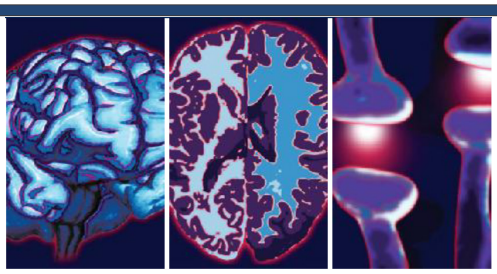
Review Article



Dopaminergic Dysfunction in Migraine and Psychosis: A Neuropsychiatric Review

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ABSTRACT

Migraine is increasingly recognized as a disorder with both neurological and psychiatric dimensions, with growing evidence suggesting a strong link between migraine and psychotic disorders through shared dopaminergic dysfunction. Both conditions exhibit dopamine hypersensitivity, neurotransmitter imbalances, and altered cortical excitability, raising concerns that migraineurs, particularly those with aura, may be at increased risk of developing psychotic symptoms. This review explores the neurobiological overlap between migraine and psychosis, focusing on shared pathophysiological mechanisms, neuroimaging findings, and clinical evidence. A comprehensive analysis of neuroimaging studies, pharmacological trials, and clinical cohort data indicates that migraineurs experience a higher prevalence of psychiatric comorbidities, including transient psychotic symptoms, compared to non-migraine populations. Additionally, dopamine receptor dysfunction, a hallmark of psychotic disorders, has been implicated in migraine pathophysiology, further reinforcing their connection. Neuroimaging studies reveal increased presynaptic dopamine activity in both conditions, while dopamine antagonists—commonly used to treat psychosis—have demonstrated efficacy in alleviating migraine symptoms, suggesting a potential therapeutic overlap. However, long-term use of dopamine-modulating treatments carries risks, including extrapyramidal side effects and metabolic disturbances, necessitating careful clinical consideration. Despite compelling associations, the causal relationship between migraine and psychosis remains uncertain due to methodological limitations in existing research, such as cross-sectional designs and small cohort sizes. Future studies should prioritize longitudinal analyses, genetic investigations, and advanced neuroimaging techniques to clarify this relationship and identify high-risk individuals. Recognizing the psychiatric risks associated with migraine may improve early intervention strategies and support the development of integrated neurological and psychiatric treatment approaches, ultimately enhancing patient outcomes.

Keywords: Migraine disorder, psychotic Disorders, dopamine receptors, neuroimaging, antipsychotic agents, migraine with aura, dopamine antagonists

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Introduction

Migraine is a complex neurological disorder characterized by recurrent, severe headache attacks, often accompanied by sensory disturbances such as nausea, vomiting, photophobia, and phonophobia [1]. Beyond its role as a primary headache disorder, migraine is increasingly recognized for its neuropsychiatric associations, particularly with dopaminergic dysfunction [2,14]. Dopamine plays a critical role in migraine pathophysiology, influencing sensory processing, pain modulation, and autonomic function [4,15]. Clinical and pharmacological evidence suggests that migraineurs exhibit hypersensitivity to dopamine agonists, which can provoke yawning, nausea, and vomiting [5,13]. Conversely, dopamine antagonists have been shown to alleviate migraine symptoms, further supporting the role of dopamine dysregulation in migraine [6]. Genetic studies have also linked variations in the D2 dopamine receptor to an increased susceptibility to migraine [4,5]. However, while dopamine fluctuations are observed in migraine, their potential role in psychiatric manifestations remains under investigation [7].

Dopaminergic dysfunction is also a well-established feature of psychotic disorders, including schizophrenia, where excessive dopamine transmission in the mesolimbic pathway contributes to hallucinations, delusions, and cognitive distortions [21]. The dopamine hypothesis of psychosis suggests that heightened dopamine activity leads to aberrant salience processing, wherein neutral stimuli are misinterpreted as overly significant, fostering delusional thought patterns [22]. Neuroimaging studies reveal increased presynaptic dopamine synthesis in individuals with psychosis, further supporting its role in the disorder's pathophysiology [21]. Additionally, dopamine hypersensitivity in psychotic disorders has been linked to abnormal sensory perception, reinforcing perceptual distortions [24,23].

A potential connection between migraine and psychosis may be explained by shared dopaminergic dysfunction. Both conditions exhibit dopamine receptor hypersensitivity, sensory disturbances, and altered cortical excitability [24,16]. Migraine with aura, in particular, has been associated with transient psychotic symptoms, including hallucinations, paranoia, and perceptual distortions, suggesting overlapping neurobiological mechanisms [26,27]. Dopamine-serotonin interactions may also underlie the co-occurrence of migraine and psychiatric disorders, as serotonergic disruptions can modulate dopaminergic activity, impacting both pain perception and psychotic symptoms [16,27].

