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ANALGESIC PHARMACOTHERAPY IN DYSMENORRHEA AND THE RISK OF DEVELOPING CHRONIC MIGRAINE

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ABSTRACT

Introduction and Purpose: Dysmenorrhea, particularly primary dysmenorrhea, is a common gynecological condition characterized by cyclical and often severe pain, typically managed with analgesics such as NSAIDs, acetaminophen, and opioids. Habitual, suboptimal, or excessive use of these medications may facilitate the transformation of episodic headaches into chronic forms through central sensitization and receptor-level changes in the central nervous system. Despite widespread pharmacotherapy, comprehensive synthesis regarding its role in chronic migraine development is lacking. This review systematically summarizes data from cohort, case-control, and interventional studies to evaluate the relationship between dysmenorrhea pharmacotherapy and chronic migraine risk. The study examines whether medication type, frequency of use, and patient-related factors—including age at dysmenorrhea onset, pain severity, comorbid psychiatric disorders, and use of migraine prophylactic agents—modulate this risk.
Results: Medication overuse headache (MOH) arises from excessive use of acute headache medications in patients with primary headache disorders, most commonly migraine. Opioids, butalbital-containing analgesics, acetaminophen–aspirin–caffeine combinations, and triptans confer the highest risk of chronification, while NSAIDs carry moderate risk and hormonal therapies may reduce pain frequency and intensity, potentially decreasing the need for frequent analgesic use. Frequent acute medication use (NSAIDs or acetaminophen ≥15 days/month, opioids/triptans ≥10 days/month), early dysmenorrhea onset, severe pain, and comorbid psychiatric conditions significantly increase MOH and chronic migraine risk. Implementation of prophylactic therapies and patient education can reduce reliance on acute medications and mitigate headache chronification.
Conclusion: Analgesic use in dysmenorrhea carries a risk of chronic migraine development, particularly with high-frequency or high-risk medications. Hormonal therapies, prophylactic strategies, individualized treatment, and monitoring of acute medication use are critical to minimize MOH and optimize long-term outcomes.

KEYWORDS
Dysmenorrhea, Chronic Migraine, Analgesics, Medication Overuse Headache, Central Sensitization, Chronifying

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1. Introduction

Dysmenorrhea, particularly primary dysmenorrhea, represents a common clinical condition characterized by periodic and often severe pain, typically managed with analgesics, including nonsteroidal anti-inflammatory drugs (NSAIDs), acetaminophen, and opioids. It has been hypothesized that habitual or suboptimal use of analgesic therapy, chronic administration, or overuse of these medications may promote the transformation of episodic headaches into a chronic form, mediated by mechanisms such as central sensitization and receptor-level alterations within the central nervous system. Currently, a comprehensive synthesis of the evidence examining the relationship between pharmacological management of dysmenorrhea and the risk of developing chronic migraine is lacking. The aim of this review is to systematically collect and summarize data from cohort, case-control, and interventional studies addressing this association. The primary objective of the present study is to evaluate the relationship between pharmacotherapy used in the management of dysmenorrhea and the risk of developing chronic migraine in women. Considering the potential mechanisms linking menstrual pain management and headache chronification, this review aims to identify pharmacological factors that may contribute to or mitigate this risk. Specifically, the study seeks to determine whether the type of medication used for dysmenorrhea (nonsteroidal anti-inflammatory drugs, opioids, acetaminophen, or hormonal therapies) differentially influences the likelihood of chronic migraine development. Furthermore, it explores whether the frequency of medication use—particularly regular intake of analgesics on ≥10 days per month—affects this risk. Additional analyses address whether clinical and psychosocial factors, including the age of dysmenorrhea onset, pain intensity, comorbid psychiatric conditions, and the use of migraine prophylactic agents, moderate the association between analgesic therapy and migraine chronification. Finally, the review examines the impact of specific pharmacological classes, such as NSAIDs, opioids, combination analgesics, and triptans, on the risk of developing medication overuse headache (MOH) and on the "chronifying" process of menstrual-related headache disorders.

2. Methodology

This systematic review was conducted in accordance with the PRISMA guidelines. Eligible studies included prospective and retrospective cohort studies, case-control studies, and cross-sectional analyses providing data on the relationship between pharmacotherapy for dysmenorrhea and the risk of developing chronic migraine or medication overuse headache (MOH). Randomized controlled trials (RCTs) were also considered if they reported long-term outcomes related to the occurrence or transformation of migraine.

