Homoeopathic Management of Migraine: A Review of Miasmatic and Therapeutic Perspectives

Dr. Priya Debnath[1], Kariya Khushi[2], Dr. Tanudipta Halder[3], Dr. Anup Kumar Das[4]
[1]*Assistant Professor, Department of Community Medicine, L. R. Shah Homoeopathy College, Rajkot, Gujarat*
[2]*Internee, L. R. Shah Homoeopathy College, Rajkot, Gujarat*
[3]*Assistant Professor, Department of Research Methodology and Biostatistics, L. R. Shah Homoeopathy College, Rajkot, Gujarat*
[4]*Principal, L. R. Shah Homoeopathy College, Rajkot, Gujarat*
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***Abstract*—Migraine is a common and disabling neurovascular disorder characterized by recurrent episodes of moderate to severe headache, often associated with nausea, vomiting, photophobia, and phonophobia. It significantly affects the quality of life and functional capacity of individuals worldwide. Conventional treatment primarily focuses on symptomatic relief and prophylaxis; however, recurrence and adverse effects remain major concerns. Homoeopathy offers a holistic and individualized therapeutic approach based on the totality of symptoms and the dynamic understanding of disease. According to homoeopathic philosophy, migraine is considered a manifestation of underlying chronic miasmatic influences, predominantly psora, sycosis, and syphilis. The miasmatic approach aids in understanding the susceptibility, progression, and chronicity of the disease, thereby guiding constitutional remedy selection. Various homoeopathic remedies such as Sanguinaria canadensis, Natrum muriaticum, Spigelia anthelmia, Bryonia alba, and Glonoinum are therapeutically indicated in migraine management based on symptom similarity and individual constitution. This review highlights the homoeopathic understanding of migraine with emphasis on miasmatic interpretation and therapeutic perspectives. Homoeopathy aims not only to relieve acute symptoms but also to address the underlying predisposition, thereby promoting long-term improvement and overall well-being.**

***Index Terms*—Chronic disease; Homoeopathy; Homoeopathic management; Homoeopathic therapeutics; Individualization; Migraine; Miasm**

I. INTRODUCTION

Headache is one of the most common neurological complaints encountered in clinical practice and constitutes a significant cause of physical suffering, functional disability, and reduced quality of life worldwide. [1] Migraine headache (MH) is a highly prevalent and complex neurovascular disorder affecting more than one billion individuals globally, making it one of the leading causes of years lived with disability (YLD), particularly among adults aged 20–59 years. [5]

Among the various types of headaches, migraine occupies a distinctive position because of its recurrent nature, characteristic symptom complex, and substantial impact on personal, social, and professional functioning.

According to the classification established by the International Headache Society, headaches are broadly categorized into primary and secondary disorders. Migraine is classified as a primary headache disorder, meaning that it is a disease entity in itself and not merely a symptom secondary to another underlying pathology. [2] the pathophysiology of migraine involves activation of the trigeminovascular system and the release of vasoactive neuropeptides, particularly calcitonin gene-related peptide (CGRP), which contributes to neurogenic inflammation and vasodilation. Altered serotonin (5-hydroxytryptamine) levels are also implicated in the modulation of pain pathways. [7]

The term migraine is derived from the Greek word Hemicrania, meaning "half of the head," which reflects the typically unilateral distribution of pain. Migraine is usually an episodic disorder characterized by attacks lasting from 4 to 72 hours, often associated with nausea, vomiting, photophobia, and phonophobia. [2]

Conventional management of migraine in modern medicine primarily focuses on symptomatic relief and prophylaxis. Commonly used medications include beta-blockers, calcium channel blockers, antidepressants, anticonvulsants, and non-steroidal anti-inflammatory drugs. Although these therapeutic measures may provide temporary relief, long-term pharmacological treatment is frequently associated with adverse effects, financial burden, and recurrence of symptoms upon discontinuation. [3]

From the homoeopathic perspective, recurrent and long-standing migraine can be understood within the framework of chronic disease as described by Samuel Hahnemann in the Organon of Medicine. In Aphorisms 72–82, chronic diseases are described as deep-seated disorders arising from underlying dynamic causes. [4]

In Aphorisms 78–81, Hahnemann further explains that true chronic diseases originate from chronic miasmatic influences—namely Psora, Sycosis, and Syphilis—which act as the fundamental cause of persistent and relapsing disease conditions. [4] According to this doctrine, chronic headaches and migraine may represent manifestations of these underlying miasmatic dyscrasias.

