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A Brief Review On: Pharmacotherapeutic Role of Aromatherapy in Migraine Management

Varad Damodar Thombre [1], Mr. Ashish Umale [2], Dr. Swati Deshmukh[3]

1. Student, Shraddha Institute Of Pharmacy, Washim , Maharashtra, India.
2. Lecturer, Department of Pharmacy, Shraddha Institute Of Pharmacy, Washim, Maharashtra, India.
3. Professor, Department of Pharmacology, Shraddha Institute Of Pharmacy, Washim, Maharashtra, India.

Abstract

A very common neurovascular condition, migraines are characterized by frequent pulsing headache attacks that are accompanied by nausea, sensory sensitivities, and functional impairment. Despite improvements in contemporary pharmacotherapy, such as NSAIDs, triptans, gepants, ditans, and CGRP-targeted medicines, therapeutic constraints such side effects, contraindications, and expense are fueling an increase in interest in alternative therapies. Aromatherapy has become a viable supplementary therapy that uses volatile plant-based essential oils applied topically or inhaled. Research indicates that essential oils influence neurochemical activity, stress reactions, and neurogenic inflammation by interacting with autonomic and olfactory-limbic pathways. Lavender is the botanical agent with the best clinical evidence for lowering migraine disability, anxiety, and pain. Lotus has neuroactive, sedative, and antioxidant qualities that may help regulate sleep and trigger control, while night jasmine offers anti-inflammatory and analgesic effects. Despite the positive safety and accessibility of aromatherapy, there are still few high-quality clinical research and standardization. Combining evidence-based aromatherapy with traditional medication may improve patient comfort, lessen the burden of triggers, and promote an integrated approach to migraine treatment.

Keywords: Migraine Aromatherapy, Lavender (*Lavandula angustifolia*), Lotus (*Nelumbo nucifera*), Night Jasmine (*Nyctanthes arbor-tristis*), Complementary and Alternative Medicine, Essential Oils, Trigeminovascular System, Neurogenic Inflammation, Pharmacotherapy

1. INTRODUCTION

Migraine is a highly prevalent neurological disorder characterized by episodic attacks of moderate to severe headache accompanied by accompanying features such as nausea, vomiting, photophobia and phonophobia. According to the International Classification of Headache Disorders 3rd edition (ICHD-3), migraine is defined by specific diagnostic criteria including attack duration of 4-72 hours untreated, typical pain characteristics (unilateral, pulsating, moderate intensity, aggravated by routine physical activity) and associated symptoms of nausea and light or sound sensitivity. (ICHD-3). This condition is not only a medical problem but has significant socio-economic consequences, it is among the top causes of years lived with disability (YLDs) globally. [1] The global burden of migraine has escalated markedly. In a 30-year trend review from 1990 to 2021, the estimated prevalence rose by approximately 58 %, from about 732.6 million to 1.16 billion cases. Similarly, incidence and disability-adjusted life years (DALYs) have increased, reflecting the growing impact of migraine across populations and regions**.** [2]

Aromatherapy the use of volatile plant-derived essential oils via inhalation, topical application or diffusion has emerged as a plausible adjunctive intervention in migraine management. The mechanistic rationale is grounded in the neurobiology of olfaction: inhaled aromatic molecules interact with olfactory sensory neurons, project to limbic system structures (such as the amygdala and hippocampus), hypothalamus and brainstem autonomic centres. These networks modulate emotional, autonomic and pain-processing pathways. Essential oils such as those derived from Lavandula angustifolia (lavender), Mentha piperita (peppermint) and others have demonstrated anxiolytic, sedative, analgesic and anti-inflammatory effects in pre-clinical and clinical studies. (e.g., linalool and linalyl acetate in lavender modulating GABAergic and serotonergic systems). [3]

1.1 MIGRAINE

Migraine is a common, often disabling primary headache disorder characterized by recurrent attacks of moderate-to-severe head pain with a characteristic set of associated features (nausea, photophobia, phonophobia) and predictable temporal patterns. The International Classification of Headache Disorders, 3rd edition (ICHD-3), provides the authoritative diagnostic framework: an untreated migraine attack typically lasts 4-72 hours, has at least two of the following pain features (unilateral location, pulsating quality, moderate-to-severe intensity, aggravation by routine physical activity), and is accompanied by nausea and vomiting or photophobia and phonophobia. The ICHD-3 taxonomy differentiates migraine into a number of clinically useful subtypes (for example, migraine with aura, migraine without aura, chronic migraine, and less common variants), which guide clinical assessment, research, and treatment choices. [4]

