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Fasting as a Trigger of Migraine: Epidemiology, Pathophysiological Mechanisms, and Clinical Approach — a Narrative Review

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Abstract

Background. Migraine is a complex neurobiological disorder and one of the leading causes of disability worldwide, affecting approximately 14.1% of the global population. Among the numerous factors capable of precipitating migraine attacks, fasting occupies a particularly prominent position, yet the underlying mechanisms and clinical implications of this relationship remain incompletely understood.

Aim. To synthesize current evidence regarding fasting as a migraine trigger, with particular emphasis on its epidemiology, proposed pathophysiological mechanisms, and clinical approach.

Material and methods. A narrative literature review was conducted using PubMed and Google Scholar. Original articles, reviews, case series, and case reports were analyzed. The studies included both clinical human studies and experimental animal models.

Results. Fasting is reported as a migraine trigger by approximately 44% of patients, making it one of the most prevalent dietary precipitants. Multiple pathophysiological mechanisms have been proposed, including hypoglycemia-driven modulation of cortical spreading depression, astrocytic glycogen depletion, hypothalamic orexinergic and neuropeptide Y pathway dysregulation, and caffeine withdrawal. Clinical data further reveal persistent gaps between trigger recognition and behavioral modification among patients, while short-term preventive pharmacotherapy with rimegepant during predictable fasting periods has been examined in migraine populations.

Conclusions. Fasting represents a prevalent, modifiable, and mechanistically plausible migraine trigger acting through multiple converging pathways. Its systematic identification during clinical assessment, combined with individualized lifestyle and pharmacological strategies, may contribute to reducing attack burden in susceptible individuals. Further prospective research and randomized trials are needed to establish evidence-based recommendations.

Keywords: migraine; fasting; migraine triggers; dietary triggers; headache.

1. Introduction

Migraine is a complex neurobiological disorder influenced by genetic predisposition and characterized by recurrent episodes of moderate-to-severe headache, typically presenting as unilateral, throbbing pain. These attacks are frequently accompanied by nausea, vomiting, and increased sensitivity to light (photophobia) and sound (phonophobia) [1]. According to the Global Burden of Disease (GBD) 2023 Study [2], approximately 2.9 billion individuals were affected by headache disorders in 2023, with migraine exhibiting a global age-standardized prevalence of 14.1%. Beyond its high prevalence, migraine constitutes a major global public health challenge and remains one of the leading causes of disability worldwide, accounting for approximately 90% of headache-attributed years lived with disability (YLDs) [2]. This highlights its substantial contribution to the global burden of neurological disease.

Migraine attacks may occur spontaneously; however, a substantial proportion are preceded by identifiable trigger factors. Commonly reported triggers include dietary, hormonal, psychological, sleep-related, and environmental factors that may precipitate migraine attacks in susceptible individuals. Among dietary and metabolic triggers, fasting occupies a particularly prominent position. In a large study of migraine trigger factors, fasting was reported more frequently than alcohol and chocolate as a dietary precipitant of migraine attacks [3]. Despite the frequent association between fasting and migraine attacks, the relationship remains incompletely understood. The underlying neurobiological mechanisms, the influence of fasting duration and fasting regimens, and the implications for clinical management remain areas of ongoing investigation. This review aims to synthesize current evidence regarding fasting as a migraine trigger, with particular emphasis on epidemiological data, proposed pathophysiological mechanisms, and practical management considerations. By integrating findings from clinical and experimental studies, this article seeks to inform clinical practice and identify priorities for future research.

2. Material and methods

A narrative literature review was conducted using the PubMed and Google Scholar databases, with the search performed in June 2026. Search terms included combinations of "migraine," "headache," "fasting," and "dietary triggers," combined using the Boolean

operators "AND" and "OR" to ensure broad yet targeted literature retrieval. The search was not restricted by publication year to ensure inclusion of both seminal and contemporary literature. Only articles published in English were considered eligible for inclusion.

