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# Migraine as a Public Health Challenge: Contemporary Perspectives on Pathophysiology, Diagnosis and Treatment

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**Abstract**

**Introduction and purpose**: Migraine is a common and disabling neurological disorder with substantial public health relevance. It affects education, work productivity, social functioning, and quality of life, particularly among adolescents and adults of working age. The aim of this review is to summarize contemporary perspectives on migraine epidemiology, risk factors, pathophysiology, clinical classification, comorbidities, and current treatment strategies.

**Brief description of the state of knowledge**: Current evidence presents migraine as a complex disorder of sensory processing and neural network dysfunction rather than a purely vascular disease. Important mechanisms include activation of the trigeminovascular system, altered cortical and brainstem excitability, calcitonin gene-related peptide signaling, cortical spreading depression in migraine with aura, and peripheral and central sensitization. Diagnosis remains primarily clinical and should follow the International Classification of Headache Disorders criteria. Treatment includes acute pharmacotherapy, preventive drugs, anti-CGRP therapies, neuromodulation, education, lifestyle modification, regular physical activity, sleep regulation, and management of comorbid psychiatric, vestibular, sleep-related, and cardiovascular conditions.

**Summary**: Migraine should be understood both as a neurological disease and as a public health challenge. Effective care requires individualized, multidisciplinary management that combines evidence-based pharmacological treatment with non-pharmacological support and long-term patient education.

**Key words:** Migraine Disorders; Calcitonin Gene-Related Peptide; Headache; Public Health; Diagnosis; Therapeutics

## 1. Introduction and purpose

Migraine is a chronic neurological disorder characterized by recurrent attacks of headache accompanied by symptoms such as nausea, vomiting, photophobia, phonophobia, and sensory hypersensitivity. It is one of the leading causes of disability worldwide and remains a major public health problem despite the availability of diagnostic criteria and effective treatment options [1-3]. The disease is especially burdensome in adolescents and adults of working age, and its impact is particularly marked in women, who are affected substantially

more often than men [1,2,4]. For many patients, migraine is not an occasional inconvenience but a recurrent condition that shapes daily planning, social participation, academic performance, and professional functioning.

The modern view of migraine differs considerably from earlier vascular theories. Although vascular changes can accompany attacks, migraine is now understood primarily as a disorder of sensory processing and network dysfunction involving both peripheral and central mechanisms [5,6]. This conceptual shift has practical consequences. It helps explain why migraine includes aura, allodynia, fatigue, cognitive slowing, neck discomfort, vertigo, and mood change in addition to head pain. It also explains why recent mechanism-based therapies have achieved better outcomes than older, nonspecific approaches [7-10].

Another reason migraine remains a major subject of clinical interest is that it is still underdiagnosed and undertreated. Some patients self-manage recurrent attacks for years without formal diagnosis. Others receive only symptomatic medication even when attack frequency and disability justify preventive treatment [10,11]. Diagnostic delay is especially common in people with atypical symptoms, prominent vestibular complaints, or marked comorbidity. In practice, this means that many patients continue to live with avoidable disability even though contemporary medicine offers far more options than it did a decade ago.

Migraine also deserves attention because it stands at the intersection of neurology, internal medicine, psychiatry, rehabilitation, and public health. Its clinical expression is shaped by sex, genetics, stress, sleep, lifestyle, and coexisting disorders [4,7,12]. As a result, high-quality migraine care requires more than prescribing an acute pain reliever. It involves accurate classification, recognition of attack phases, assessment of disability, prevention of medication overuse, education on trigger management, and when necessary, implementation of long-term preventive therapy.

## 2. Description of the state of knowledge

### 2.1. Epidemiology and risk factors

Migraine affects a large proportion of the global population and remains one of the leading causes of years lived with disability worldwide [1,2]. Its burden is not limited to headache days. Patients often lose productive time at school or work, reduce social activity, postpone travel or exercise, and adapt their lives to the unpredictability of future attacks. The World

Health Organization continues to classify headache disorders among major public health concerns because of their prevalence, recurrent nature, and tendency to remain insufficiently treated [3].

