European Journal of Neurology

european journal of neurology
the official journal of the european academy of neurology

REVIEW ARTICLE OPEN ACCESS

Interventions for Migraine and Sleep: A Systematic Review Exploring Their Bidirectional Association

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Received: 5 June 2025 | **Revised:** 29 September 2025 | **Accepted:** 16 October 2025

Keywords: insomnia | migraine | non-pharmacological interventions | pharmacological interventions | sleep disorders

ABSTRACT

Background: Migraine and sleep disturbances share a bidirectional relationship, influencing each other's frequency and severity. The aim of this systematic review is to examine the effects of migraine-targeted interventions on both migraine outcomes and sleep parameters (including sleep quality and insomnia symptoms), as well as the effects of sleep-focused interventions on both sleep and migraine outcomes.

Methods: Following PRISMA 2020 guidelines, a systematic search was conducted across six databases (PubMed, Medline, Scopus, Embase, PsycINFO, CINAHL) for studies published until December 5, 2023. Eligible studies included Randomized Clinical Trials, Controlled Clinical Trials, and observational studies assessing migraine and/or sleep-targeted interventions in adults. The risk of bias was evaluated using RoB 2 and ROBINS-E tools.

Results: Twenty-three studies (1941 participants) were included. Pharmacological treatments such as erenumab, amitriptyline, propranolol, and onabotulinumtoxinA reduced migraine frequency and pain intensity, with variable effects on sleep quality. Melatonin showed no significant impact. Among non-pharmacological treatments, percutaneous electrical nerve stimulation, greater occipital nerve block, green light therapy, binaural beats, mindfulness, and dietary modifications improved both migraine symptoms and sleep. Digital Cognitive-Behavioral Therapy for Insomnia (CBT-I) significantly reduced headache days and improved sleep parameters, whereas evidence on standard CBT-I was mixed.

Limitations: Study heterogeneity, small sample sizes, and variability in outcome measures limit generalizability. Few studies focused on sleep-targeted interventions and their effects on migraine, highlighting a research gap.

Conclusions: Integrated approaches combining migraine and sleep interventions show promise for symptom management. Further research is needed to refine treatment strategies and assess long-term effects.

Registration: CRD42024617217.

1 | Introduction

Among all primary headache disorders, migraine has the strongest association with sleep, with substantial evidence supporting their bidirectional relationship [1, 2]. This connection is reinforced by both clinical experience and an extensive body of literature demonstrating the frequent co-occurrence of sleep disturbances and migraine in the general population [3].

1.1 | The Connection Between Migraine and Sleep

Sleep plays a dual role in migraine, acting as both a trigger and a protective factor. On one hand, sleep deprivation, poor sleep quality, and irregular sleep patterns are well-established triggers of migraine attacks, contributing to increased headache frequency and severity [4–6]. On the other hand, sleep is often reported as a therapeutic factor, with many migraine sufferers experiencing symptom relief following sleep [2, 7–8]. These opposing effects highlight the importance of stabilizing sleep patterns, through regular circadian timing, consistent sleep schedules, and improvements in both subjective and objective sleep quality (e.g., sleep efficiency, reduced fragmentation), as a potential strategy for migraine management.

Patients with migraine frequently report poor sleep quality, as assessed through both self-reported questionnaires and objective sleep studies. The Pittsburgh Sleep Quality Index (PSQI), a widely used self-reported measure of sleep quality, consistently shows higher scores in migraine patients compared to healthy individuals, particularly in those with chronic migraine [9]. Objective sleep assessments further confirm these findings, revealing alterations in sleep architecture, such as reduced slow-wave sleep, prolonged sleep latency, increased sleep fragmentation, and decreased sleep efficiency [10]. However, evidence from polysomnographic studies remains limited, as most investigations are based on small sample sizes and single-night recordings, which may not fully capture patients' habitual sleep patterns.

Beyond general sleep disturbances, migraine patients are also at a higher risk of comorbid sleep disorders, including insomnia, restless legs syndrome (RLS), and obstructive sleep apnea (OSA) [7, 11–12]. These conditions can further exacerbate migraine symptoms and contribute to a more significant burden of disease, worsening disability and reducing quality of life [13].

1.2 | The Reciprocal Influence of Migraine and Sleep Treatments

Emerging evidence suggests a reciprocal influence between migraine and sleep, with migraine treatments potentially improving sleep quality and sleep-targeted interventions reducing migraine frequency and severity [2, 7, 14]. Addressing sleep disturbances may represent a valuable therapeutic strategy for alleviating migraine burden, while optimizing migraine management could lead to better sleep outcomes.

Given the increasing interest in non-pharmacological migraine treatments, several interventions have been assessed for their potential role in clinical practice. Acupuncture, mindfulness, nerve stimulation, massage therapy, and dietary modifications are frequently investigated as complementary or alternative treatments. Some of these approaches have shown promise in reducing headache frequency and improving sleep quality [15–17]. Additionally, cognitive-behavioral therapy for insomnia (CBT-I) and other behavioral sleep interventions have shown efficacy in improving both sleep parameters and migraine burden [18, 19]. However, the real-world applicability remains debated, and further studies are needed to define their optimal integration into standard migraine care.

On the pharmacological side, new migraine-specific treatments are reshaping the therapeutic landscape. Among these, monoclonal antibodies targeting the calcitonin gene-related peptide (CGRP) pathway represent a significant breakthrough in migraine prevention. These therapies have demonstrated substantial efficacy in reducing migraine frequency, but their impact on sleep quality remains under investigation [20, 21]. Furthermore, several conventional migraine preventive medications, such as tricyclic antidepressants and beta-blockers, have been reported to modulate sleep parameters, although their effects remain variable across studies [7, 22]. Understanding the impact of these pharmacological treatments on sleep could help develop personalized therapeutic strategies for patients experiencing both migraine and sleep issues.

1.3 | The Objective of This Systematic Review

This systematic review aims to evaluate the strength of current evidence regarding the bidirectional relationship between migraine and sleep by estimating:

1. The impact of migraine-targeted treatments (both pharmacological and non-pharmacological) on both migraine outcomes (frequency and intensity) and sleep parameters (sleep quality and insomnia symptoms).
2. The effects of sleep-targeted interventions (both pharmacological and non-pharmacological) on both sleep outcomes (sleep quality and insomnia symptoms) and migraine outcomes (frequency and severity).

By systematically analyzing available research, this review seeks to determine whether these effects are robust and clinically relevant, offering insights into potential integrated treatment approaches for patients with comorbid migraine and sleep disturbances. Distinct from prior reviews that examined either the association between sleep and migraine or the effect of migraine prophylaxis on sleep alone, the present review synthesizes both directions across pharmacological and non-pharmacological interventions. In doing so, it provides a comprehensive overview that spans conventional prophylactic drugs, novel anti-CGRP therapies, behavioral and digital interventions, and sleep-targeted treatments. This bidirectional framing is intended to inform more integrated and clinically relevant management strategies.

2 | Methods and Materials

The search process and this systematic review were conducted in compliance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [23], and the protocol was prospectively registered in PROSPERO (CRD42024617217).

2.1 | Information Sources and Search Strategy

Beginning in November 2023, two independent reviewers performed a systematic search of published studies across several electronic databases, including PubMed, Medline, Scopus, Embase, PsycINFO, and Cinahl. All records published before December 5th, 2023, were considered.

The literature search was conducted using the following search string in the respective databases:

(sleep OR insomnia) AND migrain AND (psych* OR cogniti* OR behav* OR mindful* OR therap* OR treat* OR intervention OR modif* OR acupuncture OR neurostimulation OR TDCS OR non-invasive brain stimulation OR NIBS OR TACS OR stimulation OR non-pharmacological OR melatonin OR drug* OR pharmacol* OR benzodiazepin* OR z-drug* OR zdrug* OR benzodiazepine receptor agonist OR DORA OR dual orexin receptor antagonist OR antidepress* OR hypnotic* OR gaba OR antiepileptic* OR beta blocker* OR gepants OR antiepileptics OR calcium channel blocker OR anti-hypertensive OR monoclonal antibodies OR anti serotoninergic OR onabotulinumtoxin OR botox).*

In order to minimize language bias, the literature search was conducted in multiple languages; however, only studies published in English were ultimately deemed suitable for inclusion.