2. ETIOLOGY OF MIGRAINE

Migraine is understood to result from a complex interplay of multiple etiologic factors rather than a single cause. These etiologies can be grouped into genetic and biological predisposition, hormonal and metabolic factors, environmental and lifestyle triggers, comorbid medical conditions, and structural or neurological susceptibilities. Below is a comprehensive list of etiologic (predisposing) factors described in the literature, followed by an in-depth discussion of three principal general etiologies [5]

2.1 Etiologic Factors

1.Genetic susceptibility and inherited variants (familial aggregation, polygenic risk) [6]

2. influences (menstrual/ovarian hormone fluctuations, estrogen withdrawal)

3.Metabolic and vascular factors (obesity, dyslipidaemia, hypertension, insulin resistance) [7]

4.Neuroinflammatory and oxidative stress states

5.Sleep disorders and circadian disruption

6.Psychological and psychiatric comorbidities (anxiety, depression, stress)

7.Lifestyle triggers (irregular meals, dehydration, caffeine overuse or withdrawal, alcohol, skipping sleep) [8]

2.2 Major Etiologies

Below we discuss in depth genetic susceptibility, hormonal or metabolic influences, and environmental or lifestyle triggers, as these represent broad, general etiologic domains with strong relevance for migraine pathogenesis and are particularly useful in the context of integrative therapies.

a. Genetic Susceptibility

Evidence from twin, family and genome-wide association studies shows that migraine has a substantial hereditary component. Estimates of heritability range from approximately 35-60% for migraine overall, and somewhat higher in the subset of migraine with aura. A recent review noted that migraine is “a complex brain disorder explained by the interaction of genetic and environmental factors.” Genetic loci identified so far number in the hundreds, most of which confer small effect sizes but cumulatively increase risk. In rare subtypes such as familial hemiplegic migraine, monogenic inherited mutations have been defined . [9]

b. Hormonal and Metabolic Influences

Hormonal fluctuations and metabolic disturbances are prominent etiologic factors in migraine, especially in women and in the transition from episodic to chronic forms. The female preponderance of migraine (especially during reproductive age) is well-recognized, and estrogen withdrawal (for example prior to menstruation) is a well-established trigger of attacks. [10]

c. Environmental and Lifestyle Triggers

While genetic and hormonal predisposition constitute the "terrain" for migraine susceptibility, environmental and lifestyle factors almost always serve as proximate triggers that precipitate attack onset or increase attack frequency. These include psychosocial stress, disturbance of sleep (insomnia, insufficient sleep, circadian disruption), irregular meals or fasting, dehydration, caffeine overuse or abrupt withdrawal, bright lights or loud noises, strong odours (olfactory triggers), changes in atmospheric pressure or weather, and air pollution. A recent open-access review of associations found air pollution, education level, gender and other environmental exposures linked with migraine occurrence. [11]

3. EPIDEMIOLOGY OF MIGRAINE

The epidemiology of migraine provides essential context for understanding its public-health significance and informing treatment strategies. Below, we present detailed data on prevalence, incidence, disability, impact, demographic and regional variation, and trends over time, based entirely on open-access reviews and large-scale burden studies.

Migraine is one of the most common neurological disorders worldwide. According to a comprehensive review, the global prevalence of migraine was estimated at about 14.0% in community-based studies. [12]

Migraine is not only common but also highly disabling. The World Health Organization (WHO) notes that headache disorders of which migraine is a major component are among the most common nervous-system disorders, and migraine ranked third among neurological disorders for age-standardised disability-adjusted life years (DALYs) in 2021. A narrative review reports that migraine is a leading cause of years lived with disability (YLDs) and imposes substantial direct and indirect costs (work loss, reduced productivity, health-care utilisation).[10] In children and adolescents, for example, a recent pediatric burden study found 7.52 million DALYs attributable to migraine in 2021 (for ages 5-19), with prevalence and incidence rising. In sum, migraine contributes a major burden both at individual and societal levels. [13]

3.1 Demographic patterns: age, sex, socio-economic strata

Sex differences: Women are disproportionately affected. Meta-analyses and burden studies confirm that prevalence among females is approximately double that among males (lifetime or annual prevalence).

Age patterns: Migraine frequently begins in adolescence and young adulthood; prevalence tends to peak in the 30- to 50-year range. For example, the age-period-cohort analysis among males indicated peak incidence in the 10-14 yr age group but highest prevalence/DALYs in the 30-44 yr window.