Therefore, the present review entitled "Homoeopathic Management of Migraine: A Review of Miasmatic and Therapeutic Perspectives" aims to explore migraine from a homoeopathic and miasmatic perspective and to evaluate the therapeutic role of individualized homoeopathic management. This review seeks to evaluate the scope and therapeutic role of individualized homoeopathic treatment based on the principles of the Organon of Medicine, with special reference to totality of symptoms, constitutional prescribing, and miasmatic analysis. Through this approach, homoeopathy endeavors not only to alleviate the suffering of migraine patients but also to achieve sustained and long-term improvement by addressing the underlying dynamic cause of the disease.

II. PATHOPHYSIOLOGY

The pathophysiology of migraine is complex and involves neurovascular, neurochemical, and hormonal mechanisms. The higher prevalence of migraine in females and the increased frequency of attacks during certain phases of the menstrual cycle strongly suggest hormonal influences, particularly fluctuations in estrogen levels. [6]

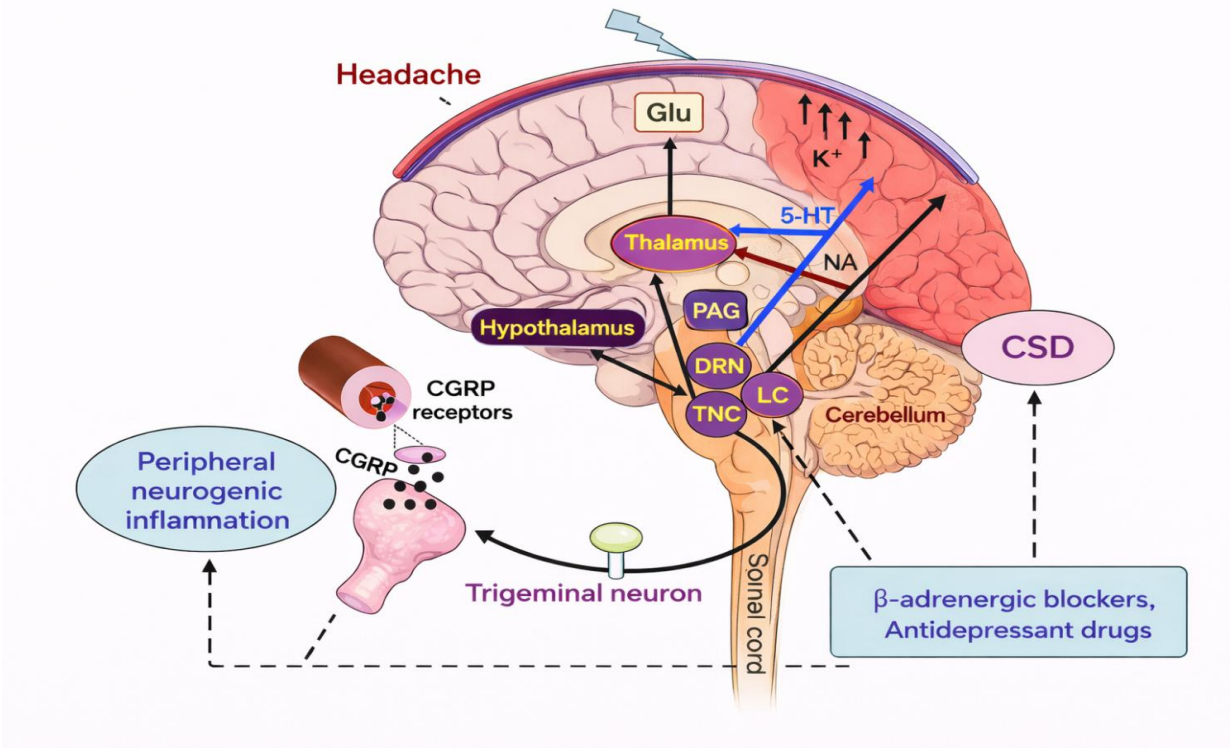
One of the central mechanisms implicated in migraine is activation of the trigeminovascular system. Stimulation of the trigeminal nerve (cranial nerve V) leads to the release of vasoactive neuropeptides, especially calcitonin gene-related peptide (CGRP), from the perivascular nerve endings. [9] CGRP plays a key role in migraine pathogenesis by causing vasodilation of intracranial blood vessels and promoting neurogenic inflammation.