2.2 | Eligibility Criteria

The selection of eligible studies followed clearly defined inclusion and exclusion criteria. Studies that met the following conditions were included in the review:

- Adult participants diagnosed with migraine with and without aura, based on the second and third editions of the International Classification of Headache Disorders (ICHD-II and ICHD-III) [24, 25].
- Assessment of sleep quality using instruments such as the Insomnia Severity Index (ISI), Pittsburgh Sleep Quality Index (PSQI), Total Sleep Time (TST), Sleep Efficiency (SE), Wake After Sleep Onset (WASO), self-reported Visual Analog Scales (VAS), or Likert scales.
- At least one intervention targeting either improvement in sleep quality or prevention of migraine episodes.

No restriction was placed on study design: both randomized and non-randomized designs, including observational studies, were considered eligible.

The exclusion criteria were as follows:

- Presence of sleep disorders other than insomnia. Studies involving participants with conditions such as sleep apnea, periodic limb movement disorder, RLS, or hypersomnia were excluded.
- Diagnosis of other primary or secondary headache disorders other than migraine and migraine with medication-overuse headache.
- Major psychiatric comorbidities, such as depression, anxiety, or post-traumatic stress disorder.

2.3 | Selection Process

The initial search yielded 7661 studies, distributed as follows: 1765 from PubMed, 2343 from Embase, 2215 from Medline, 674 from PsycINFO, 640 from CINAHL, and 24 from Scopus. A total of 2660 duplicate records were removed using the Rayyan collaboration and research tool.

The remaining 5001 articles underwent a preliminary screening based on titles and abstracts, followed by full-text evaluations. Studies that did not meet the eligibility criteria or lacked full articles, as well as reviews and meta-analyses, were excluded.

Of the 25 full-text reports assessed for eligibility, two were further excluded after discussion, as they focused on medication withdrawal therapy for medication-overuse headache, which was not considered a treatment option in this review. The flow diagram illustrating the study selection process is provided in Figure 1.

2.4 | Data Collection Process, Data Items, and Effect Measures

Two reviewers meticulously reviewed the selected articles, and the extracted data were cross-checked to ensure accuracy and consistency in the assessment. Disagreements and unresolved issues were addressed and settled through discussion with a third reviewer.

The following data were identified as relevant for extraction:

- Publication characteristics: title, authors, year of publication, journal, and study design.
- Sample characteristics: number of participants, age, gender, and type of migraine.
- Treatments: interventions targeting migraine or sleep, whether pharmacological or non-pharmacological.
- Measures: assessment tools such as questionnaires, diaries, scales, and tests.

The main outcomes for this review were the quality of sleep, the frequency of migraine and headache episodes, and the intensity of migraine pain.

Sleep quality was assessed using several established tools, including the Pittsburgh Sleep Quality Index (PSQI), Insomnia Severity Index (ISI), Total Sleep Time (TST), Sleep Efficiency (SE), Wake After Sleep Onset (WASO), or self-reported VAS and Likert scale to evaluate sleep quality. In this review, we use the term "sleep quality" in a broad sense, encompassing both subjective questionnaires (primarily PSQI and ISI) and commonly reported sleep continuity parameters (e.g., TST, SE, WASO). This choice reflects the heterogeneity of outcome measures across the available literature and acknowledges that no single instrument captures the multidimensional nature of sleep. We

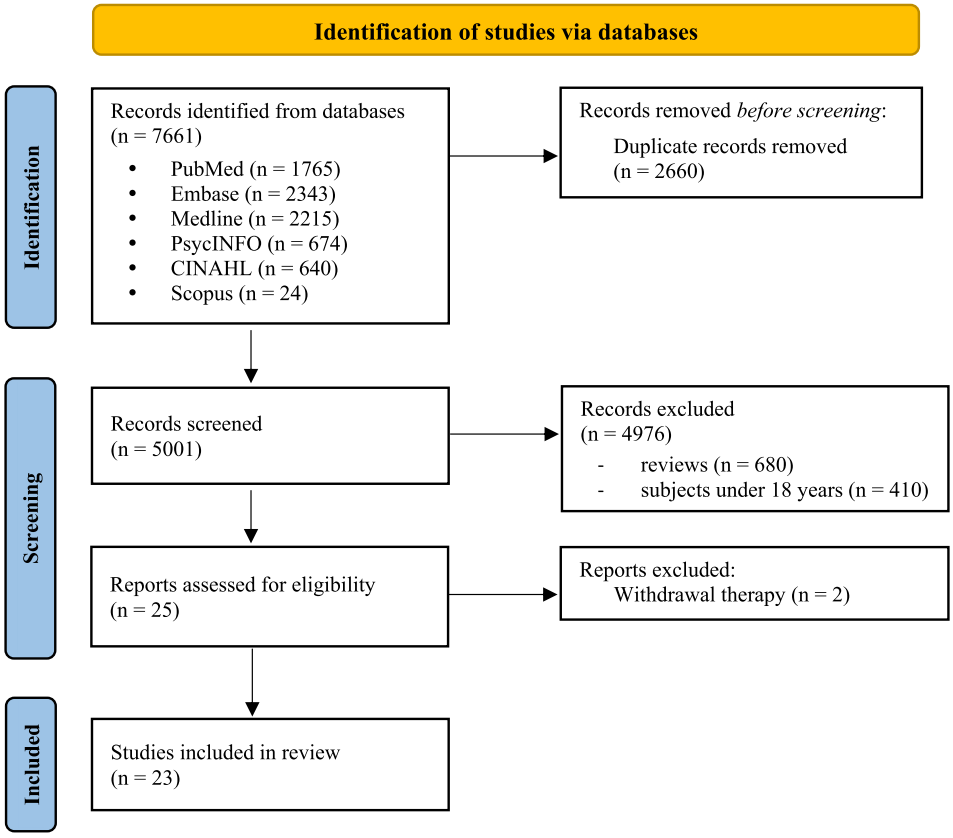

FIGURE 1 | Flowchart of the identification of studies.

recognize that these indices represent distinct aspects of the sleep experience rather than global sleep quality, yet including them provides a more comprehensive overview of how migraine treatments and sleep-targeted interventions may influence different dimensions of sleep.

For migraine-related outcomes, the primary measures were migraine days and headache days within a specific period (commonly a month). Days of headache are defined as any day on which the patient reports the presence of a headache, without necessarily meeting the criteria for migraine as outlined by the ICHD. In contrast, a migraine day is defined as a day with an episode that fulfills the criteria of the ICHD for a migraine attack or for which the patient has taken specific analgesic therapy for migraine [26]. Chronic migraine is defined as headache occurring on 15 or more days per month for more than 3 months, which, on at least 8 days per month, has the features of migraine headache [27].

Pain intensity was measured using both the VAS and the Numeric Rating Scale (NRS), each ranging from 0 (no pain) to 10 (worst possible pain). The VAS requires participants to mark a point on a line indicating their pain level, while the NRS involves selecting a number that best reflects their pain intensity. These tools are widely used for tracking headache severity and facilitating effective pain management [28, 29].

2.5 | Study Risk of Bias Assessment

The methodological quality of included studies was assessed using the Cochrane Risk of Bias 2 (RoB 2) tool for randomized controlled trials [30] and the Risk of Bias in Non-Randomized Studies of Exposures (ROBINS-E) tool for non-randomized studies [31]. These tools provide a structured approach to evaluating the risk of bias across multiple domains.

The RoB 2 tool assesses the risk of bias across five main domains: (1) randomization process, (2) deviations from the intended intervention, (3) missing outcome data, (4) quantification of the outcome, and (5) selection of the reported results. The overall risk of bias for each study is determined by the highest level of bias observed in any domain. For non-randomized studies, the ROBINS-E tool assesses seven domains: (1) bias due to confounding, (2) bias arising from measurements of the exposure, (3) bias in the selection of participants into the study or the analysis, (4) bias due to post-exposure intervention, (5) bias due to missing data, (6) bias arising from measurements of the outcome, (7) bias in the selection of the reported results. To provide a comprehensive visualization of the risk of bias assessments, the Robvis tool [32] was used to generate graphical summaries. The evaluation was performed by one author under the supervision of the entire research team, ensuring consistency and accuracy in the assessment process.

2.6 | Risk of Bias in Studies

The following figures summarize the risk of bias assessment for both non-randomized (Figure 2) and randomized (Figure 3) studies included in this review.