Socio-demographic index (SDI) / economic strata: The burden of migraine varies by socio-economic development. Pediatric studies show that low-middle SDI regions carry the highest absolute burden (prevalence, DALYs) in youth, whereas high-SDI regions show higher incidence per population. These demographic patterns imply that both biological susceptibility (age/sex) and social determinants (SDI, access to care) modulate migraine epidemiology. [14]

4. PATHOPHYSIOLOGY OF MIGRAINE

The pathophysiology of Migraine is complex and multifactorial, involving an interplay of neuronal excitability, vascular and neurogenic processes, inflammation, and central sensitization. While numerous mechanisms have been described, three of the most widely accepted and clinically relevant are,

4.1 Activation and sensitization of the trigeminovascular system

The trigeminovascular system comprising trigeminal afferent fibres (particularly the ophthalmic V1 branch), the dura mater and meningeal blood vessels, and second-order neurons in the trigeminal nucleus caudalis or trigeminocervical complex is central to the generation of migraine pain. Early vascular theories of migraine emphasized meningeal vasodilation; however, the current view emphasises nociceptive activation and sensitization of trigeminal afferents, with release of neuropeptides such as Calcitonin gene-related peptide (CGRP) as crucial mediators.

From a therapeutic perspective, the development of anti-CGRP monoclonal antibodies and CGRP-receptor antagonists underscores the clinical relevance of this mechanism. The trigeminovascular model identifies migraine not simply as a vascular disorder, but as a neurovascular and neurogenic pain condition . [15]

4.2 Cortical Spreading Depression (CSD)

CSD is a slowly propagating wave of neuronal and glial depolarization followed by suppression of cortical activity, originally discovered by Leão in 1944. In migraine, CSD is widely accepted as the underlying mechanism of migraine aura and may also trigger the subsequent headache phase via activation of the trigeminovascular system. CSD explains aura symptoms (visual, sensory, language) and provides a mechanistic link to headache onset via trigeminal activation. Therapies aimed at reducing cortical hyper-excitability (e.g., anticonvulsants) may modulate susceptibility to CSD and thereby reduce attack frequency. Because CSD involves cortical depolarization and metabolic stress, modulators of excitability (including non-pharmacologic approaches) might have preventive value. [16]

4.3 Central and Peripheral Sensitization (Neurogenic Inflammation)

Beyond initial nociceptive activation, migraine pathophysiology involves processes of sensitization both peripheral (meningeal afferents) and central (brainstem, thalamus, cortex). Sensitization explains phenomena such as cutaneous allodynia, prolonged headache, and transition from episodic to chronic migraine.

Peripheral sensitization: Following trigeminal activation and CGRP release, meningeal nociceptors and their cell bodies become hyper-responsive, reducing their activation threshold and prolonging nociceptive signalling. Satellite glial cells in the trigeminal ganglion contribute. [17]

by releasing cytokines and nitric oxide, maintaining nociceptor excitability.

Central sensitization: At the level of the trigeminal nucleus caudalis and other brainstem centres, repeated or prolonged afferent input induces changes in synaptic efficacy, receptor expression, and neural network reactivity, leading to pain persistence, allodynia, and chronification. Imaging studies show altered functional connectivity in brainstem and cortical pain-modulatory regions in migraineurs.

5. TYPES OF MIGRAINE

Below we describe the three principal clinical types of migraine that are most relevant to clinical practice, research and therapeutic decision-making: Migraine without aura, Migraine with aura, and Chronic migraine. For each type I provide diagnostic essentials (per ICHD-3), typical clinical features, relevant pathophysiologic notes, epidemiologic pointers, implications for prognosis and treatment, and brief comments on special clinical considerations. All statements are [18]drawn from open-access sources (ICHD-3, open-access reviews and PMC articles) and cited so you can paste them directly into your manuscript .

5.1 Migraine without aura (ICHD-3: G43.0) (Common Migraine)

Migraine without aura is diagnosed when a patient has had at least five attacks fulfilling the criteria: headache attacks lasting 4-72 hours (untreated or unsuccessfully treated); headache with ≥2 of the following characteristics unilateral location, pulsating quality, moderate or severe intensity, and aggravation by routine physical activity and during the headache at least one of the nauseas and vomiting or photophobia and phonophobia. The ICHD-3 text provides the full structured criteria and is the authoritative reference for clinical research and diagnosis.