The distribution of migraine across the population is not random. Sex is one of the strongest epidemiological determinants. Migraine is markedly more common in women, especially after puberty, and this difference is widely interpreted in the context of hormonal influences, particularly fluctuations in estrogen levels [4]. The clinical relevance of this observation is considerable. It affects counseling, timing of attacks, preventive planning, and interpretation of life-stage changes such as menstruation, pregnancy, and perimenopause.

Age is another important factor. Migraine often begins in adolescence or early adulthood, peaks during the years associated with study, family formation, and full professional activity, and may later decline or change in pattern [1,12]. This timing helps explain why the disorder produces a disproportionate socioeconomic burden. A recurring migraine attack in a student or young professional does not only mean pain for several hours; it may also mean reduced concentration, missed deadlines, absence from classes, disturbed sleep, and lower participation in exercise or leisure activities.

Genetic predisposition contributes substantially to susceptibility. Family aggregation has long been observed, and migraine with aura appears to show a particularly strong hereditary component [7,13]. In most patients the disorder is polygenic rather than monogenic, with multiple variants influencing ion transport, neurotransmission, synaptic regulation, and neuronal excitability [7]. These factors do not determine the disease alone, but they help establish a lower threshold at which hormonal, behavioral, or environmental factors can trigger an attack.

Triggers are a familiar and clinically useful part of migraine history taking. Commonly reported provoking factors include emotional stress, sleep deprivation, irregular sleep, fasting, dehydration, hormonal fluctuations, alcohol, and intense sensory stimulation [11,12,14]. However, trigger interpretation requires caution. Patients often identify patterns retrospectively, and not every apparent trigger has the same clinical relevance. In some cases, what seems to be a trigger may in fact be an early premonitory sign, such as food craving or fatigue. Even so, careful questioning and use of headache diaries remain valuable because they help patients recognize patterns and improve self-management.

Migraine frequently coexists with other disorders that amplify its burden. Depression, anxiety, sleep problems, vestibular symptoms, chronic pain states, and cardiovascular concerns are all common in migraine populations [4,12]. These comorbidities matter clinically because they may influence severity, response to treatment, adherence, and quality of life. A patient with migraine and insomnia, for example, has a different clinical profile from a patient with migraine alone. The same is true for those with depressive symptoms, heightened stress reactivity, or prominent dizziness.

Lifestyle-related factors are also relevant. Irregular meals, low physical activity, poor stress regulation, and unstable daily routine may all contribute to attack burden in predisposed individuals [11,12]. This is one reason why contemporary migraine care increasingly incorporates counseling on sleep hygiene, hydration, physical activity, and behavioral regulation. In this sense, epidemiology does not only describe who gets migraine; it also points toward modifiable factors that can become part of treatment.

### 2.2. Pathophysiology

The contemporary pathophysiology of migraine is built around the idea that migraine is a disorder of sensory processing involving altered excitability of neural systems, trigeminovascular activation, neuropeptide release, and dysregulation of pain modulation [5,6,15]. The older view that vascular change alone explained migraine is no longer sufficient. Vascular mechanisms remain relevant, but mainly as part of a broader interaction between nerves, meninges, brainstem structures, thalamocortical pathways, and inflammatory mediators.

A central structure in migraine biology is the trigeminovascular system. Trigeminal sensory fibers innervate the meninges and cerebral vessels and project to the trigeminal nucleus caudalis and higher-order pain-processing centers. Activation of this system leads to nociceptive transmission and release of signaling molecules, among which CGRP has emerged as especially important [8,16]. CGRP promotes vasodilation, enhances nociceptive signaling, and contributes to neurogenic inflammation and persistence of migraine pain [8,16,17]. The clinical relevance of CGRP is underscored by the success of therapies designed to block it or its receptor.

The trigeminovascular model does not operate in isolation. Brainstem and diencephalic structures also participate in attack generation and propagation. Functional studies have implicated regions involved in arousal, sensory modulation, and autonomic regulation, while

thalamic pathways help explain the integration of pain with light, sound, and touch sensitivity [6,18]. Noseda and colleagues demonstrated cortical projections of trigeminovascular thalamic neurons, providing a mechanistic basis for common associated symptoms such as photophobia and phonophobia [18]. This helps clarify why migraine is accompanied by such a wide sensory syndrome rather than by pain alone.