In non-randomized studies assessed with the ROBINS-E tool, 2 studies were rated at low risk of bias, 8 at some concerns, and 3

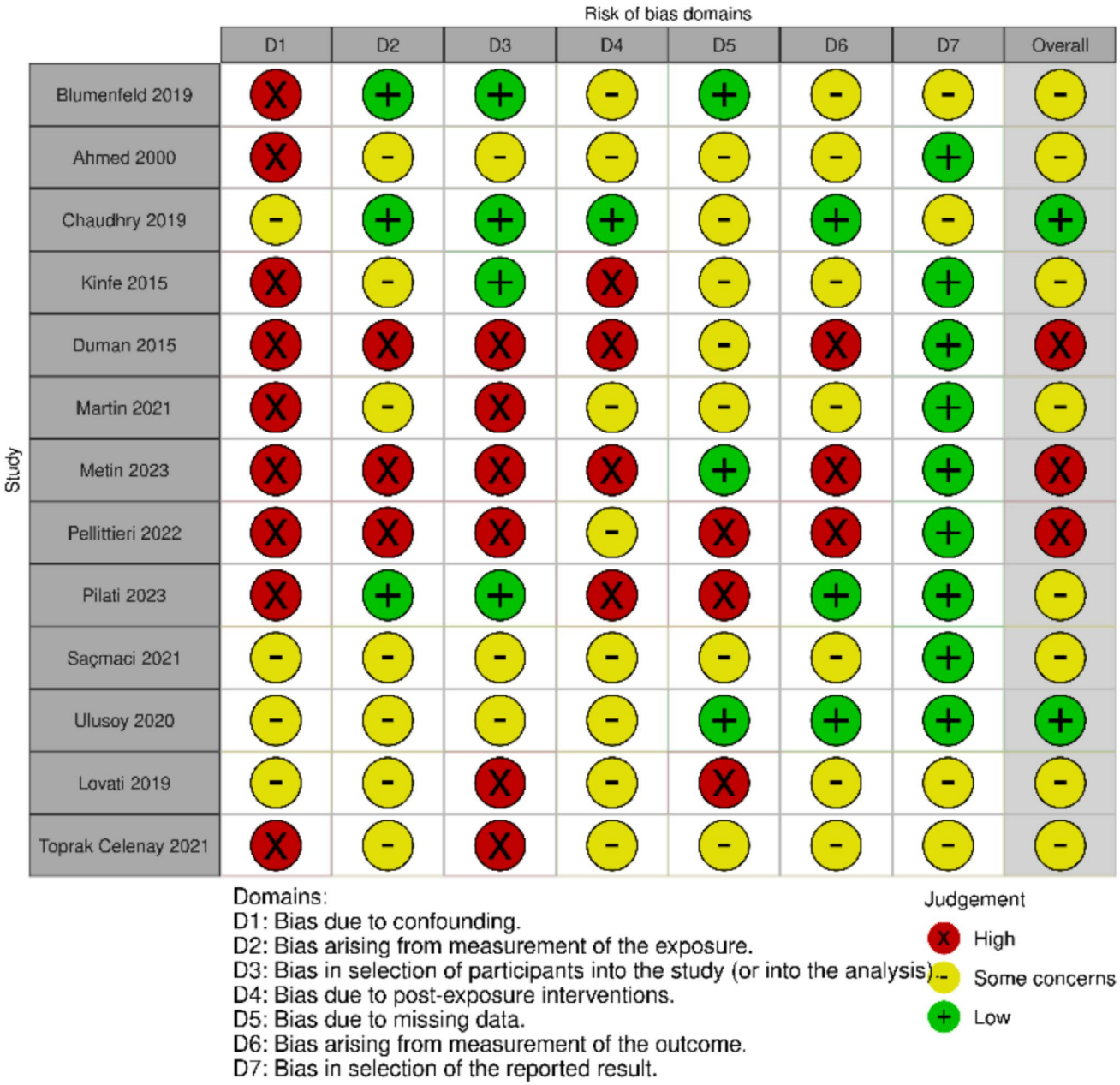

FIGURE 2 | Risk of bias evaluations for non-randomized studies assessed using the ROBINS-E tool. Each study was categorized based on bias domains, with color-coded indicators representing high risk (red), some concerns (yellow), and low risk (green).

at high risk. The domains most frequently affected were bias due to confounding (D1, 9 studies) and selection of participants (D3, 6 studies). Other domains, including outcome measurement and missing data, were generally less problematic.

In randomized controlled trials assessed with RoB 2, of a total of 8 studies, 4 were classified as green (meaning low risk of bias) and the remaining 4 studies were rated yellow (meaning some concerns); no trial was judged at high risk of bias. Most concerns related to selection of the reported result (D5), while additional issues were noted in the randomization process (D1, 3 studies) and measurement of the outcome (D4, 3 studies). Deviations from intended interventions (D2) were less common (2 studies), and no concerns emerged for missing outcome data (D3).

3 | Results

A summary of the primary extracted data is reported in Table 1.

To enhance coherence with our research questions, we first report findings for migraine-targeted interventions (effects on sleep and migraine outcomes), followed by sleep-targeted interventions (effects on sleep and migraine outcomes).

3.1 | Pharmacological Migraine Treatments

Five studies in the selected literature focused on the use of pharmacological treatments for migraine management and evaluated their effect on sleep outcomes.

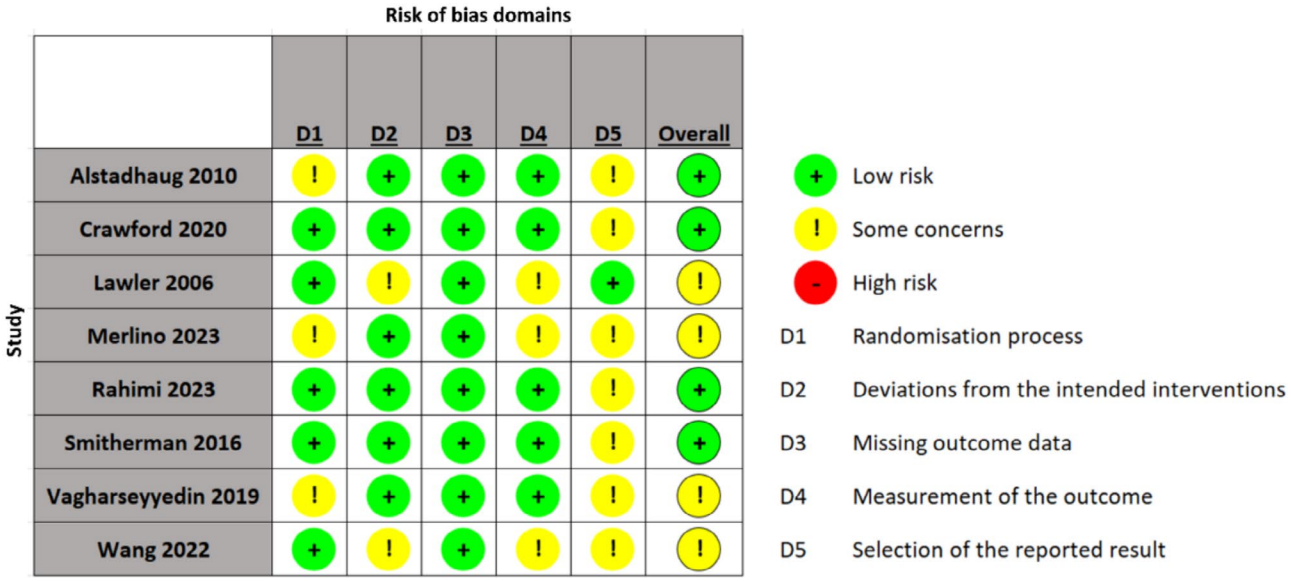

FIGURE 3 | Risk of bias assessment for randomized controlled trials using the RoB 2 tool, detailing potential concerns in areas such as randomization, missing data, and measurement bias. Each study was categorized based on bias domains, with color-coded indicators representing high risk (red), some concerns (yellow), and low risk (green).

3.1.1 | Amitriptyline

Amitriptyline, a tricyclic antidepressant commonly used for migraine prophylaxis, was examined in a study conducted by Duman et al. [22]. In this research, 40 patients with migraine were treated with amitriptyline, and the outcomes on migraine frequency, pain intensity, and sleep quality were assessed over 3 months.

The study demonstrated significant reductions in the number of headache attacks throughout the treatment. Baseline migraine attacks were 5.37 ± 4.42, which decreased to 4.06 ± 3.25 after 1 month and further declined to 2.56 ± 1.86 after 3 months ($p = 0.045$). Additionally, pain intensity, measured using the VAS, also showed significant improvement. The VAS score started at 8.00 ± 1.23 at baseline and dropped to 6.11 ± 2.05 after 1 month and to 4.51 ± 2.06 after 3 months ($p = 0.003$).

Sleep quality, as assessed by the PSQI, also improved significantly over the course of the treatment. The baseline PSQI score of 5.56 ± 2.70 decreased to 4.06 ± 2.11 after 1 month and further dropped to 2.56 ± 1.75 by the third month ($p = 0.008$).