The study population comprised women of reproductive age (12–50 years) diagnosed with primary or secondary dysmenorrhea, based on diagnostic criteria defined by the original authors. The exposure of interest included pharmacological treatment for menstrual pain—specifically nonsteroidal anti-inflammatory drugs (NSAIDs), acetaminophen, short-acting opioids, hormonal therapies (e.g., combined oral contraceptives or progestin-releasing intrauterine systems), and other analgesic agents. Both short-term and chronic treatment regimens, as well as medication overuse patterns (MOH), were evaluated.

Comparators included women not exposed to analgesic pharmacotherapy, those receiving different classes of medications, or subgroups defined by varying frequency or intensity of medication use (e.g., regular use ≥10 days per month versus occasional use). The primary outcome was the development of chronic migraine, defined according to the International Classification of Headache Disorders (ICHD) criteria—namely, ≥15 headache days per month for at least three months, with migraine features in a portion of attacks. Secondary outcomes included time to onset of chronic migraine and risk indices such as odds ratios (OR), relative risks (RR), and hazard ratios (HR).

The literature search encompassed PubMed/MEDLINE, Embase, Cochrane CENTRAL, Web of Science, Scopus, ClinicalTrials.gov, and EPOC databases, with additional screening of grey literature, including conference proceedings and academic theses when relevant. Reference lists of included studies and recent reviews were also examined to identify additional eligible publications. No date or language restrictions were applied, and publication year was recorded as a potential moderating variable in the analysis.

Studies were excluded if they involved animal models, investigated non-dysmenorrhea-related pain (e.g., postoperative pain), focused exclusively on pharmacokinetic or mechanistic outcomes without clinical headache data, or lacked accessible full-text data despite author contact attempts. Case reports and case series without control groups were excluded from the quantitative synthesis but considered for contextual qualitative discussion.

3. Results

3.1 Medication Overuse Headache (MOH)

Definition and Classification

Medication overuse headache (MOH) is a secondary form of headache resulting from the excessive use of acute headache medications in individuals with a pre-existing primary headache disorder, most commonly migraine or tension-type headache. In other words, a patient suffering from migraine or tension-type headache who regularly uses analgesic or antimigraine medications may develop a paradoxical condition in which these drugs themselves perpetuate or exacerbate headache symptoms. (Carlsen et al., 2020)

Epidemiology and Clinical Significance

Medication overuse headache (MOH) commonly occurs in individuals who experience frequent headache episodes, typically 15 or more days per month. The condition is associated with a substantial clinical burden, including reduced quality of life, increased disability, and significant consumption of acute medications. Despite being a fully treatable disorder, MOH often remains underdiagnosed. Diagnosis relies primarily on a thorough clinical history—specifically, the pattern of acute medication use, frequency of intake, number of headache days per month, and the exclusion of alternative causes of headache through neurological examination, which is typically unremarkable. According to the current International Classification of Headache Disorders (ICHD-3), specific diagnostic thresholds and criteria define the presence of MOH. (Carlsen et al., 2020)

Several neurobiological and psychosocial mechanisms play a crucial role in the pathogenesis of medication overuse headache (MOH). A key factor involves disturbances in pain modulation, particularly dysfunction of the descending antinociceptive pathways, resulting in a reduced ability to inhibit pain transmission and possible dysregulation of central control centers within the brainstem and spinal cord. Another important process is central sensitization, characterized by increased excitability of pain-transmitting neurons following repeated exposure to analgesic medications, leading to a lowered pain threshold and the perpetuation of chronic pain symptoms (Sebastianelli et al., 2023). The phenomenon of “chronifying” refers to the transformation of episodic headache disorders, such as migraine, into chronic forms—a hallmark feature of MOH (Perrotta et al., 2010). Additionally, behavioral and psychological factors contribute to MOH development, including a tendency toward self-medication, dependency-related mechanisms linked to excessive use of acute medications, as well as stress and anxiety disorders, which can modulate pain perception and promote the escalation of medication intake frequency (Hagen et al., 2012).