CGRP may directly act on vascular smooth muscle cells to induce vasodilation, or it may stimulate mast cells to release inflammatory mediators such as histamine and cytokines. This results in increased vascular permeability and inflammation within the meninges. The inflammatory process activates nociceptive fibers of the trigeminal nerve, transmitting pain impulses to the trigeminal ganglion, then to the thalamus, and finally to the cerebral cortex, where pain perception occurs. [9]

Another important mechanism involves serotonin (5-hydroxytryptamine). Altered serotonin levels are observed during migraine attacks. A decrease in serotonin may increase vascular permeability and facilitate the release of inflammatory mediators, thereby contributing to activation of pain pathways. [10] Serotonin receptors also play a role in modulating cranial blood vessel tone and nociceptive transmission.

These neurovascular and neurochemical events primarily occur in the meninges, the protective coverings of the brain, where trigeminal nerve endings are richly distributed. The interaction between vascular changes, neurogenic inflammation, and central pain processing ultimately results in the characteristic throbbing headache of migraine.

Thus, migraine is now understood not merely as a vascular disorder but as a complex neurovascular condition involving peripheral and central sensitization mechanisms.



III. TRIGGER OF MIGRAINE

Migraine attacks are often precipitated by specific internal or external factors known as triggers. These triggers do not directly cause migraine but are believed to precipitate attacks in individuals with an underlying neurobiological susceptibility.

A study conducted by Mollaoglu (2012) reported that the most common trigger factors among migraine patients were emotional stress (79%), sleep disturbances (64%), and dietary factors (44%). [8] Emotional stress was identified as the most significant precipitating factor.

Sleep-related disturbances, including sleep deprivation, irregular sleep patterns, and excessive sleep, are frequently associated with the onset of migraine attacks. Stress and sleep disturbances were found to be particularly significant in patients suffering from migraine with aura. In contrast, environmental factors such as bright light, loud noise, weather changes, and strong odors were reported to be more commonly associated with migraine without aura. [8]

Other commonly reported trigger factors include hormonal fluctuations (especially during the menstrual cycle), fasting, dehydration, certain foods (such as chocolate, cheese, and processed foods), alcohol consumption, and sensory overstimulation. [11]

It is important to note that triggers vary among individuals, and multiple triggers may interact to precipitate an attack. Moreover, migraine episodes may sometimes occur in the absence of any identifiable trigger, further supporting the concept of inherent neuronal hypersensitivity in migraine sufferers.

Understanding trigger factors is essential in the comprehensive management of migraine, as identification and avoidance of individualized triggers may reduce the frequency of attacks and improve quality of life.

IV. EPIDEMIOLOGY

Migraine is a highly prevalent neurological disorder affecting approximately 12–15% of the global population. [5] It is more common in women than in men, with an estimated annual prevalence of 12% among females and 6% among males. [6] the gender disparity is largely attributed to hormonal influences, particularly fluctuations in estrogen levels during the reproductive years.

Migraine commonly begins during adolescence, with prevalence rising after puberty. The peak incidence is observed between 35 and 39 years of age, after which it gradually declines, especially following menopause. [6]

Migraine is a significant cause of healthcare utilization. It ranks among the fourth or fifth leading causes of emergency department visits and accounts for approximately 3% of all emergency room consultations annually. [13]

In terms of disability burden, migraine is recognized as one of the leading causes of years lived with disability (YLD) worldwide. According to global burden studies, migraine ranks second only to low back pain as a cause of disability when measured by YLD among neurological and musculoskeletal disorders. [5]

The substantial prevalence, recurrent nature, and associated disability highlight migraine as a major public health concern requiring effective long-term management strategies.