In summary, amitriptyline significantly reduced migraine frequency, decreased pain intensity, and improved sleep quality over a 3-month treatment period in migraine patients [22]. These findings may be partly explained by amitriptyline's sedative effects, which could contribute to its beneficial impact on sleep in addition to its efficacy in migraine prophylaxis.

3.1.2 | Propranolol

The effects of propranolol, a beta-blocker frequently prescribed for migraine prevention, were also evaluated in the same study by Duman et al. [22]. This study involved 44 patients, and the results showed a similar improvement in migraine frequency and sleep quality as observed with amitriptyline.

The number of migraine attacks significantly decreased from 5.27 ± 2.89 at baseline to 3.54 ± 2.26 after 1 month and 1.92 ± 1.32 after 3 months of propranolol treatment ($p < 0.001$). However, while the severity of migraine pain, measured through VAS, showed a reduction (from 7.98 ± 1.54 at baseline to 4.36 ± 2.50 at 3 months), this change was not statistically significant ($p = 0.666$).

Propranolol also had a significant positive impact on sleep quality. PSQI scores decreased from 6.24 ± 2.27 at baseline to 3.68 ± 1.57 after 1 month and 2.55 ± 1.42 after 3 months of treatment ($p < 0.001$).

To sum up, propranolol significantly reduced migraine attack frequency and improved sleep quality, though the reduction in pain intensity was not statistically significant [22]. However, in contrast to these findings, propranolol and other beta-blockers are widely known to be associated with sleep disturbances, including fragmented sleep, vivid dreams, and nightmares [48]. This discrepancy suggests that the effects of propranolol on sleep may be complex and potentially influenced by individual patient characteristics or compensatory mechanisms in migraine patients.

3.1.3 | Erenumab

Two studies examined the efficacy of erenumab, a monoclonal antibody targeting the calcitonin gene-related peptide receptor, for migraine prevention [20, 21]. Both studies reported significant reductions in the number of monthly migraine days and pain severity following 3 and 12 months of treatment.

Pellitteri et al. [20] reported that 27.6% of patients achieved a $\geq 50\%$ reduction in monthly migraine days at 3 months ($p = 0.001$), with similar results at 12 months ($p = 0.001$). The proportion of patients rating their migraines as "severe" also decreased (27.6%–17.2%).

TABLE 1 | Primary extracted data.

Article	Country	Sample, *N* (f/m)	Study design	Treat. target	Treat. type	Treatment
Ahmed et al. (2000) [33]	United States	12 (8/4)	CT	M	Non-ph	Percutaneous electrical nerve stimulation
Alstadhaug et al. (2010) [34]	Norway	46 (39/7)	RCT	M	ph	Melatonin
Blumenfeld et al. (2019) [35]	United States	716 (607/109)	CT	M	ph	Onabotulinumtoxina 155 U
Burrowes et al. (2022) [16]	United States	98 (89/9)	RCT	M	Non-ph	Mindfulness-based stress reduction
Chaudhry et al. (2019) [36]	Germany	48 (44/4)	CCT	M	Non-ph	Non-invasive vagus nerve stimulation
Crawford et al. (2020) [19]	United States	42 (42/0)	RCT	S	Non-ph	Digital Cognitive-Behavioral Therapy for Insomnia
Duman et al. (2015) [22]	Turkey	84 (77/7)	CT	M	ph	Propranolol, amitriptyline
Faurot et al. (2023) [37]	United States	182 (161/21)	RCT	M	Non-ph	Dietary intakes of omega fatty acids
Kinfe et al. (2015) [38]	Germany	20 (16/4)	OS	M	Non-ph	Non-invasive vagus nerve stimulation
Lawler et al. (2006) [39]	New Zealand	47 (39/8)	RCT	M	Non-ph	Massage therapy
Lovati et al. (2019) [40]	Italy	21 (20/1)	OS	M	Non-ph	Binaural beats
Martin et al. (2021) [41]	United States	29 (27/2)	CT	M	Non-ph	Green and white light exposure
Merlino et al. (2023) [42]	Italy	70 (58/12)	CT	M	Non-ph	Ketogenic diet
Metin et al. (2023) [43]	Turkey	20 (20/0)	OS	M	Non-ph	Greater occipital nerve block with lidocaine
Pellitteri et al. (2022) [20]	Italy	29 (25/4)	OS	M	ph	Erenumab
Pilati et al. (2023) [21]	Italy	88 (75/13)	OS	M	ph	Erenumab
Rahimi et al. (2023) [17]	Iran	63 (63/0)	RCT	M	Non-ph	Eye movement, diaphragmatic breathing
Saçmacı et al. (2021) [44]	Turkey	37 (31/6)	OS	M	Non-ph	Greater occipital nerve block
Smitherman et al. (2016) [18]	United States	31 (28/3)	RCT	S	Non-ph	Cognitive-Behavioral Therapy for Insomnia
Toprak Celenay et al. (2023) [45]	Turkey	16 (16/0)	CCT	M	Non-ph	Connective tissue massage
Ulusoy et al. (2020) [46]	Turkey	84 (72/12)	OS	M	Non-ph	Greater occipital nerve block
Vagharseyyedin et al. (2019) [47]	Iran	76 (30/46)	RCT	M	Non-ph	Acupressure
Wang et al. (2022) [15]	China	82 (82/0)	RCT	M	Non-ph	Yang style Tai Chi Chuan

Abbreviations: CCT, controlled clinical trial; CT, clinical trial; M, migraine; Non-ph, non-pharmacological; OS, observational study; ph, pharmacological; RCT, randomized clinical trial; S, sleep.

Pilati et al. [21] found a significant reduction in monthly migraine days with erenumab, particularly at 140 mg. At 3 months, migraine days decreased from 21.62 ± 5.10 to 10.94 ± 7.87 ($p < 0.001$) in the 70 mg group and from 25.71 ± 5.67 to 9.32 ± 8.75 ($p < 0.001$) in the 140 mg group, with benefits persisting at 12 months.

In terms of sleep quality, Pellitteri et al. [20] reported significant improvements in several components of the PSQI after 3 months of erenumab treatment, including "subjective sleep quality" ($p = 0.023$) and "sleep disturbances" ($p = 0.034$). After 12 months, improvements in "daytime dysfunction" were also noted ($p = 0.046$). On the other hand, Pilati et al. [21] found no significant changes in PSQI scores over time, although sleep efficiency decreased significantly among responders to erenumab, from 78% ± 15% at baseline to 57% ± 0% after 12 months.

In conclusion, erenumab effectively reduced the frequency and severity of migraine, with mixed results regarding its impact on sleep quality. While Pellitteri et al. [20] found improvements in several sleep parameters, Pilati et al. [21] observed a decline in sleep efficiency among responders, though no significant overall changes in PSQI scores were reported.

3.1.4 | OnabotulinumtoxinA 155 U

OnabotulinumtoxinA, more commonly known as Botox, has been approved for the prevention of chronic migraine. Blumenfeld et al. [35] conducted a study to assess the long-term effects of this treatment on migraine frequency and sleep quality in chronic migraine patients.

The study showed a statistically significant reduction in the number of headache days over the 108-week trial period. At baseline, patients experienced an average of 22.0 (4.8) headache days per month. This number decreased by 7.4 days after 24 weeks of treatment and by 10.7 days after 108 weeks. Additionally, sleep quality, as measured by the PSQI, improved significantly over the treatment period. The average PSQI score decreased from 13.3 (3.7) at baseline to 11.0 (3.7) after 108 weeks ($p < 0.001$), with an intermediate score of 11.7 (3.6) at week 60.

In conclusion, OnabotulinumtoxinA 155 U led to significant reductions in headache days and improvements in sleep quality among chronic migraine patients [35].

3.1.5 | Melatonin

Alstadhaug et al. [34] investigated the effect of prolonged-release melatonin (2 mg taken 1 h before bedtime) as a migraine prophylactic treatment in a crossover study. The primary outcome was the mean attack frequency during melatonin treatment compared to placebo.

The study found no significant differences in the reduction of migraine frequency between the melatonin and placebo groups. Both groups experienced a similar reduction in migraine attack frequency, with a 33% reduction in the melatonin group and a 30% reduction in the placebo group. However, the study did observe improvements in sleep quality with melatonin treatment. There was a notable decrease in global PSQI scores following melatonin therapy compared to placebo, although this was not statistically significant ($p = 0.09$). Among patients with insomnia, melatonin showed a more pronounced effect on improving sleep quality.

In summary, while melatonin did not significantly reduce migraine frequency compared to placebo, it did show some potential in improving sleep quality, particularly among patients with pre-existing sleep disturbances [34].

The following Table 2 summarizes the results of pharmacological treatments for migraine.