Head pain is often unilateral (although it can be bilateral) and described as throbbing/pulsatile. Associated symptoms commonly include nausea, vomiting, photophobia, phonophobia and octophobia. Attacks may last from several hours to three days if untreated; interictal periods are typically pain-free. Prodromal and postdromes symptoms (mood change, neck stiffness, cognitive slowing) are common. [18]

Migraine with aura (ICHD-3: G43.1) (Classic Migraine Migraine with aura is defined by the occurrence of transient focal neurological symptoms that typically develop gradually over ≥5 minutes, last 5-60 minutes and are usually followed by the headache phase (although headache can be absent in some attacks). Auras are most often visual (scintillations, fortification spectra, scotoma) but may also be sensory, speech-related, motor (rare hemiplegic migraine), brainstem-type, or retinal. Full ICHD-3 descriptors enumerate typical aura features and subtypes.)

Aura phase: Gradual evolution of positive (e.g., flashing lights) and/or negative (e.g., hemianopia scotoma) neurologic phenomena, usually resolving within an hour. Visual auras account for the overwhelming majority of aura manifestations .

Headache phase: Follows aura in many cases (but not invariably); headache characteristics mirror migraine without aura.

Variability: Some patients experience aura without headache (typical aura without headache) or have alternating patterns of aura and non-aura attacks. [19]

5.3 Chronic migraine (ICHD-3 related coding; G43.7 in some schemes)

Chronic migraine is defined clinically as headache occurring on ≥15 days per month for >3 months, of which ≥8 days per month have the features of migraine (i.e., meet criteria for migraine or respond to migraine-specific therapy). The ICHD-3 criteria and subsequent clinical reviews outline the classification and the need to exclude medication-overuse headache (a frequent complicating factor) or secondary causes.

Patients with chronic migraine experience very frequent or near-daily headaches, often with superimposed migrainous features on many days. Chronic migraine commonly evolves from episodic migraine and is associated with greater functional impairment, higher rates of psychiatric comorbidity (anxiety, depression), sleep disturbance, and medication overuse. Allodynia and features of central sensitization are more commonly observed in chronic migraine.

Migraine without aura is the most common clinical phenotype and represents the majority of cases evaluated in trials; it is the main target for both acute and preventive strategies.Migraine with aura is distinguished by transient focal neurological phenomena (visual symptoms most commonly) and is mechanistically linked to cortical spreading depression; it carries specific diagnostic and vascular-risk considerations. Chronic migraine is a high-burden, frequency-defined phenotype characterized by central sensitization and often by medication overuse; it requires multimodal and often specialist management. [20]

6. SIGNS AND SYMPTOMS

The clinical presentation of Migraine is multifaceted, widely variable, and often temporally phased. Recognizing the constellation of signs and symptoms beyond just headache is essential for accurate diagnosis, effective management, and patient education. This section provides a detailed description of the typical and less-common manifestations of migraine attacks, structured around the commonly described phases (prodrome, aura, headache phase, postdrome) and including associated symptoms (sensory, autonomic, cognitive) supported by evidence from open-access literature. [21]

7.1 Risk Factors for Migraine Development

Risk factors for migraine refer to characteristics (demographic, biological, behavioral) that increase the likelihood of developing the disorder, or of progressing from episodic to chronic migraine. A recent open-access narrative review categorized risk factors into five domains: migraine-disease characteristics, treatment-related factors, comorbidities, lifestyle/exogenous factors and demographic factors. Good evidence supports many of these .

Key risk factors include,

1.Female sex, especially during reproductive years.

2.Younger age of migraine onset, positive family history (genetic susceptibility).[10]

3.High frequency of headache days (≥ 5 per month) and existence of cutaneous allodynia.

4.Medication overuse (frequent acute treatment use) and suboptimal acute therapy.

5.Comorbid conditions: sleep disorders, obesity/metabolic syndrome, psychiatric disorders (depression, anxiety).

6.Lifestyle/exogenous factors: physical inactivity, tobacco use, high caffeine intake, poor sleep hygiene.

7.Sociodemographic/educational factors: lower level of education attainment, financial constraints in some studies.

8. These risk factors are particularly relevant when considering preventive strategies: identifying and modifying risk factors can reduce the frequency or severity of migraine attacks or slow progression to chronic migraine. [22]

8. DIAGNOSIS OF MIGRINE

Diagnosis of migraine is clinical, based primarily on a detailed patient history and symptom pattern aligned with the International Classification of Headache Disorders, 3rd edition (ICHD-3) criteria. Investigations such as neuroimaging or laboratory tests are used only to exclude secondary causes when red-flag features are present. The diagnosis therefore integrates history, physical/neurological examination, and appropriate use of investigations based on clinical suspicion.