V. CLASSIFICATION

According to the International Headache Society in the International Classification of Headache Disorders, 3rd edition (ICHD-3), migraine is classified into several subtypes based on clinical presentation. [2]

1. Migraine Without Aura (Common Migraine)

Migraine without aura is the most common subtype, accounting for approximately 70–80% of cases. [14] It is characterized by recurrent headache attacks without preceding neurological warning symptoms. The headache is typically unilateral, pulsating, moderate to severe in intensity, aggravated by routine physical activity, and associated with nausea, photophobia, or phonophobia.

2. Migraine With Aura (Classical Migraine)

Migraine with aura occurs in approximately 20–30% of patients. [14] It is characterized by transient focal neurological symptoms (aura) that usually precede the headache phase. The most common aura is visual (e.g., scintillating scotoma, zigzag lines), but sensory, motor, or speech disturbances may also occur.

3. Retinal Migraine

Retinal migraine is characterized by repeated attacks of monocular visual disturbance, such as scintillations, scotoma, or temporary blindness in one eye, lasting less than one hour and followed by headache. [14]

4. Migraine with Brainstem Aura
(Previously termed Vertebrobasilar Migraine)

This subtype involves aura symptoms originating from the brainstem without motor weakness. Symptoms may include vertigo, dysarthria, diplopia, ataxia, tinnitus, or decreased level of consciousness. Occipital headache is common. [14]

5. Hemiplegic Migraine

Hemiplegic migraine is a rare form characterized by migraine attacks associated with reversible motor weakness (hemiparesis) as part of the aura. The weakness may last hours to days but is typically reversible. [14]

6. Vestibular Migraine

Vestibular migraine presents with recurrent episodes of vertigo associated with migraine headache or migrainous features such as photophobia and phonophobia. [14]

7. Menstrual Migraine

Menstrual migraine occurs in close temporal relation to menstruation, typically from two days before to three days after onset of menses. Hormonal fluctuations are considered the primary triggering factor. [14]

8. Migraine in Childhood (Periodic Syndromes Associated with Migraine)

Certain episodic syndromes in childhood are considered precursors of migraine, including:

- Cyclic vomiting syndrome
- Abdominal migraine
- Benign paroxysmal vertigo

These conditions may later evolve into classical migraine. [14]

9. Post-Traumatic Migraine

Migraine-like headache that develops following minor head trauma may be classified under post-traumatic headache with migrainous features. [14]

10. Complicated Migraine

The term "complicated migraine" is not currently preferred in ICHD-3 terminology. Conditions previously described under this heading are now categorized as:

- Migrainous infarction
- Persistent aura without infarction
- Migraine-triggered seizure

These are considered rare complications of migraine. [14]

VI. CLINICAL FEATURES AND DIAGNOSTIC CRITERIA

Clinical Features

Migraine is characterized by recurrent episodes of headache with specific associated symptoms. The common clinical features include:

- Throbbing or pulsating pain, typically unilateral in location
- Moderate to severe intensity
- Aggravation by routine physical activity or head movement
- Photophobia (sensitivity to light)
- Phonophobia (sensitivity to sound)
- Osmophobia (sensitivity to odors)
- Nausea and/or vomiting
- Loss of appetite
- Abdominal discomfort (more common in children)
- Pallor or facial flushing
- Fatigue and malaise
- Dizziness or vertigo
- Blurred vision
- Scalp tenderness

Some patients may experience prodromal symptoms such as mood changes, food cravings, neck stiffness, or fatigue hours to days before the onset of headache. In migraine with aura, transient neurological symptoms such as visual disturbances, sensory symptoms, or speech difficulty precede the headache phase. [14]

Motion sensitivity – headache aggravated by head movement or physical movement.