Migraine with aura introduces another key mechanism: cortical spreading depression. This phenomenon consists of a slowly propagating wave of neuronal and glial depolarization followed by suppression of activity, most often in the occipital cortex [19,20]. It is regarded as the most plausible biological substrate of visual aura and can also help explain sensory and language symptoms in some patients. Importantly, cortical spreading depression may secondarily activate trigeminal nociceptive pathways, linking transient cortical dysfunction with later headache [19]. In this way, aura is not a separate event unrelated to pain, but part of the broader pathophysiological cascade in susceptible individuals.

Current literature also suggests that migraine with aura and migraine without aura may share major mechanisms while still retaining meaningful biological differences [21]. Clinically, this distinction matters because aura has implications for diagnosis, differential diagnosis, vascular risk assessment, and patient counseling. From a mechanistic perspective, the debate reflects a broader question in migraine research: whether migraine should be viewed as one disorder with several clinical manifestations or a family of overlapping but partially distinct conditions [21].

Sensitization is another crucial concept. During an attack, nociceptive neurons may become increasingly responsive, first peripherally and later centrally. Peripheral sensitization contributes to throbbing pain and aggravation with movement, whereas central sensitization is associated with cutaneous allodynia, persistence of pain, and reduced responsiveness to delayed treatment [6]. This is one reason early administration of acute medication often works better than late treatment taken after pain has become widespread and amplified.

The premonitory and postdromal phases of migraine also support the view that migraine is a dynamic brain-state disorder rather than an isolated episode of pain. Symptoms such as yawning, fatigue, mood change, neck stiffness, food craving, and cognitive slowing can occur before or after the headache itself [22,23]. Their presence suggests that systems regulating motivation, autonomic balance, homeostasis, and sensory gating are involved throughout the attack cycle.

In summary, current pathophysiological models present migraine as a network disorder in which genetically and biologically predisposed brains respond abnormally to internal or external challenges. The result is not just headache, but a coordinated syndrome involving pain, altered perception, autonomic symptoms, and temporary dysfunction across several domains.

### 2.3. Clinical presentation and classification

The diagnosis of migraine is primarily clinical and should be based on a careful history rather than on routine imaging. The standard framework is the International Classification of Headache Disorders, 3rd edition, which defines migraine subtypes and provides criteria used in both research and practice [24]. A structured history should include attack duration, location and character of pain, associated symptoms, prodromal features, aura, trigger patterns, frequency, medication use, and the degree to which attacks interfere with daily life [10,25,26].

Migraine without aura is the most common subtype. It typically presents with recurrent attacks lasting 4 to 72 hours, moderate or severe intensity, pulsating quality, aggravation by routine activity, and associated symptoms such as nausea, photophobia, or phonophobia [10,24,25]. In clinical practice, however, presentations are not always textbook. Some patients describe bilateral pain, pressure rather than pulsation, or dominant symptoms such as nausea, fatigue, or sensory sensitivity. This variation is a common source of underrecognition, particularly outside specialist care.

Migraine with aura is defined by transient focal neurological symptoms that usually precede the headache phase but may also accompany it [20,24]. Visual aura is the most frequent form and may include flashing lights, zig-zag lines, blind spots, shimmering areas, or transient distortion of part of the visual field [19,20]. Sensory aura and language disturbances are less common but clinically important. The gradual evolution and complete reversibility of typical aura help distinguish it from sudden vascular events, although caution is always needed when symptoms are atypical, prolonged, or first occurring in later life.

One of the most helpful ways to understand migraine clinically is to view it as a sequence of phases. Many patients experience a premonitory phase characterized by irritability, fatigue, neck discomfort, food craving, concentration difficulties, or yawning [22]. This is followed by the headache phase and, in a large proportion of patients, by a postdromal phase marked by tiredness, “brain fog,” mood change, or a sense of being “drained” [23]. Recognition of

these phases is useful because it improves patient education and may support more timely treatment decisions.