Articles were initially screened by title and abstract, followed by full-text assessment of potentially eligible records. Publications addressing the relationship between migraine or headache and fasting were eligible for inclusion, encompassing original research articles, reviews, case series, and case reports, and comprising both clinical studies in human migraine populations and experimental animal models. Articles were excluded if they were published as conference abstracts or non-peer-reviewed sources, or if they were not published in English. Additionally, a small number of publications were included to provide background information where relevant. Reference lists of retrieved articles were additionally screened to identify further relevant sources. Following this process, a total of 24 publications were selected for inclusion in the final review.

No formal systematic methodology was applied, consistent with the narrative design of this review; instead, study selection was guided by relevance, methodological quality, and contribution to the subject of this review. Evidence was synthesized thematically rather than chronologically, organized around three converging strands: epidemiological data, the underlying pathophysiological mechanisms, and clinical approach.

2.1. AI

Artificial intelligence tools were used only for editorial support during manuscript preparation. Their role was limited to linguistic refinement, structural polishing and assistance in reducing repetition. Study selection, interpretation of findings, synthesis of mechanisms and all final scientific conclusions remained under direct human control. AI was not used to generate data or references.

3. Results

3.1. Prevalence of fasting as a migraine trigger

Fasting is one of the most reproducibly identified dietary triggers in migraine epidemiology. A frequently cited systematic review reported fasting as a trigger in approximately 44% of migraine patients, making it one of the most commonly reported

precipitating factors, right after stress (58%) and auditory stimuli (56%) [4]. At the same time fasting has been recognized as the most consistently reported dietary trigger across studies — more common than alcohol, chocolate, or specific food items [5].

The most robust epidemiological evidence for fasting headache comes from prospective and cross-sectional studies conducted in the context of religious fasting, which provide a natural experimental model with clearly defined onset and offset of the fasting exposure. One study has demonstrated significantly increased headache frequency and severity during Ramadan fasting compared to non-fasting periods, with the effect being most pronounced in the late afternoon hours corresponding to the longest interval since the last meal [6]. These findings have been confirmed specifically in diagnosed migraine populations: one cohort study found patients with migraine had approximately threefold more migraine days during Ramadan fasting than during the control month [7]. Another study conducted among practicing Muslim migraine patients in Algeria similarly demonstrated a significant increase in monthly attack frequency, headache days, and analgesic intake during Ramadan compared to the preceding month [8]. Ramadan has not been the only religious practice studied for its relation to fasting-induced headache. Mosek and Korczyn [9] reported increased prevalence of headache (migraine and other types) during the Hebrew feast of Yom Kippur, where Jewish people fast for 25 hours. Among those who fasted completely, 39% reported headaches, compared to just 7% in the control group.

3.2. Clinical characteristics of fasting-triggered migraine

Fasting-triggered migraine attacks do not appear to differ substantially in semiology from attacks precipitated by other triggers. However, some observational studies suggest they may be associated with longer duration [7], although direct comparative data remain limited.

3.3. Pathophysiological Mechanisms

3.3.1. Hypoglycemia

The brain, consuming approximately 25% of total body glucose at rest, is particularly vulnerable to even moderate reductions in circulating glucose. Pearce [10] provided early experimental evidence for this relationship, demonstrating that insulin-induced hypoglycemia provoked migrainous symptoms in 2 of 20 susceptible individuals while

sparing matched controls, suggesting that acute cerebral glucose deprivation can directly initiate migraine pathophysiology.

One principal mechanism through which this may occur involves cortical spreading depression (CSD) — a slowly propagating wave of neuronal and glial depolarisation followed by sustained suppression of electrical activity, widely regarded as the neurophysiological substrate of migraine aura and a trigger of headache onset. Hoffmann et al. [11] demonstrated in an in vivo rat model that hypoglycaemia significantly prolongs CSD duration and delays ionic recovery, whereas elevated cerebral glucose raises the CSD threshold and confers relative resistance to its initiation. Martins-Oliveira et al. [12] further demonstrated in vivo that neuroendocrine signals associated with fasting — including fluctuations in insulin and glucagon — directly modulate nociceptive processing at the level of the trigeminocervical complex, a brainstem structure that serves as the principal relay for pain signals arising from intracranial structures and is considered a key site of central sensitisation in migraine.