3.2 Drug Classes Involved and Their Association with Increased Risk of Chronic Migraine and Chronic Headache

Medication overuse headache (MOH) may be induced by various classes of drugs used for the acute treatment of headache disorders; however, the risk of its occurrence differs depending on the pharmacological agent. The highest likelihood of developing MOH has been observed with the use of opioids, combination analgesics containing butalbital, and acetaminophen–aspirin–caffeine combinations. Due to their mechanisms of action and addictive potential, these medications most frequently and rapidly contribute to the transformation of episodic headaches into chronic forms. Triptans represent another group associated with MOH, although the risk appears lower compared to opioids or barbiturate-containing preparations. A moderate yet clinically relevant risk is observed with prolonged and frequent use of nonsteroidal anti-inflammatory drugs (NSAIDs). The lowest potential for inducing MOH has been reported with calcitonin gene-related peptide (CGRP) receptor antagonists; however, current literature emphasizes the need for further long-term studies to fully assess their safety profile in this context (Fischer & Jan, 2025).

3.3 Types of Medications Used in Dysmenorrhea and Their Association with MOH

Nonsteroidal anti-inflammatory drugs (NSAIDs) are considered the first-line therapy for primary dysmenorrhea. Commonly used agents include ibuprofen, naproxen, diclofenac, and aspirin. Their mechanism of action involves the inhibition of prostaglandin synthesis within the endometrial lining and menstrual fluid, leading to reduced uterine contractility and pain relief (Furniss, 1982). Literature indicates that frequent use of NSAIDs may induce medication overuse headache (MOH), particularly when their use exceeds 15 days per month. In contrast, the use of triptans or opioids—commonly prescribed for migraine coexisting with dysmenorrhea—is associated with an increased risk of MOH when taken ≥10 days per month (Ashina et al., 2023). Although NSAIDs are effective, their long-term use is limited due to potential adverse effects, particularly gastrointestinal and renal complications. (Kirsch et al., 2024).

Paracetamol (acetaminophen) represents an alternative option, particularly for patients with contraindications to NSAIDs or those experiencing gastrointestinal intolerance (Kirsch et al., 2024). In a comparative study, ibuprofen demonstrated superior efficacy over acetaminophen in terms of pain relief and inhibition of prostaglandin F2α in menstrual fluid (Dawood & Khan-Dawood, 2007). Available data suggest that while acetaminophen offers a more favorable safety profile in patients for whom NSAIDs are unsuitable, it exhibits comparatively lower clinical efficacy (Zhang & Li Wan Po, 1998).

Among NSAIDs, mefenamic acid is frequently used and has been compared to naproxen and ibuprofen in several meta-analyses assessing efficacy (Nie et al., 2020). Some evidence indicates that hormonal contraceptive therapy may reduce the frequency and intensity of menstrual pain (De Sanctis et al., 2015). Hormonal methods—such as oral contraceptives, progestin-releasing intrauterine devices, or other hormonal options—achieve this effect by suppressing ovulation or decreasing uterine contractility (Kirsch et al., 2024). Current medical literature does not provide conclusive evidence that hormonal contraceptive methods, including combined oral contraceptives or levonorgestrel-releasing intrauterine systems (IUDs), increase the risk of developing MOH (Schroll et al., 2023).

Selective agents, such as cyclooxygenase-2 (COX-2) inhibitors, have shown comparable efficacy to traditional NSAIDs in smaller studies assessing menstrual pain relief. In some cases, combination therapies—such as acetaminophen with caffeine or diuretics—are employed to enhance analgesic effectiveness (Kirsch et al., 2024). Clinical practice often recommends initiating NSAID therapy prior to the onset of menstrual bleeding or cramping, or at the first appearance of pain, and continuing for several days to achieve optimal results (Osayande & Mehulic, 2014).

The frequency of acute medication use plays a critical role in the risk of developing chronic migraine in the context of medication overuse headache (MOH). Evidence indicates that regular intake of analgesics over a high number of days per month promotes the transformation of episodic headaches into a chronic form. For nonsteroidal anti-inflammatory drugs (NSAIDs) and acetaminophen, the risk of MOH increases when these medications are used for ≥15 days per month, whereas for triptans, ergotamines, and opioids, chronic headache transformation may occur with ≥10 days of use per month (Ashina et al., 2023).

Medical literature also highlights that the relationship between analgesic treatment for dysmenorrhea and the risk of developing chronic migraine or MOH is moderated by several factors. An earlier age of dysmenorrhea onset influences the duration of exposure to regular acute medication use, with earlier symptom onset associated with a higher likelihood of headache chronification. Studies have shown that women with a history of medication overuse headache were more likely to have used analgesics for more than three days per month to manage dysmenorrhea, which may contribute to the development of chronic migraine (G. Merki-Feld et al., 2024).