Migraine can also include vestibular and auditory manifestations. Dizziness, motion sensitivity, imbalance, tinnitus, and other sensory symptoms are increasingly recognized as part of the migraine spectrum [27]. Their presence may complicate diagnosis but should not automatically lead the clinician away from migraine, particularly when the overall temporal pattern is suggestive.

The differential diagnosis of migraine includes tension-type headache, cluster headache and other trigeminal autonomic cephalalgias, medication-overuse headache, and secondary causes such as intracranial pathology, infection, vascular disorders, or systemic disease [25]. Neuroimaging is not indicated in every patient with recurrent migraine-like headache. It is generally reserved for red flags such as new neurological deficits, sudden change in pattern, abnormal examination, late onset, systemic symptoms, or suspicion of secondary headache [10,25].

Chronic migraine represents the most disabling end of the spectrum and is associated with substantial functional limitation. In these patients, headache occurs on many days of the month and often coexists with medication overuse, sleep problems, mood symptoms, and reduced quality of life [10,24,28]. Early recognition of progression toward chronicity is therefore an important objective of clinical care.

### 2.4. Management and treatment strategies

Migraine treatment has advanced from relatively nonspecific symptomatic care to a more individualized and mechanism-based model. Modern management includes acute treatment, preventive treatment, education, identification of aggravating factors, and non-pharmacological support [10,11,26]. The choice of therapy should depend on attack frequency, severity, disability, comorbidity, previous treatment response, patient preference, and the risk of medication overuse.

#### 2.4.1. Acute treatment

The goal of acute treatment is to stop or substantially reduce an ongoing attack while restoring function as early as possible. For mild or moderate attacks, NSAIDs and simple analgesics remain common first-line options and have evidence of efficacy in episodic migraine [14,29]. Timing matters. Medication taken early in the course of the attack is often more effective than medication taken after central sensitization has developed.

For moderate to severe attacks or for attacks that do not respond to simple analgesics, triptans remain a key part of acute treatment [9,30]. They act through serotonin receptors and reduce trigeminal signaling and neuropeptide release. Despite their long-established role, triptans are not appropriate for all patients because of contraindications, incomplete response, or adverse effects.

A major change in recent years has been the introduction of newer acute therapies such as gepants and ditans [9,31]. These drugs are especially relevant for patients who do not tolerate triptans, have insufficient benefit, or need alternatives because of cardiovascular concerns. Their emergence reflects a broader therapeutic shift: modern migraine treatment increasingly targets pathways known to be directly involved in the disease rather than relying only on repurposed agents.

### 2.4.2. Preventive treatment

Preventive treatment should be considered when attacks are frequent, prolonged, disabling, or poorly controlled with acute medication, and also when the pattern suggests a risk of chronification or medication overuse [10,11]. Traditional preventive agents include beta-blockers, certain antiepileptic drugs, and selected antidepressants. These medications remain useful, especially when a patient has comorbid conditions that make one option more attractive than another.

The most important contemporary breakthrough in prevention has been the development of monoclonal antibodies targeting CGRP or its receptor [8,17,31,32]. These therapies emerged directly from pathophysiological research and have shown clinically meaningful efficacy with generally good tolerability in many patients with episodic and chronic migraine. Their introduction changed expectations in migraine care. For the first time, clinicians had preventive drugs designed specifically around migraine biology rather than borrowed from other areas of medicine.

Recent reviews continue to support the role of anti-CGRP antibodies as a major component of modern preventive treatment [32]. At the same time, other pharmacological innovations remain under investigation, including small molecules and selected plant-derived compounds with possible anti-migraine action [33]. Although these newer approaches require further validation, they illustrate the ongoing diversification of migraine therapeutics.

Polish review literature similarly emphasizes that modern migraine management has entered an era in which targeted pharmacotherapy and individualized preventive planning have

become central to care [34]. This practical perspective is important because real-world treatment decisions often involve balancing efficacy, tolerability, cost, accessibility, and comorbidity rather than simply choosing the newest drug.

### 2.4.3. Neuromodulation

Neuromodulation has become an increasingly relevant option for selected patients, especially those who prefer non-drug treatment, have contraindications to medication, or require adjunctive strategies [35-38]. Non-invasive vagus nerve stimulation has shown benefit in episodic migraine prevention in controlled settings [35]. More broadly, the International Headache Society has published evidence-based guidance on non-invasive neuromodulation devices for acute and preventive treatment, reflecting the maturation of this field [36].