Migraine is commonly underdiagnosed especially in primary care due to overlapping features with tension-type headache, sinus headache, and medication-overuse headache. Using structured diagnostic tools improves accuracy, especially for non- specialists.

8.1 List of Diagnostic Approaches in Migraine

a. Clinical Diagnostic Tools & Criteria

1. ICHD-3 diagnostic criteria (gold standard)
2. Headache history and symptom profile (pain characteristics, associated symptoms)
3. Migraine screening tools ID-Migraine™ questionnaire

HIT-6 (Headache Impact Test)

MIDAS (Migraine Disability Assessment)

4. **Headache diary or trigger diary**

b. Physical & Neurological Examination

1. Full neurological exam (to rule out secondary causes)
2. Autonomic and cranial nerve evaluation
3. Assessment for allodynia and neck involvement

c. Diagnostic Investigations

1. MRI brain (preferred in atypical cases)
2. CT scan (in acute severe situations)
3. Blood investigations (if infection, systemic illness suspected)
4. Lumbar puncture (rare; only if meningitis/SAH suspected [23]

9. TREATMENT OF MIGRAINE

The pharmacologic treatment of migraine may be acute (abortive) or preventive (prophylactic), and patients with frequent severe headaches require both approaches. Preventive therapy is used to reduce the frequency, duration, or severity of attacks. Additional benefits may include enhancement of response to acute treatments, improvement of a patient's ability to function, and reduction of disability.3 Preventive treatment may also result in reduction of health care costs.

Modern management of migraine involves a combination of acute (abortive) and preventive (prophylactic) therapies, supported by lifestyle and behavioral interventions. Allopathic (evidence-based medical) treatment focuses on targeting the underlying neurovascular and neurochemical mechanisms, especially trigeminovascular activation and CGRP pathways.[39]

9.1 Acute (Abortive) Pharmacotherapy

Used to stop or relieve symptoms during an active migraine attack.

NSAIDs & Analgesics

Ibuprofen, naproxen, diclofenac, aspirin, acetaminophen

First-line for mild to moderate attacks

5.Reduce inflammation and nociceptive signaling

Triptans (5-HT1B/1D agonists)

Sumatriptan, rizatriptan, zolmitriptan

Gold standard for moderate to severe attacks

Mechanism: vasoconstriction + inhibition of CGRP release + trigeminal inhibition

Gepants (CGRP receptor antagonists)

Ubrogepant, rimegepant

Suitable for patients who cannot take triptans

No vasoconstriction; effective in acute treatment[38]

Ditans (5-HT1F selective agonist)

Lasmiditan

Reduces trigeminal nociception without vascular effects

Useful for patients with cardiovascular contraindications

Antiemetics

Metoclopramide, domperidone, prochlorperazine

Helpful in nausea/vomiting; improve absorption of other drugs

Emergency-care options

IV NSAIDs, steroids (dexamethasone), magnesium sulfate

Used for status migrainosus (>72 hours) [24]

10. AROMATHERAPY IN MIGRAINE

A growing body of research suggests that aromatherapy defined as the inhalation or topical application of plant-derived essential oils may serve as a complementary therapeutic strategy in the management of migraine. This section reviews the mechanistic rationale, evidence base, delivery methods, safety considerations, and limitations of aromatherapy in migraine care, drawing exclusively on open-access literature.

10.1 Mechanistic Rationale

a. Olfactory–Limbic–Autonomic Modulation

Aromatherapy relies on inhaled volatile compounds that bind olfactory receptors, project via the olfactory bulb to the limbic system (amygdala, hippocampus), hypothalamus and brainstem autonomic and nociceptive modulatory centres. These pathways influence emotion, autonomic tone (sympathetic/parasympathetic balance), pain threshold and stress responses. In migraine, where dysregulation of sensory, autonomic, neurovascular and limbic networks occurs, olfactory modulation offers a plausible adjunctive pathway to reduce cortical excitability, lower trigeminovascular activation, and modulate neurogenic inflammation. [25]

b .Ainflammatory and Analgesic Phytochemistry

Many essential oils contain active constituents known to exert analgesic, anti-inflammatory, sedative and vasorelaxant effects. For example, the volatile compounds linalool and linalyl acetate (in lavender oil) have demonstrated GABAergic modulation, reduction in calcium influx, and vasorelaxant activity in preclinical model further, a 2023 review found that essential oils reduced migraine intensity and attack frequency in a meta-analysis of small studies [26]

c.Trigger Modulation and Autonomic Reset

Because many migraine attacks are precipitated by triggers such as stress, strong odours, sensory overload, poor sleep and autonomic imbalance, aromatherapy may serve as an intervention to blunt these triggers by promoting relaxation, improving sleep and reducing sympathetic overactivity. For instance, inhalation of calming essential oils may reduce arousal, improve sleep latency and reduce wake episodes. [27]