3.2 | Non-Pharmacological Migraine Treatments

In addition to primary pharmacological approaches for migraine, several non-drug therapies have emerged as valid alternatives.

3.2.1 | Nerve Stimulation

Of the reviewed studies, three specifically explored nerve stimulation as a potential treatment for migraine [33, 36, 38]. Specifically, Ahmed et al. [33] evaluated percutaneous electrical nerve stimulation (PENS), while Kinfe et al. [38] and Chaudhry et al. [36] investigated non-invasive vagus nerve stimulation (nVNS).

In the study by Ahmed et al. [33], PENS was compared to a "needles only" placebo. Both groups underwent the insertion of 32-gauge (0.2 mm), 15-mm-long stainless-steel needles into the soft tissue at specific points on the neck and scalp, following a standardized protocol. However, only the PENS group had their needles connected to electrical leads. The study measured changes in headache frequency, pain severity, and sleep quality. After PENS treatment, weekly headache occurrences decreased from 6 ± 1 to 3 ± 2 ($p < 0.05$), while the placebo group showed no improvement. Pain scores evaluated by a VAS scale dropped from 7.6 ± 1.1 to 3.0 ± 0.7 in the PENS group ($p < 0.05$), compared to a modest decrease in the placebo group. Sleep quality also significantly improved in the PENS group compared to the placebo group (VAS baseline = 5.2 ± 0.8; VAS post PENS = 2.9 ± 0.6).

Similarly, two studies examined the effects of nVNS. Kinfe et al. [38] demonstrated a substantial reduction in headache days per month, from 14.7 ± 0.9 to 8.9 ± 0.8 days ($p < 0.001$), with improvements in both episodic and chronic migraine patients. Chaudhry et al. [36], however, reported no significant differences in the number of headache days between the sham and nVNS groups. However, they found a significant reduction in the number of severe migraine attacks per month in the nVNS group, from 7.64 ± 1.44 to 2.93 ± 1.03, compared to the sham group ($p < 0.05$). Pain intensity, as measured by a visual analog scale, was significantly reduced in Kinfe et al.'s [38] study, particularly in both episodic and chronic migraine subgroups (from 8 points, interquartile range 7.5–8.0, to 4 points interquartile range 3.5–5, after 3 months of nVNS use; $p < 0.001$). No significant changes in pain intensity were observed in Chaudhry's [36] study. Sleep quality, assessed by the PSQI, improved significantly in Kinfe et al.'s [38] study (from 7 points interquartile range 5.5–11.5, to post nVNS 5 points

TABLE 2 | Outcomes of pharmacological migraine treatments.

Migraine pharmacological treatments	(Dose—frequency)	Effect on migraine frequency (f)	Effect on migraine intensity (i)	Effect on sleep quality (sq)
Amitriptyline	(12 weeks)	↓ f (number of headache attacks per month)	↓ i	↑ sq
Propranolol	(12 weeks)	↓ f (number of headache attacks per month)	ns	↑ sq
Erenumab	(70/140 mg monthly—52 weeks)	↓ f (monthly migraine days)	/	contradictory results
OnabotulinumtoxinA	(155 Units every 12 weeks—108 weeks)	↓ f (monthly headache days)	/	↑ sq
Melatonin	(2 mg every day—8 weeks)	ns	/	ns

Note: ↓ f = significant decrease in migraine frequency; ↓ i = significant decrease in migraine intensity; ↑ sq. = significant improvement in sleep quality.
Abbreviation: ns, non-significant result.

interquartile range 5–8.5, p-value < 0.001) but showed no significant differences in Chaudhry's [36] work.

In summary, nerve stimulation has shown promise in treating migraine patients with sleep disturbances. Ahmed et al. [33] observed significant reductions in headache frequency and intensity with PENS, alongside improved sleep quality. Kinfe et al. [38] found that nVNS reduced headache days and pain intensity and improved sleep, while Chaudhry et al. [36] reported no major changes in headache frequency but did find a reduction in severe attacks in the nVNS group.

3.2.2 | Visual and Auditory Stimulation

Sensory stimulation therapies, which include visual and auditory interventions, can be used as approaches that may reduce migraine severity and frequency. Two key studies focused on these approaches: Lovati et al. [40] investigated auditory stimulation using binaural beats, a music-therapeutic approach that uses two different frequencies (alpha and theta) played through headphones to foster relaxation and enhance sleep, while Martin et al. [41] studied the impact of visual stimulation through exposure to green and white light.

In Lovati et al.'s [40] study, participants listened to binaural beats before sleep for 90 consecutive nights. The results showed that the average number of migraine days per month dropped from 14.9 to 13.3 across all participants. Among responders—defined as those who experienced more than a 30% reduction in migraine days—the frequency of migraine days decreased significantly, from 12.9 to 6.3 ($p = 0.009$). Overall, 52.4% of participants reported an improvement in their headaches, while 38.1% noticed no change and 9.5% experienced a worsening.

Martin et al. [41] explored the effects of white light-emitting diodes (WLED) and green light-emitting diodes (GLED) on patients with episodic and chronic migraines. The study showed a slight, non-significant reduction in headache days after WLED exposure, from 7.75 ± 1.35 to 5.87 ± 1.26 for episodic migraine patients, and from 23.12 ± 1.43 to 21.47 ± 1.92 for chronic migraine patients. However, GLED exposure led to significant reductions in headache days for both groups: from 7.86 ± 1.59 to 2.43 ± 1.11 ($p = 0.0156$) in the episodic migraine group and from 22.32 ± 1.25 to 9.42 ± 1.63 days ($p = 0.0003$) in the chronic migraine group. Overall, headache days decreased from 18.42 ± 1.62 to 7.54 ± 1.36 days ($p < 0.0001$), with a responder rate of 86% for EM and 63% for migraine patients.

In terms of pain intensity, Lovati et al. [40] reported a significant decrease among responders, as measured by the VAS scale, with a reduction in pain intensity ($p = 0.02$). Similarly, Martin et al. [41] noted that pain scores decreased significantly after GLED exposure, from 7.29 ± 0.56 to 3.43 ± 0.43 for EM patients ($p = 0.0156$) and from 8.23 ± 0.27 to 3.09 ± 0.53 for migraine patients ($p < 0.0001$). When data from both groups were combined, pain scores dropped from 8.00 ± 0.25 to 3.17 ± 0.41 ($p < 0.0001$).

Both studies also examined the impact of sensory stimulation on sleep quality. Lovati et al. [40] found no significant changes in sleep onset latency or sleep fragmentation (such as arousals during the night) using a sleep diary, though 52.4% of participants reported a global subjective improvement in sleep satisfaction. In contrast, Martin et al. [41] used a modified version of the University of Arizona Pain Clinic follow-up questionnaire and found that both episodic and chronic migraine patients experienced significant improvements in their ability to fall asleep and stay asleep after GLED exposure. For episodic migraine patients, the ability to fall asleep improved from 7.50 ± 5.26 to 70.00 ± 11.25 ($p = 0.0013$), and the ability to stay asleep improved from 10.00 ± 5.34 to 70.00 ± 11.25 ($p = 0.0023$). Chronic migraine patients similarly showed improved sleep, with falling asleep scores rising from 6.32 ± 4.60 to 55.88 ± 7.33 ($p < 0.0001$) and staying asleep scores increasing from 5.00 ± 4.32 to 59.33 ± 7.84 ($p < 0.0001$).

In conclusion, visual and auditory stimulation therapies have shown promise in reducing migraine frequency and intensity. Lovati et al.'s [40] binaural beats therapy yielded positive results in reducing migraine days and pain intensity for responders,

while Martin et al.'s [41] green light therapy significantly reduced headache days and pain intensity in both episodic and chronic migraine patients, also leading to improved sleep quality.

3.2.3 | Greater Occipital Nerve Block (GONB)

The use of greater occipital nerve block (GONB) as a migraine treatment involves the administration of a local anesthetic near the greater occipital nerve to alleviate pain and reduce inflammation [49]. Three studies focused on the impact of this treatment on migraine patients [43, 44, 46]. Saçmacı et al. [44] and Ulusoy et al. [46] used bupivacaine, a long-acting local anesthetic, while Metin et al. [43] opted for lidocaine, a short-acting alternative.

All three studies reported significant reductions in the frequency of headaches among chronic migraine patients. In Saçmacı et al.'s [44] study, headache days per month decreased from 20.2 ± 5.5 to 4.8 ± 5.5 ($p < 0.001$), while Ulusoy et al. [46] observed a reduction from 21 (15–30) to 4 (1–24) in the first month, which further dropped to 3 (1–18) by the third month ($p < 0.001$). Similarly, Metin et al. [43] reported a decrease in headache days from 18 (15.5–25.5) to 7 (6–8) after treatment ($p < 0.001$).