Transcranial direct current stimulation has also been evaluated in randomized trials and meta-analytic work, with promising though still heterogeneous results [37]. For highly selected patients with refractory chronic migraine, more invasive approaches such as high-frequency spinal cord stimulation have been studied, but these remain specialized interventions rather than routine care [38].

Neuromodulation is unlikely to replace pharmacotherapy for most patients, but it may broaden options and improve personalization. This is particularly valuable in migraine, where interindividual variability in treatment response remains substantial.

### 2.4.4. Non-pharmacological management, physical activity, and lifestyle

Non-pharmacological treatment is an essential part of long-term migraine care. Education on attack phases, medication timing, hydration, sleep regularity, meal timing, and trigger awareness can reduce both anxiety and attack burden [10,11]. These interventions are especially valuable in people whose attacks are strongly linked to stress, irregular routine, or sleep disturbance.

Physical activity deserves particular attention. During an acute attack, movement often worsens pain; however, this should not be confused with the role of regular exercise between attacks. Evidence suggests that aerobic exercise can reduce migraine frequency and may compare favorably with other exercise modalities in some patients [39,40]. Exercise may improve sleep, stress tolerance, vascular function, and mood, all of which are clinically relevant in migraine.

From the perspective of sport and rehabilitation, structured physical activity can also help counter the behavioral cycle in which patients become less active because they fear triggering headache. Over time, inactivity may contribute to deconditioning, poorer stress resilience, weight gain, and reduced quality of life. For this reason, advice on exercise should be individualized rather than avoided. Patients may benefit from gradual progression, adequate hydration, realistic pacing, and choosing forms of activity that are well tolerated.

Physiotherapy can also be relevant, particularly when migraine coexists with neck pain, muscle tenderness, posture-related discomfort, or balance dysfunction [41]. These features do not mean that migraine is caused by cervical problems, but they can still contribute to disability and may respond to supportive intervention. Manual therapies have been reviewed as adjunctive approaches, though evidence quality varies and these methods should be integrated into broader treatment plans rather than presented as stand-alone cures [42].

Dietary interventions remain an area of active interest. Reviews suggest that selected dietary strategies may benefit some patients, but responses are individual and should be interpreted cautiously [43]. In practice, regular meals, hydration, and identification of personally relevant dietary triggers are often more useful than rigid elimination regimens.

Taken together, recent reviews suggest that non-pharmacological treatment is not an optional extra. It is part of comprehensive care and can meaningfully complement drug treatment, especially when tailored to the patient's triggers, disability profile, and daily routine [44].

### 2.5. Comorbidities and quality of life

Migraine is closely linked with comorbidity, and this relationship strongly shapes prognosis and daily functioning. Psychiatric comorbidities, especially depression and anxiety, are common and clinically important [4,12]. They may intensify the experience of pain, reduce adherence to treatment, and increase the likelihood that the patient withdraws from work, study, social participation, or physical activity. The association appears to be bidirectional: recurrent migraine can worsen mood and functioning, while mood disorders can increase stress sensitivity and headache-related disability.

Sleep disturbance is another frequent companion of migraine. Irregular sleep can act both as a trigger and as a consequence of repeated attacks. Sleep loss lowers tolerance to pain, worsens concentration, and increases fatigue the next day. In students and working adults, this can create a vicious circle in which migraine disrupts sleep, poor sleep increases attack risk, and both together worsen academic or professional performance.

Vestibular and auditory symptoms are particularly relevant because they may be misinterpreted as evidence for a separate disorder [27]. Dizziness, motion sensitivity, and balance problems often reduce confidence in everyday mobility and physical activity. Patients may begin avoiding public transport, exercise, or crowded sensory environments. This withdrawal itself reduces quality of life even on days without severe head pain.

The burden of migraine should therefore be assessed more broadly than by counting headache days alone. Many patients fear the unpredictability of the next attack, plan social events around migraine risk, and adjust work or study patterns to preserve functioning. The psychological cost of this uncertainty can be high even when the absolute number of attacks is not extreme.