3.3.2. Astrocytic glycogen depletion

Brain glycogen is stored predominantly in astrocytes and serves as an energy reserve that supports neuronal activity during periods of increased metabolic demand [13]. Under fasting conditions, astrocytic glycogen stores may become depleted, potentially impairing energy-dependent homeostatic processes [14]. Consequently, glycogen depletion has been proposed as a factor that lowers the threshold for CSD [14].

Support for this hypothesis was subsequently provided by Kılıç et al. [15] in a rodent model, using two complementary approaches that reduce astrocytic glycogen availability: pharmacological inhibition of glycogen breakdown with 1,4-dideoxy-1,4-imino-D-arabinitol, and a physiological manipulation (six hours of sleep deprivation) known to decrease brain glycogen levels. Both conditions lowered the threshold for CSD induction and activated downstream inflammatory signalling pathways.

3.3.3. Hypothalamic Orexinergic and Neuropeptide Y Pathways in Migraine

The hypothalamus plays a central role in the regulation of energy homeostasis and has been increasingly implicated in migraine pathophysiology. A recent review by Salinas-Abarca et al. [16] highlighted evidence suggesting that hypothalamic dysfunction may

contribute to migraine initiation through interactions between metabolic, autonomic, and nociceptive networks.

Among the hypothalamic systems activated during fasting, orexinergic and neuropeptide Y (NPY) pathways have received particular attention. Martins-Oliveira et al. [17] reviewed evidence indicating that caloric deprivation and hypoglycaemia increase orexinergic activity, while orexin signalling modulates trigeminovascular nociceptive processing in experimental models. The same review [17] also summarised findings showing that food deprivation upregulates hypothalamic NPY expression and that NPY can inhibit trigeminovascular neuronal firing through Y1 receptor-mediated mechanisms.

3.3.4. Caffeine Withdrawal

Caffeine withdrawal is another mechanism through which fasting may trigger migraine. For regular caffeine consumers, fasting often means missing their usual morning coffee, introducing a pharmacological trigger operating in parallel with the metabolic mechanisms discussed above. In a randomised, double-blind, crossover trial, Alstadhaug et al. [18] demonstrated that sudden caffeine withdrawal triggered severe migraine attacks in the majority of participants (7 of 9 evaluable subjects); notably, the trial was terminated prematurely due to low recruitment. Nowaczewska et al. [19] further concluded in their narrative review that while the overall relationship between caffeine and migraine is complex and the evidence insufficient to classify a single caffeine dose as a trigger, sudden cessation — particularly in the context of chronic overuse — consistently emerges as a migraine precipitant.

3.4. Clinical Approach

3.4.1. Trigger Identification

Despite the recognition of fasting as a trigger, knowledge and awareness gaps persist among patients. In the cross-sectional study by Ghimire et al. [20], a semi-structured headache questionnaire was successfully used to capture fasting as a trigger. Among the

headache types, migraine was the most prevalent. Surprisingly, merely 19% of individuals afflicted with headaches endeavored to mitigate their headache triggers, even though 66.8% of these individuals perceived lifestyle alterations as the best therapeutic intervention for headaches in contrast to pharmacological treatments. The majority of patients had knowledge of the etiological factors, precipitating elements, and alleviating variables, yet implementation remained limited. Among patients seeking help, 55% sought the expertise of a physician for the first time, whereas the remaining portion opted for assistance from a pharmacist or engaged in self-medication practices [20].

3.4.2. Lifestyle interventions

Scientific literature addressing migraine management recommends mode of living assessment as a central element of non-drug management strategies. The paramount interventions that may be endorsed for the prophylaxis of migraines encompass the removal of triggering factors, alterations in lifestyle habits, and the incorporation of consistent physical activity [21]. However, the authors of this article have not been able to identify a trial that specifically addressed lifestyle changes in patients suffering from fasting-triggered migraines.

3.4.3. Eating Behavior

Eating behavior in migraineurs extends beyond meal timing to include appetite dysregulation. A recent case-control study of 531 migraine patients and 1,062 healthy controls found that individuals suffering from migraines exhibited a diminished level of enjoyment associated with food intake and elevated scores in both hunger and satiety responsiveness, compared to controls, suggesting that modified dietary patterns are often documented among migraineurs, thereby exacerbating the overall burden associated with the condition [22].