Pain intensity also saw notable declines. Saçmacı et al. [44] recorded a 53% reduction in pain intensity, with scores dropping from 8.4 ± 1.2 to 4.5 ± 2.3. Ulusoy et al. [46] reported a drop in VAS scores from 10 (8–10) to 7 (5–9) in the first month, with similar results persisting in the third month. Metin et al. [43] noted a reduction in VAS scores from 8 (7–9) to 5 (4–5.5) after treatment ($p < 0.001$).

In terms of sleep quality, Saçmacı et al. [44] observed significant improvements in PSQI scores, which decreased from 7.6 ± 3.6 to 3.4 ± 2.2, indicating a 45% improvement. Subgroup analysis revealed significant improvements in sleep latency, efficacy, and subjective sleep quality, with sleep duration increasing from 6.8 ± 1.3 h to 7.3 ± 0.8 h after treatment ($p = 0.004$). Ulusoy et al. [46] also reported significant reductions in PSQI scores from baseline (11 [1–20]) to the first (6 [1–18]) and third months post-treatment (5 [0–16]) ($p < 0.001$). Metin et al. [43] similarly noted improvements in PSQI scores ($p = 0.026$).

In conclusion, GONB using both bupivacaine and lidocaine significantly reduced headache frequency and pain intensity in chronic migraine patients. Improvements in sleep quality were also observed, with significant reductions in PSQI scores reported by Saçmacı et al. [44] and Ulusoy et al. [46], while Metin et al. [43] noted non-significant improvements.

3.2.4 | Mind–Body Exercise

Several studies have investigated the effectiveness of mind–body techniques in reducing migraine frequency and improving sleep quality. Three specific studies examined mindfulness, tai chi, and combinations of eye movement exercises with diaphragmatic breathing [15–17].

Burrowes et al. [15, 17, 35] compared enhanced mindfulness-based stress reduction (MBSR+) with active control, while Wang et al. [15] evaluated the effects of a modified 12-week Tai Chi Chuan training program. Rahimi et al. [17] investigated body–mind training techniques, incorporating eye movement exercises paired with jogging (EME + J) and diaphragmatic breathing paired with jogging (DB + J).

Mindfulness meditation significantly reduced the number of headache days from 7.8 (6.9–8.8) to 4.6 (3.7–5.6) over 52 weeks in the MBSR+ group, with statistically significant differences at 10 and 20 weeks ($p = 0.04$) [16]. Rahimi et al. [17] reported that EME + J decreased headache days from 9.15 (2.68) to 3.61 (1.24) at post-test and to 3.44 (1.79) at follow-up ($p < 0.001$), while DB + J reduced them from 8.22 (4.00) to 3.61 (1.46) at the post-test and to 4.70 (1.21) at follow-up (12 months) ($p < 0.001$). On the other hand, Wang et al. [15] did not assess migraine symptoms, such as frequency or intensity, as primary outcomes. Instead, their study focused on the beneficial effects of Tai Chi in reducing systolic blood pressure and mitigating migraine-triggering factors, including stress levels, fatigue, and sleep quality [15].

Regarding migraine intensity, Burrowes et al. [16] found no significant changes in headache pain intensity following mindfulness meditation. However, Rahimi et al. observed a statistically significant reduction in pain intensity in both EME + J (from 6.50 (1.67) to 2.94 (0.72) at post-test and 3.50 (1.29) at 12-month follow-up) and DB + J groups (from 6.11 (1.32) to 2.20 (0.83) at post-test and 3.20 (0.95) at follow-up) ($p < 0.001$) [17].

Sleep quality improved significantly in both mindfulness and Tai Chi groups, as reflected in PSQI score reductions compared to controls ($p < 0.01$) [15, 16]. Additionally, Rahimi et al. [17] found that EME + J and DB + J significantly improve sleep patterns, including waking-up mode (feeling refreshed vs. tired upon waking) and sleep regularity anticipating the bad time ($p < 0.05$).

Overall, mind–body exercises such as mindfulness meditation and combined breathing exercises were associated with a reduction in both migraine frequency and intensity [16, 17]. While, Tai chi demonstrated beneficial effects on secondary migraine triggers such as stress, fatigue, and blood pressure [15]. Furthermore, eye movement exercises, diaphragmatic breathing with jogging, tai chi, and mindfulness all contributed to significant improvements in sleep quality [15, 17, 35].

3.2.5 | Massage Therapy

Massage therapy has also been studied as a complementary and alternative treatment for migraine, as demonstrated by three studies [39, 45, 47]. Lawler et al. [39] focused on the effects of a 13-week massage therapy regimen, during which migraine patients were randomly assigned to either a massage group or a control group. The massage group received weekly 45-min sessions targeting the back, shoulders, neck, and head. Celenay et al. [45] investigated the use of connective tissue massage (CTM), while Vagharseyyedin et al. [47] examined acupressure, a non-invasive manual stimulation of specific acupoints.

Lawler et al. [39] found a significant reduction in migraine frequency in the massage group compared to the control group. The average number of migraine days dropped from baseline to intervention (1.00 ± 0.24; $p < 0.01$), and this reduction was maintained at follow-up (1.07 ± 0.28, $p < 0.05$). In Celenay et al.'s [45] study, the CTM group experienced a decrease in migraine frequency from a median of 4 (2–7) to 1 (0–3) at follow-up ($p < 0.001$), while the control group showed no significant changes.

Regarding pain intensity, Lawler et al. [39] found no significant Group × Time interaction effects when comparing the massage group to the control group at both the intervention and follow-up stages. Similarly, in Celenay's [45] study, the CTM group experienced a reduction in pain intensity during treatment, from 6.3 (5.2–7.8) to 0.0 (0.0–5.4), but these improvements were not sustained at follow-up.

In terms of sleep quality, Lawler et al. [39] reported a significant improvement in the massage group, as sleep quality increased between baseline (23.33 ± 0.86) and intervention (24.75 ± 0.88; $p < 0.01$), with further improvements at follow-up (25.24 ± 0.87; $p < 0.005$). Celenay et al. [45] found that PSQI scores improved post-treatment for the CTM group, from a median of 9.0 (6.0–10.8) to 6.0 (5.3–10.3), although the results were not statistically significant. Vagharseyyedin et al.'s [47] acupressure study recorded a reduction in PSQI scores, but the difference between the acupressure and control groups was not significant.

In summary, massage therapy, including neuromuscular and connective tissue techniques, reduced migraine frequency and improved sleep quality. However, no significant changes in pain intensity were observed [39, 45]. Acupressure had no statistically significant effect on sleep quality compared to a control group [47].

3.2.6 | Diet-Based Interventions

Two studies focused on dietary approaches to manage migraine and improve sleep quality. One examined the effects of a ketogenic diet (KD) [42], while the other investigated the role of a high omega-3 diet (H3) and a high omega-3 and low omega-6 diet (H3L6) [37].

Merlino et al. [42] reported that all migraine-related symptoms improved significantly after following a ketogenic diet. Migraine intensity decreased from 8.1 ± 1.0 at baseline to 5.3 ± 2.8 at follow-up ($p < 0.001$), and the number of headache days per month reduced from 17.9 ± 8.3 to 8.4 ± 9.2 ($p < 0.001$). Similarly, Faurot et al. [37] found that omega fatty acid intake led to a 19% and 22% improvement in pain intensity across the two intervention groups, compared to 9% in the control group.

Regarding sleep quality, Merlino et al. [42] noted that PSQI scores decreased significantly from 8.0 ± 4.0 at baseline to 5.7 ± 3.7 at follow-up ($p < 0.001$). All components of the PSQI showed improvements following the ketogenic diet. Faurot et al. [37] also reported improvements in sleep quality (evaluated employing a 4-level Likert-style question) in the omega fatty acid diet groups compared to the control group, with overall sleep quality scores increasing from 2.5 (0.49) at baseline to 2.7 (2.6–2.7) post-intervention in the H3L6 group and 2.7 (2.6–2.8) in the H3 group.

In conclusion, diet-based interventions, such as the ketogenic diet and omega fatty acid supplementation, were found to significantly reduce migraine intensity and frequency, while also improving sleep quality [37, 42]. The ketogenic diet in particular showed marked improvements in both migraine symptoms and sleep among patients [42].

The following Table 3 summarizes the results of non-pharmacological treatments for migraine.