Quality of life is especially poor in chronic migraine. Compared with episodic migraine, chronic migraine is associated with greater disability, more frequent comorbidity, higher medication use, and higher healthcare utilization [10,24,28,45]. Early intervention is therefore not only about reducing current pain; it is also about preventing progression toward a more entrenched and disabling state.

## 3. Discussion

The literature reviewed in this paper shows how decisively the understanding of migraine has changed. Migraine is no longer adequately explained as a vascular headache with a few associated symptoms. The current framework, centered on trigeminovascular activation, altered sensory processing, and CGRP-mediated signaling, better matches both the clinical complexity of migraine and the effectiveness of recent treatments [5,6,8,15,16].

One of the clearest successes of translational medicine in migraine has been the rise of anti-CGRP therapies. These treatments did not emerge from empirical trial-and-error alone. They were built on mechanistic insight, and their clinical efficacy in turn strengthened that mechanistic model [8,17,32]. Few examples in modern headache medicine demonstrate so clearly how basic science can shape everyday clinical practice.

At the same time, the reviewed evidence makes it clear that migraine cannot be reduced to a single peptide or pathway. Aura, premonitory symptoms, sensory hypersensitivity, central sensitization, chronification, and the role of comorbidity all point to a disorder of distributed neural regulation rather than a single malfunctioning node [6,15,19,21]. This complexity

explains why treatment response varies widely between individuals and why no single approach is sufficient for every patient.

Another important conclusion is that migraine management must remain multidisciplinary. Effective care does not end with selecting an acute medication. It also involves educating the patient, limiting medication overuse, treating comorbid depression or insomnia, identifying lifestyle patterns that increase vulnerability, and in some cases supporting the return to regular physical activity [10,11,26,39-41,44]. This broader view is especially relevant in young adults, whose headaches often interfere with study routines, exercise, and social development.

The exercise-related evidence deserves careful interpretation. Physical exertion during an attack can aggravate symptoms, which sometimes leads patients to avoid movement altogether. However, regular exercise between attacks may reduce migraine frequency and improve overall well-being [39,40]. This distinction is clinically important and should be communicated clearly. In practice, well-planned exercise can become part of prevention rather than a feared trigger.

The main limitation of this review is its narrative design. It does not provide pooled quantitative estimates or a formal risk-of-bias assessment. Nevertheless, it brings together major classification documents, mechanistic reviews, treatment reviews, and clinically relevant studies in a way that reflects current practice and current therapeutic thinking.

## 4. Summary

Migraine is a common and highly disabling neurological disorder with major social, educational, and occupational consequences. Contemporary evidence supports understanding migraine as a disorder of sensory processing involving the trigeminovascular system, altered neural excitability, CGRP signaling, cortical spreading depression, and sensitization. Diagnosis remains primarily clinical and should be guided by the ICHD-3 criteria, careful history taking, and awareness of atypical or overlapping presentations. Acute treatment includes NSAIDs, simple analgesics, triptans, and newer options such as gepants and ditans. Preventive treatment has advanced significantly with the introduction of monoclonal antibodies targeting CGRP or its receptor. Non-pharmacological care, including patient education, regular physical activity, physiotherapy, sleep regulation, and selected dietary interventions, can usefully complement drug treatment. Comorbidities strongly influence

prognosis and should be assessed routinely. Future progress in migraine care will depend on increasingly personalized treatment pathways and better integration of neurological, psychological, and rehabilitation-oriented approaches.

## DISCLOSURE

**Authors' contributions:**

Pola Kobryń - conceptualization, methodology, formal analysis, writing – review and editing, supervision

Igor Nowiński - resources, writing – original draft preparation

Dawid Ocicki - investigation, resources, formal analysis

Michał Bojarski - investigation, formal analysis, project administration

Michał Kwiatkowski - formal analysis, resources

Michał Musielak - formal analysis, resources

Iga Kobryń - investigation, data curation

Aleksandra Zybert - resources, data curation

Aleksandra Borowska - data curation, writing – review and editing

Jakub Grabowski - data curation, writing – original draft preparation

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