VII. DIAGNOSTIC CRITERIA (MIGRAINE WITHOUT AURA – ICHD-3)

According to the International Headache Society (ICHD-3), migraine without aura is diagnosed when the following criteria are fulfilled: [14]

A. At least five attacks fulfilling criteria B–D

B. Headache attacks lasting 4–72 hours (untreated or unsuccessfully treated)

C. Headache has at least two of the following characteristics:

- Unilateral location
- Pulsating quality
- Moderate or severe intensity
- Aggravation by or causing avoidance of routine physical activity

D. During headache, at least one of the following:

- Nausea and/or vomiting
- Photophobia and phonophobia

E. Not better accounted for by another diagnosis

For migraine with aura, additional criteria include fully reversible visual, sensory, speech, motor, brainstem, or retinal symptoms lasting 5–60 minutes. [14]

VIII. SCREENING AND INVESTIGATIONS

Migraine is primarily a clinical diagnosis based on detailed history, symptomatology, and neurological examination. According to the International Headache Society guidelines, routine laboratory or imaging investigations are not required in typical cases fulfilling the diagnostic criteria for migraine. [14]

A thorough clinical evaluation includes:

- Detailed headache history (onset, duration, frequency, character, associated symptoms)
- Identification of trigger factors
- Family history of migraine
- Physical and neurological examination

Neuroimaging or additional investigations are recommended only when the clinical presentation is atypical, unusually severe, progressively worsening, or associated with abnormal neurological findings. [15]

These are primarily performed to exclude secondary causes of headache.

Neuroimaging Modalities
1. Magnetic Resonance Imaging (MRI)

Magnetic Resonance Imaging (MRI) utilizes a strong magnetic field and radiofrequency waves to produce high-resolution images of the brain and intracranial structures. MRI is particularly useful in detecting:

- Brain tumors
- Intracranial hemorrhage
- Ischemic stroke
- Demyelinating disorders
- Structural abnormalities

MRI is preferred over CT scan in most cases due to superior soft tissue resolution. [15]
2. Computed Tomography (CT) Scan

Computed Tomography (CT) scan uses X-ray beams to generate cross-sectional images of the brain. It is particularly useful in emergency settings for rapid detection of:

- Acute intracranial hemorrhage
- Skull fractures
- Space-occupying lesions
- Acute traumatic injury

CT scan is often used when sudden severe headache (e.g., suspected subarachnoid hemorrhage) is present. [15]

Thus, investigations in migraine are primarily aimed at ruling out secondary causes of headache rather than confirming migraine itself.

IX. DIFFERENTIAL DIAGNOSIS

- Temporal arteritis (Giant cell arteritis)
- Meningitis
- Acute angle-closure glaucoma
- Subarachnoid haemorrhage

X. COMPLICATIONS OF MIGRAINE

- Status migrainosus
- Migrainous infarction
- Migraine-triggered seizure

XI. HOMOEOPATHIC VIEW

Manifestation of Symptoms in Various Miasms
(As described by H.A. Roberts)

According to the miasmatic theory propounded by Samuel Hahnemann and further elaborated by Herbert A. Roberts, chronic diseases such as migraine arise from underlying miasmatic influences. The symptomatology varies according to the predominating miasm. [15]

1. Psoric Manifestation

Modern clinical observations often associate migraine with emotional disturbances such as grief, anxiety, and prolonged stress. This corresponds with Hahnemann's doctrine that psoric individuals are highly susceptible to ailments arising from emotional excitations.
Psoric headaches are generally:

- More common during the daytime
- Associated with emotional strain
- Ameliorated (>) by rest and lying down
- Ameliorated (>) by warm applications

These headaches are functional in nature and lack destructive tendencies. [15]

2. Syphilitic Manifestation

Syphilitic headaches are typically characterized by:

- Night aggravation (<)
- Occipital location (back of the head)
- Dull, heavy, or lancinating pain
- Deep-seated and persistent character

Modalities commonly observed include:

- Aggravation (<) by motion or exertion
- Aggravation (<) from warmth
- Aggravation (<) during night and sleep
- Amelioration (>) by cold applications
- Sometimes amelioration (>) after epistaxis

These headaches are frequently associated with profound weakness, chilliness, sadness, and prostration.