10.2 Methods of Delivery & Protocols

a. Inhalation (via diffuser or direct sniffing of essential oil drops) is the most commonly studied method in migraine and appears to allow rapid central nervous system access via olfactory pathways. For example, in the Sasannejad trial, patients inhaled lavender oil for 15 minutes.

b.Topical application (e.g., temple/forehead rubbing of essential-oil diluted with carrier oil) has also been used in headache studies though direct migraine data are fewer.

Important parameters to standardize in research and practice include oil chemotype and purity (GC-MS profiling), dosage or number of drops, inhalation time or distance, control/placebo conditions, frequency of use, duration of intervention, and co-interventions (e.g., foot bath, massage). [28]

10.3 Safety, Contraindications & Practical Considerations

Aromatherapy is generally well-tolerated when used appropriately, but certain safety issues must be considered,

- Essential oils may cause skin irritation, allergic contact dermatitis or phototoxicity if used undiluted topically.
- Inhalation may trigger respiratory symptoms in individuals with asthma or airway hyperreactivity

- Because migraine patients often have sensory hypersensitivity, fragrances may paradoxically act as triggers unless carefully chosen and controlled.
- Essential oils may interact with the autonomic system; caution in individuals with severe cardiac disease, epilepsy or pregnancy (data lacking).

Standardization issues: Many commercial essential-oil products vary in composition, concentration and purity this variability limits reproducibility and comparability of studies. [29]

10.4 Practical Role in Migraine Management

Given the current evidence, aromatherapy may be considered as an adjunctive therapy in migraine, rather than a standalone replacement for evidence-based pharmacotherapy. Practical clinical roles include,

• Early symptom intervention: using inhalation of calming essential oils (such as lavender) during prodromal or mild headache phase to potentially reduce progression or shorten attack duration.

• Trigger-modulation: integrating aromatherapy into self-management plans aimed at stress reduction, sleep improvement, autonomic regulation or odor-trigger desensitisation.

• Supportive care: combined with relaxation/massage for patients with frequent attacks, high anxiety/comorbidity burden or seeking non-pharmacologic adjuncts.

• Careful implementation should involve patient education on safe use, monitoring for adverse effects, consistent choice of high-quality essential-oil product, and documentation of response (headache diary, frequency or intensity tracking, disability scores such as MIDAS or HDI). Early small trials demonstrate measurable benefits in disability reduction with lavender inhalation.[46]

• Aromatherapy represents a promising complementary modality in migraine management, with mechanistic plausibility (via olfactory-limbic-autonomic pathways and analgesic/anti-inflammatory phytochemistry) and early clinical evidence demonstrating reduced headache intensity and disability. Particularly, lavender inhalation has the strongest evidence among essential oils studied. However, given methodological limitations, aromatherapy should currently be regarded as an adjunct to, not replacement for, guideline-based pharmacologic and non-pharmacologic migraine care. With further high-quality research, aromatherapy may increasingly support integrative migraine treatment models, especially in patients with high trigger burden, sensory/autonomic dysregulation or intolerance to conventional therapy. [30]

11. HERBS USED IN TREATMENT OF MIGRAINE

Herbs Used in Treatment of Migraine by Aromatherapy

11.1 Lavender - *Lavandula angustifolia*

A .Pharmacology (neurobehavioral & analgesic effects) 1.Anxiolytic / sedative: Preclinical and human studies show lavender EO reduces anxiety and produces calming effects; mechanisms include modulation of GABAergic activity and interaction with serotonergic transporters (SERT). Linalool shows anxiolytic effects in rodents and appears to enhance GABA-ergic neurotransmission. [31]

2.Analgesic / antinociceptive :

Lavender EO and linalool produce analgesic effects in several pain models; proposed actions include inhibition of NMDA-mediated excitatory . [32]

3. **Autonomic modulation:** Inhalation produces measurable changes in autonomic markers (reduced sympathetic tone), which is relevant because autonomic imbalance and stress are migraine triggers. [31]

B.MOA (Role of Lavender in Migraine):

1.Olfactory → limbic modulation: rapid effects on amygdala/hypothalamus reduce stress/anxiety and HPA activation, lowering attack probability in stress-triggered migraine. [31]

2.GABAergic and serotonergic modulation: may increase inhibitory tone and oppose cortical hyperexcitability (theoretical preventive benefit) and modulate pain perception. [32]

3.Anti-inflammatory / antioxidant activity: reduces neurogenic inflammation that sustains trigeminovascular sensitization. [33]

C.**Use:** Lavender is one of the most widely studied aromatic medicinal plants; its essential oil (EO) is used for anxiolysis, sleep

disorders, and pain relief across cultures .