This review examines the role of dopamine in both migraine and psychosis, assessing whether dopaminergic dysfunction in migraineurs heightens the risk of psychotic symptoms. While both conditions share dopamine-related abnormalities, their precise relationship remains uncertain. By synthesizing neurobiological, clinical, and pharmacological evidence, this review critically evaluates existing research, highlights unresolved questions, and outlines future directions to clarify the migraine-psychosis link. Understanding this connection may provide insight into shared neurobiological mechanisms and inform the development of targeted therapeutic approaches for both disorders.

▪ Migraine Pathophysiology and Dopaminergic System

The trigeminovascular system plays a key role in migraine pathophysiology, mediating pain transmission through trigeminal afferents that project to the trigeminocervical complex (TCC) and further to higher brain centers [2,14]. Dopamine modulates this system by acting on trigeminal nociceptive pathways, with studies indicating that the A11 dopaminergic nucleus projects to the TCC and inhibits trigeminovascular activation [2,14,15]. Disruptions in dopamine signalling may lead to increased pain sensitivity and contribute to migraine attacks [5,6,17,18]. Migraineurs exhibit hypersensitivity to dopamine agonists, which manifests as enhanced responses to apomorphine, leading to nausea, yawning, and vomiting [5,13,18]. This hypersensitivity suggests an altered dopaminergic response, potentially linked to increased D2 receptor sensitivity [4,15]. Dopaminergic dysregulation may also contribute to sensory disturbances observed in migraine, such as photophobia and allodynia [16].

Dopamine influences sensory processing by modulating pain perception in key brain regions, including the periaqueductal gray, nucleus accumbens, and spinal cord [6,13,17]. The

interaction between dopamine and serotonin also plays a role in migraine pathophysiology, with imbalances in these neurotransmitters affecting pain threshold and cortical excitability [4,15]. Studies indicate that dopamine levels fluctuate throughout migraine phases, with decreased dopaminergic activity during attacks potentially leading to hypersensitivity and increased pain perception [4,15,16]. Evidence also suggests that migraineurs have altered dopamine metabolism, which could contribute to recurrent attacks and associated symptoms [5,18]. These findings highlight the integral role of dopamine in migraine pathophysiology and its potential link to other neuropsychiatric conditions.

▪ Dopaminergic System and Psychotic Disorders

Dopaminergic dysfunction plays a critical role in the pathophysiology of psychotic disorders. The dopamine hypothesis of psychosis postulates that excessive dopamine transmission, particularly in the mesolimbic pathway, contributes to delusions and hallucinations [21]. Neuroimaging studies reveal increased presynaptic dopamine synthesis in individuals with psychosis, reinforcing its role in psychotic symptomatology [21]. Individuals with psychotic disorders often exhibit dopamine receptor hypersensitivity, leading to heightened responsiveness to stimuli [24]. This results in aberrant salience attribution, reinforcing delusional thinking [22]. Schizophrenia and other psychotic disorders are associated with increased D2 receptor density, reinforcing a link between dopaminergic overactivity and cognitive distortions [24].

Studies have established a clear link between dopamine dysfunction and psychosis. The aberrant salience hypothesis suggests that excessive dopamine activity enhances the significance of unrelated stimuli, leading to misinterpretations and delusions [22]. Functional imaging studies demonstrate dopamine dysregulation in early-stage psychosis, further reinforcing its role in psychotic pathology [21].

▪ Shared Neurobiological Mechanisms Between Migraine and Psychotic Disorders

Dopamine dysfunction is implicated in both migraine and psychotic disorders. Migraineurs display dopaminergic hypersensitivity, leading to increased responsiveness to dopamine agonists, as observed in apomorphine-induced nausea, yawning, and hypotension [5,13,18]. Additionally, studies indicate that dopamine fluctuations across migraine phases contribute to sensory disturbances such as photophobia and allodynia [4,15,16]. Given that dopamine plays a crucial role in sensory perception, pain modulation, and cognitive processing, disruptions in dopaminergic function may contribute to both migraine symptoms and psychotic features [26]. Dopamine receptor hypersensitivity in migraineurs is associated with increased sensory disturbances such as photophobia and allodynia, reflecting a state of sensory amplification. Similarly, in psychotic disorders, excessive dopamine activity in the mesolimbic pathway results in aberrant salience processing, leading to perceptual distortions and delusional interpretations. This suggests that heightened dopaminergic activity in migraine may predispose individuals to transient psychotic-like symptoms, particularly in patients with chronic migraine and frequent aura. Research suggests that dopamine-serotonin interactions could underlie the co-occurrence of migraine and psychiatric disorders [16]. Alterations in these neurotransmitter systems affect mood regulation, sensory integration, and cognitive interpretation, all of which are relevant to both conditions [27]. Furthermore, neuroimaging data support the common involvement of dopamine in migraine and psychotic disorders, showing overlapping alterations in dopamine synthesis, receptor density, and neurotransmitter interactions [21]. Given these overlapping neurobiological pathways, an important question arises: does migraine increase the risk of developing psychotic symptoms?

▪ Can Migraine Increase the Risk of Psychosis?