3.3 | Non-Pharmacological Sleep Treatments

Two studies examined non-pharmacological interventions targeting sleep in migraine patients, specifically CBT-I [18, 19]. One study, by Smitherman et al. [18] evaluated the efficacy of CBT-I in a cohort of migraine patients with comorbid insomnia, while Crawford et al. [19] investigated the use of a digital version of CBT-I (dCBT-I).

In Smitherman et al. [18] both the CBT-I group and the control group showed reductions in headache frequency during the study. The CBT-I group experienced a 26.9% reduction in headache frequency after treatment, from 22.7 to 16.6 days per month, while the control group showed a 36.2% reduction, from 19.6 to 12.5 days per month. However, during the follow-up period, the CBT-I group showed further reductions, with a 48.9% decrease in headache frequency compared to a 25.0% reduction in the control group. Participants who received CBT-I were also 60% less likely to experience headaches at follow-up. Seven (43.8%) CBT-I participants experienced at least a 50% reduction in headache frequency during follow-up compared to five (33.3%) participants in the control group. Although these findings are promising, the differences between the CBT-I and control groups were not statistically significant regarding migraine symptoms, suggesting that the effect of CBT-I on headache frequency may not be robust or specific.

Crawford's [19] dCBT-I study found that migraine frequency decreased from 21 days per month (SD = 7.0) to 18.4 days per month (SD = 8.7) after treatment (mean difference = −2.6, 95% CI: −4.58; −0.7). The severity of migraine pain also decreased significantly, from a baseline of 6.5 (SD = 1.4) to 5.4 (SD = 1.3) after treatment, with a mean difference of 1.1 (95% CI: −1.5; −0.6).

In terms of sleep quality, Smitherman et al. [18] reported significant improvements in PSQI scores in the CBT-I group, from 11.3 (4.4) at baseline to 7.0 (3.1) at follow-up, while the control group showed no significant changes. Crawford et al. [19] reported a significant decrease in ISI scores after dCBT-I, from a baseline of 17.6 (SD 4.0) to 7.7 (SD 4.1), with a mean difference of −9.9 (95% CI: −11.7; −8).

In conclusion, non-pharmacologic sleep interventions such as CBT-I and dCBT-I have shown promising results in improving sleep quality in migraine patients. While dCBT-I was associated with a reduction in headache frequency and pain intensity, traditional CBT-I primarily improved sleep parameters without a

TABLE 3 | Outcomes of non-pharmacological migraine treatments.

Migraine non-pharmacological treatments	Intervention	Effect on migraine or headache frequency (f)	Effect on pain intensity (i)	Effect on sleep quality (sq)
Nerve stimulation	PENS	↓ f (headache days per week)	↓ i	↑ sq
	nVNS	Contradictory results	Contradictory results	Contradictory results
Visual and auditory stimulation	Binaural tones	↓ f (migraine days per month)	↓ i	ns
	Green light	↓ f (headache days per month)	↓ i	↑ sq
Occipital nerve block	Bupivacaine	↓f (headache days per month)	↓ i	↑ sq
	Lidocaine	↓ f (number of days with pain)	↓ i	↑ sq
Mind–body exercises	Mindfulness	↓ f (headache days per month: significant results at 10 and 20 weeks, but not at 52 weeks)	ns	↑ sq
	Tai Chi	/	/	↑ sq. (significant results at 12 weeks, but not at 24 weeks)
	Eye movement	↓ f (headache days per month: significant change between pre-test and post-test, but not between post-test and follow-up)	↓ i (significant change between pre-test and post-test, but not between post-test and follow-up)	↑ sq
	Diaphragmatic breathing	↓ f (headache days per month)	↓ i	↑ sq.
Massage	Massage	↓ f (migraines per week)	↓ i	↑ sq
	Connective tissue	↓ f	↓ i	↑ sq
	Acupressure	/	/	ns
Diet	Ketogenic diet	↓f (headache days per month)	↓ i	↑ sq
	High omega-3 diet	/	↓ i	↑ sq
	High omega-3 low omega-6 diet	/	↓ i	↑ sq

Note: ↓ MD = significant decrease in migraine days; ↓ HD = significant decrease in headache days; ↓ i = significant decrease in pain intensity; ↑ sq. = significant improvement in sleep quality.
Abbreviation: ns, non-significant result.

significant impact on migraine symptoms in comparison with the control group [18, 19].

The following Table 4 summarizes the results of non-pharmacological treatments for sleep.

4 | Discussion

This review systematically examined the effects of both pharmacological and non-pharmacological interventions on migraine management and sleep quality, assessing treatments aimed at migraine, sleep quality, insomnia symptoms, or a combination of these factors. The overall findings indicate that significant improvements in sleep quality, migraine frequency, and pain severity were observed with both types of interventions.

These observed therapeutic effects may be partially explained by the shared anatomical and neurochemical pathways between migraine and sleep, which further underscore their intrinsic connection. Key brain structures involved in both conditions include the brainstem, hypothalamus, thalamus, and cerebral cortex, many of which are components of the trigeminovascular system [50]. Neurotransmitters such as serotonin, dopamine, norepinephrine, and orexin, which play roles in both pain modulation and sleep regulation, are likely to mediate the link between sleep disturbances and migraine attacks [7]. For instance, serotonin, a key regulator of sleep–wake cycles, has been implicated in migraine pathophysiology, with alterations in serotonergic transmission being associated with both poor sleep quality and increased migraine susceptibility [2].

Given the overlap in neurophysiological mechanisms, several non-pharmacological treatments have been explored for their potential to simultaneously target migraine and sleep disturbances. Among these, nerve stimulation techniques, particularly PENS, showed promise as complementary short-term interventions for reducing migraine frequency and intensity while improving sleep quality. Although the exact mechanisms behind PENS-induced analgesia remain unclear, it is hypothesized that electrical stimulation may modulate neural activity and increase levels of endogenous opioid-like substances within the central nervous system, contributing to its pain-relieving effects [33]. While nVNS has also shown potential, findings are more inconsistent, requiring further research to identify optimal protocols and understand its variability in efficacy [36, 38].

Research into GONB as a migraine treatment demonstrated consistent reductions in headache burden, improvements in pain intensity (measured via VAS scores), and enhancements in sleep quality. Across these studies, GONB was associated with a notable decrease in headache days and significant improvements in multiple aspects of sleep quality, reinforcing its value as a treatment option for migraine sufferers [43, 44, 46].

Sensory stimulation therapies, particularly auditory and visual, were also shown to benefit migraine patients [40, 41]. Lovati et al. [40] demonstrated a significant reduction in migraine frequency and pain intensity through binaural stimulation, with most patients reporting subjective improvements in their condition. Martin et al. [41] further demonstrated the effectiveness of green light exposure in reducing both headache frequency and intensity, particularly when compared to white light exposure. Additionally, both studies noted improvements in sleep quality, with participants reporting enhanced sleep onset and maintenance following sensory stimulation interventions [40, 41]. The mechanisms underlying these effects may involve modulation of the visual and auditory cortices, as well as their connectivity with brainstem structures involved in migraine pathophysiology and sleep regulation.

The evaluation of mind–body interventions, such as Tai Chi, MBSR+, and other relaxation techniques, also revealed their effectiveness in managing migraine and improving sleep quality [15–17]. Studies showed that these approaches contributed to reducing the frequency of headaches and enhancing sleep patterns, particularly for patients undergoing Tai Chi, who demonstrated significant improvements in sleep duration and overall quality compared to controls [15]. Furthermore, the combination of eye movement exercises and diaphragmatic breathing with jogging was shown to reduce headache frequency and pain intensity, with sustained improvements in sleep quality [17]. These findings support the role of stress reduction techniques in managing both migraine and sleep quality, potentially by decreasing sympathetic nervous system activation and promoting parasympathetic dominance.

Massage therapies, such as CTM and acupressure, were examined in three studies and demonstrated varying degrees of success in reducing migraine frequency and improving sleep quality [39, 45, 47]. Lawler et al.'s study [39] highlighted the potential of massage therapy to improve sleep and reduce migraine frequency, although it did not significantly affect pain intensity. Celenay et al. [45] reported that CTM significantly decreased both migraine frequency and pain intensity, though no notable difference was observed in sleep quality between groups. Similarly, acupressure resulted in significant reductions in migraine frequency and improvements in sleep quality, though these sleep-related changes were not statistically significant post-intervention [47].

TABLE 4 | Outcomes of non-pharmacological sleep treatments.

SLEEP non-pharmacological treatments	Effect on migraine frequency (f)	Effect on migraine intensity (i)	Effect on sleep quality (sq)
CBT-I	ns	ns	↑ sq
dCBT-I	↓ f (headache days per month)	↓ i	↑ sq

Note: ↓ f = significant decrease in migraine frequency; ↓ i = significant decrease in migraine intensity; ↑ sq. = significant improvement in sleep quality.
Abbreviation: ns, non-significant result.