3.4.4. Integration with pharmacological management

When fasting is identified as a predictable migraine trigger, pharmacological strategies can be adapted for short-term prevention, with emerging evidence supporting targeted preventive dosing during known fasting periods [23, 24].

A randomized open-label study evaluated the administration of a once-daily small-molecule calcitonin gene-related peptide (CGRP) receptor antagonist — rimegepant, taken at a dosage of 75 mg as an orally disintegrating tablet (ODT) for short-term prophylactic intervention for headaches induced by fasting during Ramadan in individuals aged 18 to 65 years who have been diagnosed with migraine and possessed a history of headache episodes attributed to fasting periods. In this trial, participants received rimegepant either throughout the entire four-week fasting period (Immediate Start group; n=52) or during the final three weeks only (Staggered Start group; n=53), with the primary outcome comparing the amount of headache days of any severity between the two groups during the first week. The results of this study showed a significantly lower incidence of migraine-type headaches during the first week in the first group of patients; furthermore, no side effects were reported [23].

An additional phase 4 clinical trial, which spanned a duration of 24 weeks and was conducted under open-label conditions, investigated the safety profile and tolerability associated with the extended administration of rimegepant at a daily dosage of 75 mg within adult populations for the purpose of prevention of episodic migraine, revealing favorable safety and acceptability characteristics when utilized at the specified dosage [24].

4. Discussion

This review synthesizes current evidence regarding fasting as a migraine trigger, highlighting its high prevalence, multifactorial pathophysiology, and implications for clinical management. Several key themes emerge from the available literature.

Fasting stands out as one of the most consistently identified dietary migraine triggers, reported by approximately 44% of patients — a figure that underscores its clinical relevance and places it ahead of more commonly publicized triggers such as alcohol and chocolate [4, 5]. The natural experimental model provided by religious fasting periods,

particularly Ramadan and Yom Kippur, has been instrumental in generating robust epidemiological data, demonstrating not only increased attack frequency but also a temporal relationship between fasting duration and headache onset [6, 7, 8, 9].

From a mechanistic standpoint, the pathophysiology of fasting-triggered migraine appears to be genuinely multifactorial. Hypoglycemia-driven modulation of cortical spreading depression threshold [10, 11, 12], astrocytic glycogen depletion [14, 15], dysregulation of hypothalamic orexinergic and NPY pathways [16, 17], and caffeine withdrawal [18, 19] represent distinct but potentially interacting mechanisms. Importantly, no single mechanism appears universally sufficient; rather, these pathways may converge and potentiate one another in susceptible individuals. This aligns with the broader threshold model of migraine, in which trigger burden — rather than any isolated factor — ultimately determines attack probability.

Despite this mechanistic plausibility, several gaps in the evidence base remain. Most available data derive from observational and cross-sectional studies, which limit causal inference. Prospective studies with objective dietary monitoring are scarce, and head-to-head comparisons of dietary interventions specifically targeting fasting-related attacks are largely absent.

On the clinical side, the observed discrepancy between patients recognizing fasting as a trigger and those actively modifying their behavior [20] points to an unmet need for structured, patient-centered education and behavioral support. Emerging pharmacological evidence, including the use of rimegepant as short-term preventive therapy during predictable fasting periods [23, 24], offers a promising avenue for patients in whom behavioral modification alone proves insufficient.

5. Conclusions

Fasting emerges from the available evidence as a clinically meaningful and mechanistically grounded migraine trigger, acting through multiple converging metabolic and neuroendocrine pathways rather than a single causative mechanism. Its modifiable nature makes it a practical target for both behavioral and pharmacological intervention, provided it is systematically identified during clinical assessment — a step that remains underutilized in routine practice. Structured dietary screening, individualized trigger management, and targeted preventive pharmacotherapy where

appropriate represent a coherent framework for reducing attack burden. Future prospective research and randomized trials are needed to translate these insights into standardized, evidence-based recommendations.

Disclosure

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Conflict of interest

The authors declare no conflict of interest.

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