11.2 Night Jasmine - *Nyctanthes arbor-tristis*

Pharmacology (analgesic, anti-inflammatory, neuroprotective):

1. Anti-inflammatory & antioxidant: Multiple invitro and in-vivo studies show inhibition of proinflammatory mediators, antioxidant scavenging, and decreased inflammatory markers actions relevant for neurogenic inflammation in migraine. [34]

2. Analgesic / antipyretic: Traditional and preclinical evidence supports analgesic and antipyretic effects, possibly via peripheral cyclooxygenase (COX) suppression and central nociceptive modulation. [34]

3. Neuroprotective / sedative effects: Some extracts show CNS depressant activity in animal models, suggesting potential calming effects that could reduce stress-related triggers. [35]

b.Mechanisms relevant to migraine (MOA)

1.Anti-inflammatory attenuation of neurogenic inflammation: By reducing inflammatory mediators and oxidative stress, night jasmine constituents could lower peripheral trigeminal nociceptor sensitization and production of pro-nociceptive peptides (e.g., NO, cytokines). This addresses the inflammatory component of the trigeminovascular cascade. [34]

2.Analgesic action via central and peripheral pathways: Flavonoids and iridoids may modulate nociceptive neurotransmission centrally and peripherally, reducing pain perception during attacks.

3.Anxiolytic / sedative adjunct: Sedative constituents may complement lavender's anxiolysis, helping to blunt stress-mediated triggers. [35]

Use: Night jasmine is a traditional Ayurvedic plant used for fever, headache, inflammation and nervous complaints. [34]

11.3 Lotus *Nelumbo nucifera*

Pharmacology (neuroactive, antioxidant, sedative):

- **Sedative / anxiolytic effects**: Alkaloids such as nuciferine show sedative and antipsychotic-like activities in animal models, with modulation of dopaminergic and serotonergic receptors; these effects may improve sleep and reduce stress-related triggers. [36]

- **Antioxidant / anti-inflammatory:** Lotus extracts demonstrate free-radical scavenging and reduced inflammatory markers in vitro and in vivo properties that could counter oxidative stress implicated in migraine pathogenesis. [37]

- **Vasoactive and cardioprotective influences:** Some lotus constituents affect vascular tone and platelet aggregation relevant because cerebrovascular reactivity contributes to migraine pathophysiology. However, inhalation of volatile lotus constituents is less studied for direct vascular effects. [38]

Mechanisms relevant to migraine (MOA):

1. **Modulation of neurotransmitter systems:** Alkaloids can interact with dopaminergic/serotonergic systems, potentially stabilizing neurotransmitter imbalances associated with migraine susceptibility (e.g., dopaminergic hypersensitivity). This may reduce prodromal symptoms and attack permissiveness.

2. **Antioxidant/anti-inflammatory action:** By reducing oxidative stress and inflammation, lotus constituents could attenuate processes that sustain central sensitization and neuronal hyperexcitability. [39]

3. **Sedative/sleep-promoting effects:** Improving sleep quality decreases a major trigger for attacks; the calming pharmacology of lotus supports this preventive pathway. [40]

Use: Sacred lotus has a long history in Ayurvedic and Traditional Chinese Medicine for calming, sleep disorders, cardiovascular conditions and CNS complaints. Multiple parts (seed, leaf, plumule, flower) yield different phytochemical profiles. [36]

Lavender has the strongest open-access clinical evidence among the three herbs for symptomatic migraine relief via inhalation, acting primarily through olfactory-limbic GABAergic/serotonergic and autonomic modulation. Night jasmine and lotus offer plausible and complementary pharmacology anti-inflammatory/analgesic and neuroactive/antioxidant effects respectively but lack direct inhalation trials in migraine. A standardized, GC-MScharacterized perfume blend combining these herbs has theoretical

multimodal benefit (anxiolytic + anti-inflammatory + neuroprotective) but requires rigorous preclinical standardization and randomized clinical trials (with safety monitoring, chemotype reporting and mechanistic biomarkers) before clinical recommendation. [31]