Comorbid psychiatric conditions, such as depression and anxiety, may amplify pain perception and increase the frequency of analgesic use, thereby further elevating the risk of developing chronic migraine and medication overuse headache (MOH). Studies indicate that patients with migraine and MOH frequently present with coexisting mood and anxiety disorders, which can exacerbate pain symptoms and promote overuse of medications. For instance, one study demonstrated that patients with chronic migraine and MOH exhibited higher rates of mood and anxiety disorders compared to patients without MOH, suggesting that these psychiatric conditions may constitute a risk factor for MOH development (Wang et al., 2021). Another study highlighted the association between obsessive-compulsive disorder and chronic migraine with MOH, suggesting that personality traits related to these conditions may predispose individuals to analgesic overuse and contribute to migraine chronification (Curone et al., 2012).

Comorbid psychiatric disorders may therefore play a significant role in MOH pathogenesis by increasing pain sensitivity, reducing treatment efficacy, and promoting a tendency toward medication overuse.

Consequently, these factors should be considered when assessing risk and planning treatment strategies in patients with migraine and dysmenorrhea. Implementation of prophylactic therapy, including both migraine-preventive medications and hormonal therapies to regulate dysmenorrhea, may reduce the need for frequent acute medication use and, in turn, lower the risk of MOH development (G. S. Merki-Feld et al., 2022).

4. Discussion

Findings from the literature suggest that the use of analgesics for dysmenorrhea may, under certain conditions, contribute to the development of chronic migraine through mechanisms characteristic of medication overuse headache (MOH). The frequency of acute medication use is a critical risk factor—regular intake of opioids, butalbital-containing preparations, or combination analgesics for ≥10 days per month, and nonsteroidal anti-inflammatory drugs (NSAIDs) for ≥15 days per month, promotes central sensitization and dysfunction of descending pain inhibitory pathways, leading to a lowered pain threshold and consolidation of chronic migraine (Ashina et al., 2023). Neurobiological mechanisms involve both impaired pain modulation and central sensitization, resulting in increased nervous system responsiveness to painful stimuli. The "chronifying" process, whereby episodic headaches transform into a chronic form, is closely associated with repeated acute medication use, mood disorders, anxiety, and personality traits that predispose to medication overuse (Perrotta et al., 2010).

The use of hormonal methods in the treatment of dysmenorrhea, such as oral contraceptives or progestin-releasing intrauterine devices, may reduce the intensity and frequency of menstrual pain, potentially decreasing the need for frequent analgesic use and thereby lowering the risk of MOH chronification (De Sanctis et al., 2015). However, there is a lack of prospective studies conclusively demonstrating that hormonal therapy reduces the risk of "chronifying" in the context of dysmenorrhea.

Additional factors, including earlier age at dysmenorrhea onset, severity of menstrual pain, comorbid psychiatric disorders, and the absence of prophylactic therapy, modulate the risk of developing chronic migraine. Patients experiencing severe dysmenorrhea in combination with depression or anxiety are at higher risk of analgesic overuse, which in turn increases the likelihood of developing medication overuse headache (MOH) (Ashina et al., 2023).

5. Conclusions

The use of analgesics in the management of dysmenorrhea, particularly opioids, butalbital-containing preparations, combination analgesics, and triptans, is associated with a risk of transforming episodic headaches into a chronic form, characteristic of medication overuse headache (MOH). This risk is significantly increased with frequent use of acute medications, especially ≥10–15 days per month, and in patients with severe menstrual pain, early onset of dysmenorrhea, or comorbid psychiatric disorders.

Hormonal therapies, such as oral contraceptives or progestin-releasing intrauterine devices, may reduce the intensity and frequency of menstrual pain, potentially decreasing the need for frequent analgesic use and thereby lowering the risk of "chronifying." Implementation of preventive strategies and monitoring of acute medication use are crucial for the prevention of chronic migraine development in women with dysmenorrhea.

Clinical recommendations emphasize the necessity of monitoring the frequency of acute medication use in patients with dysmenorrhea and implementing preventive strategies, including hormonal contraceptive therapy or migraine prophylactic pharmacotherapy. Individualized treatment planning and patient education regarding the risk of medication overuse headache (MOH) are essential components of chronic migraine prevention. Furthermore, clinical management should consider risk factors such as comorbid psychiatric disorders to minimize the potential adverse consequences of analgesic therapy and reduce the likelihood of chronic migraine development.

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