga can also help with tension in the muscle of the neck, back and head which contribute to headaches. Yoga helps to release tight muscles and improve blood circulation in that region.[8]

7.4.2. Yoga meditation

There is not one type of meditation which is “yogic meditation”, so here it is meant the several meditation types taught in the yoga tradition. Yoga means “union”. Tradition goes as far as 1700 B.C, and as its highest goal spiritual purification and self-knowledge.[8]

Fig. 18: Yoga meditation.

8. CONCLUSION

Migraine is a multifactorial chronic neurological condition that varies in frequency, severity and its effect on the quality of life. Genetic makeups play a significant role in defining an individual's susceptibility to migraine. In these patients, the pathophysiology stresses the presence of different triggers that initiate a headache attack or increase the frequency of the attacks.

To conclude, while migraine is a prevalent condition, it is often under-diagnosed. Also, it is not perceived adequately by certain patients who suffer from migraine and are eligible for prophylactic care. If provided, these therapies could decrease the physical and functional disabilities of migraine and increase patients' quality of life.

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