1. **DIFFERENCE BETWEEN ALLOPATHIC AND HERBAL TREATMENT**

Parameter	**Allopathic (Modern Pharmacotherapy)**	**Herbal (Lotus, Night Jasmine, Lavender**
Primary Aim	Rapid relief of acute symptoms; prevention of future attacks through pharmacologic modulation	Modulation of stress, inflammation, autonomic balance and sensory pathways to reduce attack severity/frequency
Mechanism of Action	Triptans → 5-HT1B/1D agonism; Gepants → CGRP receptor blockade; NSAIDs → COX inhibition; Preventives → β-blockade, GABAergic stabilization, CGRP suppression	Lavender → GABAergic & serotonergic modulation, anxiolysis; Night Jasmine → anti-inflammatory & analgesic activity; Lotus → antioxidant, neuroactive (dopaminergic/serotonergic) balancing
Target Pathways	Trigeminovascular system, CGRP signaling, cortical excitability, neuroinflammation	Limbic–olfactory pathways, autonomic nervous system, oxidative stress reduction, mild anti-inflammatory and analgesic pathways
Onset of Action	Minutes to hours (acute therapy like triptans); weeks for preventive drugs	Rapid calming effect within minutes (lavender); anti-inflammatory/preventive effects over
Clinical Evidence (Migraine)	Strong evidence (large RCTs, clinical guidelines, long-term data)	Limited but growing evidence; lavender has small RCTs; night jasmine and lotus mostly preclinical/ethnopharmacologic
Effectiveness	High when appropriately prescribed; significant reduction in intensity and frequency	Mild to moderate benefit; best used as adjunct therapy; consistent ben
Side Effects	Nausea, dizziness, chest pressure (triptans); weight gain, sedation (preventives); medication-overuse headache if overused	Generally mild: possible fragrance sensitivity, headache in odor-sensitive patients, rare allergies; limited inhalation safety data for lotus/night jasmine
Risk Level	Moderate–high if contraindications present (CV disease, pregnancy, drug interactions	Low–moderate; avoid in asthma, pregnancy, or fragrance-triggered migraine
Accessibility	Requires prescription (preventives, triptans, CGRP mAbs); costs may be high	Easily accessible; low cost; can be selfadministered with guidance
Personalization	Adjusted based on comorbidities, severity, attack frequency, and response	Personalized based on preferred scents, sensitivity to odors, trigger profile, and response

Preventive Role	Strong preventive options: CGRP-mAbs, β-blockers, topiramate, Botox	May support long-term prevention by improving sleep, reducing stress & oxidative burden
Appropriate Use	Primary treatment for moderate–severe migraine; essential for chronic migraine	Adjunctive therapy; useful for mild attacks, prodrome phase, relaxation & stress-trigger modulation
Limitations	Cost, adverse effects, drug interactions, need for monitoring	Lack of standardization, variable purity of essential oils, limited RCTs, placebo challenges
Overall Role in Migraine Care	Core, evidence-based medical management	Supportive, complementary therapy that enhances wellbeing & may reduce trigger load .

Table No. 1: Difference Between Allopathic and Herbal Treatment

CONCLUSION

Conclusion

Migraine is a complex neurovascular disorder that involves trigeminovascular activation, cortical hyperexcitability, and dysregulation of sensory and autonomic pathways. While modern medical therapies such as NSAIDs, triptans, gepants, ditans, and CGRP-targete remain central to migraine management, their side effects, cost, and limitations highlight the value of supportive complementary approaches. Aromatherapy offers a practical adjunct by influencing olfactory–limbic pathways, autonomic balance, and neurotransmitter activity, with additional mild anti-inflammatory and antioxidant benefits.

Among the essential oils studied, **lavender** has the strongest evidence for reducing migraine pain and emotional distress. **Night jasmine** provides notable anti-inflammatory and analgesic effects, while **lotus** contributes calming, neuroactive, and antioxidant properties that may help reduce stress-related triggers. Together, these botanicals offer a multi-targeted aromatherapeutic approach that can complement conventional treatment.

Although current findings are promising, larger and more standardized clinical studies are needed to confirm efficacy, establish optimal dosing, and ensure long-term safety. Overall, aromatherapy with lavender, night jasmine, and lotus represents a scientifically supported and patient-friendly adjunct that may enhance integrative migraine care and improve quality of life.

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