In terms of dietary interventions, both the KD and omega fatty acid intake showed the potential to reduce migraine symptoms and enhance sleep quality [37, 42]. Patients on KD therapy experienced substantial reductions in migraine frequency, pain intensity, and sleep disturbances, reinforcing the diet's role in alleviating migraine symptoms and improving overall quality of life [42]. Similarly, both diets with high omega-3 and high omega-3 fatty acids combined with reduced omega-6 intake were shown to significantly reduce pain intensity and improve sleep, with participants in intervention groups reporting better overall sleep quality by the end of the studies [37]. The beneficial effects of dietary modifications may be linked to their ability to modulate inflammation, neurotransmitter balance, and energy metabolism, all of which play crucial roles in both migraine and sleep regulation.

Pharmacological interventions, including erenumab, amitriptyline, propranolol, and onabotulinumtoxinA, demonstrated effectiveness in reducing migraine frequency and intensity while improving sleep quality.

Both erenumab studies showed significant reductions in monthly migraine days and severity, with improvements in sleep quality reported by some patients [20, 21]. It is worth noting that Pilati et al. [21] observed a marked reduction in self-reported sleep efficiency among responders, despite no significant changes in global PSQI scores. While the PSQI mainly reflects overall perceived sleep quality, sleep efficiency specifically refers to sleep stability and continuity. This divergence suggests that anti-CGRP therapies may differentially influence distinct dimensions of sleep, an aspect that warrants further exploration. Of note, a more recent multicenter prospective study [51] also found that both oral prophylactic drugs and anti-CGRP monoclonal antibodies were associated with significant improvements in subjective sleep quality, with the strongest effects observed in the anti-CGRP group. Similarly, long-term treatment with onabotulinumtoxinA resulted in sustained reductions in headache days and improvements in sleep quality over 108 weeks [35].

Amitriptyline, a tricyclic antidepressant, showed significant improvements in migraine frequency, pain intensity, and sleep quality. Its sedative properties, primarily mediated by histaminergic and serotonergic pathways, may contribute to its beneficial effects on sleep in addition to its well-documented efficacy in migraine prophylaxis [22]. Conversely, propranolol, a beta-blocker frequently used for migraine prevention, paradoxically improved sleep quality despite beta-blockers being widely associated with sleep disturbances, such as fragmented sleep and nightmares [48]. This discrepancy suggests that propranolol's effects on sleep may vary depending on individual patient characteristics, compensatory physiological mechanisms, or the presence of migraine-related sleep alterations.

In terms of non-pharmacologic sleep interventions, both traditional and digital CBT-I (dCBT-I) have shown improvements in sleep quality in migraine patients [18, 19]. However, the study assessing traditional CBT-I did not achieve statistical significance for migraine symptom reduction compared to the control group [16]. This lack of significance may be attributed to the nature of the sham control intervention, which included structured lifestyle modifications such as consistent meal timing, controlled liquid intake, light stretching, and acupressure exercises. These elements, designed to mimic an active intervention, may have provided therapeutic benefits of their own, reducing the contrast between the CBT-I and control groups and potentially blunting the observable effect of CBT-I on migraine outcomes. Conversely, dCBT-I demonstrated a more pronounced impact, significantly reducing headache frequency and migraine pain intensity, alongside substantial improvements in sleep parameters [17]. The digital format may have contributed to higher adherence rates and accessibility, enhancing its efficacy. Given these findings, dCBT-I appears to be a promising option for addressing both insomnia symptoms and migraine burden, whereas further research is needed to refine and optimize the implementation of traditional CBT-I for migraine management. While behavioral interventions hold promise, it should also be considered that some pharmacological insomnia treatments, such as dual orexin receptor antagonists (DORAs), may induce headache as a common side effect [52, 53]. This highlights the complexity of targeting sleep in migraine patients.

An additional consideration is that most of the included studies were designed to primarily target either migraine or sleep, rather than both concurrently. This separation reflects current clinical practice, where migraine and sleep disorders are often managed in parallel but not always in an integrated fashion. Given the strong bidirectional links, combined interventions addressing both conditions simultaneously may provide greater benefits than targeting either alone. This integrated approach could be particularly relevant for patients with chronic migraine and comorbid insomnia, who may experience compounded improvements in both headache burden and sleep quality when therapies are applied synergistically. This concept is visually summarized in the graphical abstract (Figure S1), which integrates current evidence on migraine- and sleep-focused interventions and highlights the potential of dual-targeted strategies.

Overall, these findings align with broader literature exploring the bidirectional relationship between migraine and sleep, suggesting that treating one condition can improve outcomes for the other. This comprehensive review supports the potential of a dual-targeted approach, addressing both migraine and sleep disturbances to maximize treatment efficacy and patient outcomes. Future research should further explore these interconnections, focusing on developing personalized treatment strategies that address the overlapping mechanisms of these conditions.

5 | Conclusions

Both pharmacological and non-pharmacological treatments, including nerve stimulation, relaxation techniques, dietary interventions, and sensory therapies, were found to reduce migraine frequency and severity while improving sleep quality. Although the evidence for some treatments, such as nVNS and erenumab, remains mixed, the overall trend supports their use as part of a comprehensive migraine management strategy. Similarly, sleep-focused treatments, such as CBT-I, demonstrated the potential to reduce migraine frequency and improve sleep quality, particularly in patients with comorbid insomnia.

Despite its strengths, this review has several limitations. One notable limitation is the asymmetry in the available evidence: while numerous studies have explored migraine treatments and their impact on sleep, there is a relative scarcity of research on sleep-targeted pharmacological and non-pharmacological interventions and their effects on migraine. Although our search strategy and inclusion criteria allowed for the identification of such studies, we found a lack of published research meeting these criteria. This gap in the literature limits the ability to draw firm conclusions on the effectiveness of sleep-specific treatments in migraine management and highlights an important area for future investigation. Another limitation concerns the heterogeneity of sleep assessment across studies. While most trials relied on subjective instruments such as the PSQI or ISI, others reported parameters like total sleep time, sleep efficiency, or wake after sleep onset. These indices may not fully capture the multidimensional nature of "sleep quality," and discrepancies between subjective and objective measures further complicate the interpretation of results. In addition, the term "sleep quality" itself has been used broadly across the included studies and in our synthesis, encompassing both questionnaire-based measures and sleep continuity parameters. These different dimensions allowed to capture the multifaceted aspects of sleep, even if they may not always be directly comparable. In summary, the small number of studies, their methodological heterogeneity, the scarcity of physiological sleep data, and the variability in study designs, populations, and outcome measures represent further challenges in synthesizing the existing evidence.

Nonetheless, this review provides valuable insights into the interconnected nature of migraine and sleep disorders, emphasizing the need for integrated treatment strategies. Addressing both conditions concurrently could significantly improve patients' quality of life, reduce absenteeism, and enhance daily functioning. Healthcare providers may benefit from developing multidisciplinary treatment plans, incorporating personalized therapies and medication adjustments to optimize patient outcomes. These findings could also have public health implications, encouraging further research, awareness campaigns, and comprehensive care programs that recognize the dual burden of migraines and sleep disorders.

Future research should aim to expand the pool of studies, address current gaps in sleep-targeted migraine treatments, and explore the role of gender, study design, and treatment duration in shaping outcomes. Additionally, interdisciplinary research bridging neurology, sleep medicine, and psychology may lead to more effective therapeutic approaches, while the development of biomarkers could help identify individuals susceptible to sleep-related migraine triggers.

Author Contributions

M.S.: conceptualization; data curation; investigation; methodology; project administration; supervision; validation; visualization; writing – original draft; and writing – review and editing. I.M.: data curation; investigation; methodology; project administration; visualization; writing – review and editing. F.F.: data curation; investigation; methodology; project administration; visualization; writing – review and editing. F.G.: conceptualization; investigation; project administration; supervision; validation; writing – review and editing. A.S.: data curation; investigation; methodology; project administration; visualization; writing – review and editing. M.F.: project administration; supervision; writing – review and editing. P.P.: project administration; supervision; writing – review and editing. A.G.: project administration; supervision; writing – review and editing. R.M.: project administration; supervision; writing – review and editing. L.F.-S.: conceptualization; project administration; supervision; validation; writing – review and editing.

Ethics Statement

The authors have nothing to report.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data sharing not applicable to this article as no datasets were generated or analyzed during the current study.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section. **Figure S1:** ene70420-sup-0001-FigureS1.docx.