Roberts also described that headaches occurring during periods of inactivity (e.g., Sundays) may represent a mixed psoric–syphilitic state. [15]

3. Sycotic Manifestation

- Sycotic headaches are often:
- Located at the vertex or frontal region
- Worse (<) at night, especially after midnight
- Worse (<) on lying down
- Associated with restlessness
- Occasionally observed in febrile conditions in children
- Motion may temporarily ameliorate (>) symptoms.

The sycotic type shares certain features with the syphilitic miasm, particularly night aggravation and vertigo originating from the base of the brain. [15]

XII. HOMOEOPATHIC THERAPEUTIC PERSPECTIVES

The homoeopathic management of migraine is based on the principles of individualization, totality of symptoms, and miasmatic analysis. The following remedies are commonly indicated in migraine according to classical homoeopathic literature.[12]

1. Sanguinaria canadensis

Indicated in congestive headaches with marked throbbing pain, as if the eyes would be pressed out. The pain typically begins in the occipital region and extends upward, settling over the right eye.

Key features:

- Right-sided headache
- Periodicity (often every seventh day)
- Begins in the morning, increases during the day, and subsides by evening
- Distended veins and flushed face
- Climacteric headaches

Modalities:

- Aggravation (<) from noise, light, odors, and motion
- Amelioration (>) by sleep, pressure, lying in a dark quiet room, Relief after vomiting or passing flatus

There may be associated nausea with salivation and burning vomiting. A characteristic description is pain in the occiput "like a flash of lightning." [12]

2. Spigelia anthelmia

Especially suited for left-sided neuralgic migraine. The pain begins in the occiput and radiates to the vertex and frontal region, finally localizing above the left eye.

Key features:

- Violent, throbbing, semi-lateral headache
- Marked involvement of the left eye
- Redness and lachrymation of the affected eye
- Sensation as if the eyes were too large

Modalities:

- Aggravation (<) from noise, motion, touch, and jarring
- Pain increases at sunrise, reaches its peak at noon, and declines by sunset

The pain may be described as pulsating, pressing, or as if a band were around the head. [12]

3. Bryonia alba

Indicated in splitting or bursting headaches, often frontal or over one eye (frequently left-sided).

Key features:

- Severe headache as if the head would burst
- Sensation as if struck by a hammer
- Associated nausea and vomiting
- Dry mouth with intense thirst

Modalities:

- Aggravation (<) from the slightest motion, even movement of the eyes
- Amelioration (>) from absolute rest and pressure, Patient prefers to lie still and avoid conversation, Vertigo and faintness on rising may be present. [12]

4. Glonoinum

Indicated in congestive and sun-induced headaches. The patient feels as if the head would burst or enlarge.

Key features:

- Intense throbbing headache
- Sensation of fullness and heat in the head
- Protruding eyes
- Headache from prolonged exposure to sun

The remedy is particularly useful in heat-related or sunstroke-associated migraine. [12]

5. Natrum muriaticum

Frequently indicated in migraine associated with emotional causes such as grief or disappointment.

Key features:

- Headache often left-sided
- Worse from sun exposure
- Occurs before or after menstruation
- Throbbing pain described as "little hammers beating in the brain"
- Visual disturbances such as zigzag lines or temporary blindness preceding headache

Modalities:

- Aggravation (<) after menstruation and from sunrise to sunset
- Amelioration (>) by sleep

Often seen in anemic, reserved, or emotionally sensitive individuals. [12]

XIII DISCUSSION

Migraine is a multifactorial neurovascular disorder with significant individual variability in clinical presentation and trigger factors. Homoeopathy offers a personalized therapeutic approach based on the totality of symptoms and miasmatic background, enabling deeper understanding of disease susceptibility and chronicity. The miasmatic theory provides insight into the underlying dynamic disturbance responsible for recurrent migraine attacks. Remedies such as Natrum muriaticum, Sanguinaria canadensis, Spigelia anthelmia, Bryonia alba, and Glonoinum are frequently indicated based on characteristic symptom similarity and individual constitution. By addressing both functional and constitutional aspects, homoeopathic management aims to reduce frequency, intensity, and recurrence of migraine episodes. This integrative understanding highlights the therapeutic scope of homoeopathy in migraine management.

XIV.CONCLUSION

Migraine is a prevalent and disabling neurovascular disorder that significantly impairs the quality of life and functional capacity of affected individuals. From a homoeopathic standpoint, migraine represents a manifestation of underlying chronic miasmatic influences, reflecting an individual's inherent susceptibility. Homoeopathy emphasizes a holistic and individualized approach to treatment, focusing on the totality of symptoms, constitutional characteristics, and miasmatic background rather than merely addressing symptomatic relief.

The miasmatic perspective provides deeper insight into the chronicity, recurrence, and progression of migraine, thereby facilitating the selection of appropriate constitutional remedies. Homoeopathic medicines such as Sanguinaria canadensis, Natrum muriaticum, Spigelia anthelmia, Bryonia alba, and Glonoinum demonstrate significant therapeutic applicability when prescribed according to the principle of similars and individualization.

Homoeopathic management aims not only to alleviate acute migraine attacks but also to address the underlying dynamic imbalance, thereby reducing the frequency, intensity, and recurrence of episodes. This holistic approach contributes to long-term improvement and overall well-being. Further clinical research and evidence-based studies are recommended to strengthen the scientific validation of homoeopathic management in migraine.

REFERENCES

[1] World Health Organization. Headache disorders. Fact sheet. Geneva: WHO; 2016.
[2] International Headache Society. The International Classification of Headache Disorders, 3rd edition (ICHD-3). Cephalalgia. 2018;38(1):1-211.
[3] Harrison TR, Kasper DL, Fauci AS, Hauser SL, Longo DL, Jameson JL, editors. Harrison's Manual of Medicine. 18th ed. New York: McGraw Hill; 2012.
[4] Hahnemann S. Organon of Medicine. 6th ed. Translated by Dudgeon RE. New Delhi: B. Jain Publishers; 2002.

[5] Dong L, Dong W, Jin Y, Jiang Y, Li Z, Yu D. The global burden of migraine: A 30-year trend review and future projections by age, sex, country, and region. Pain Ther. 2025;14(1):297–315.
[6] Vetvik KG, MacGregor EA. Sex differences in the epidemiology, clinical features, and pathophysiology of migraine. Lancet Neurol. 2017;16(1):76–87.
[7] Davidson S, Innes JA. Davidson's Essentials of Medicine. 2nd ed. London: Churchill Livingstone; 2016.
[8] Mollaoglu M. Trigger factors in migraine patients. J Health Psychol. 2012;18(7):984–94.
[9] Goadsby PJ, Holland PR, Martins-Oliveira M, Hoffmann J, Schankin C, Akerman S. Pathophysiology of migraine: A disorder of sensory processing. Physiol Rev. 2017;97(2):553–622.
[10] Silberstein SD. Migraine pathophysiology and its clinical implications. Cephalalgia. 2004;24 Suppl 2:2–7.
[11] Kelman L. The triggers or precipitants of the acute migraine attack. Cephalalgia. 2007;27(5):394–402.
[12] Boericke W. Pocket Manual of Homoeopathic Materia Medica. Reprint ed. New Delhi: B. Jain Publishers; 2007
[13] Jen JC, Kim GW, Dudding KA, Baloh RW. No mutations in CACNA1A and ATP1A2 in probands with common types of migraine. Arch Neurol. 2004;61(6):926–928.
[14] Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition (ICHD-3). Cephalalgia. 2018;38(1):1–211.
[15] Roberts HA. The Principles and Art of Cure by Homoeopathy. New Delhi: B. Jain Publishers; Reprint edition.
[16] Harrison TR, Kasper DL, Fauci AS, Hauser SL, Longo DL, Jameson JL, editors. Harrison's Principles of Internal Medicine. 20th ed. New York: McGraw Hill; 2018.