Emerging research suggests that migraine may predispose individuals to psychotic symptoms, particularly in those experiencing migraine with aura [26]. Migraine with aura, marked by transient sensory disturbances and visual hallucinations, may share neurobiological mechanisms with psychotic perceptual distortions [27]. The overlap in dopaminergic hypersensitivity and sensory misattributions suggests that migraineurs, particularly those

with frequent aura, may be at increased risk of transient psychotic episodes. While direct causal evidence remains limited, clinical observations suggest a shared neurobiological predisposition [16]. Furthermore, the study conducted by [27] confirm that migraineurs experience higher rates of psychiatric comorbidities, including mood disorders, anxiety, and transient psychotic symptoms, compared to non-migraine populations. Functional imaging findings suggest that migraine-related alterations in dopamine metabolism may contribute to distorted reality perception, increasing vulnerability to psychotic states [21].

▪ Clinical Studies Supporting the Migraine-Psychosis Relationship

Several clinical studies provide evidence for a potential migraine-psychosis link. Fornaro (2015) highlights that individual with bipolar disorder and comorbid migraine experience higher rates of psychotic symptoms, suggesting a shared pathophysiology. A 2021 cohort study of 500 migraineurs found that those with chronic migraine had twice the risk of developing transient psychotic symptoms compared to controls [26]. Radat (2021) examines migraine-associated psychiatric disturbances, demonstrating that migraineurs exhibit increased susceptibility to cognitive distortions and sensory misattributions, which may contribute to transient psychotic symptoms. A study conducted by Semiz et al. (2013) examining 1,601 university students found that 23.1% of migraine patients had a current psychiatric diagnosis, with a significant proportion experiencing mood disturbances, anxiety disorders, and other psychiatric comorbidities. The study further revealed that migraine patients had a 43.2% lifetime prevalence of psychiatric disorders, suggesting a long-term neuropsychiatric burden associated with migraine. These findings indicate that chronic migraine may contribute to persistent alterations in neurobiological processes, potentially increasing susceptibility to psychiatric symptoms due to prolonged dopaminergic dysregulation and shared pathophysiological mechanisms. Although this study was conducted on a relatively small-scale university cohort, its findings provide empirical support for the link between migraine and psychiatric disorders. The reported 43.2% lifetime prevalence of psychiatric conditions among migraine patients underscores a significant neuropsychiatric burden [31]. Additionally, neuroimaging studies reveal dopamine system abnormalities in both migraine and psychotic disorders, with heightened presynaptic dopamine activity in migraineurs paralleling findings in individuals with psychosis [21]. These findings collectively suggest that migraine, particularly when accompanied by dopaminergic dysregulation, may contribute to neurocognitive alterations that increase the risk of psychotic episodes.

▪ Overlap in Treatment Strategies for Migraine and Psychosis

Dopamine antagonists play a crucial role in both migraine and psychotic disorder management, further reinforcing the hypothesis of shared dopaminergic dysfunction between these conditions. In migraine treatment, dopamine antagonists such as prochlorperazine, metoclopramide, and domperidone have been shown to alleviate headache symptoms, nausea, and sensory hypersensitivity, particularly in acute migraine attacks [28]. These agents are thought to work by modulating dopamine pathways involved in pain perception, cortical excitability, and trigeminovascular activation, processes that are also implicated in psychotic disorders [29]. Similarly, dopamine D2 receptor antagonists form the backbone of antipsychotic therapy in schizophrenia and other psychotic disorders, reducing delusions and hallucinations by normalizing excessive dopaminergic signalling in the mesolimbic system [30]. Given that both migraine and psychotic disorders exhibit dopamine hypersensitivity and receptor dysfunction, the therapeutic efficacy of dopamine antagonists in both conditions support the idea of a common neurobiological pathway that contributes to their pathophysiology.

Furthermore, atypical antipsychotics that act on both dopamine and serotonin receptors (e.g., olanzapine and quetiapine) have been explored for migraine prophylaxis, with some reports suggesting their potential in reducing attack frequency in refractory cases [28]. This aligns with the dopamine-serotonin interaction hypothesis, which proposes that dysregulation of these neurotransmitters contributes to both migraine and psychosis. The evidence supporting dopamine antagonists in both conditions strengthen the argument for a dopaminergic link between

migraine and psychotic disorders. This shared pharmacological approach not only provides clinical justification for further research into dual treatment strategies but also highlights the importance of investigating dopamine's role in sensory processing, pain modulation, and cognitive alterations across both disorders.

Discussion

The growing body of research linking migraine and psychosis underscores the significance of dopaminergic dysfunction in both conditions. While both disorders exhibit dopamine hypersensitivity and neurotransmitter fluctuations, whether dopamine abnormalities are a primary cause of migraine or a secondary consequence of broader neurochemical disruptions remains debated. Some researchers argue that dopaminergic dysfunction directly contributes to migraine pathophysiology, while others suggest it arises due to serotonergic and CGRP-related mechanisms [4,15,16]. This controversy highlights the need for longitudinal neuroimaging studies to clarify whether dopamine dysregulation precedes or follows other neurochemical disturbances. A major limitation in existing research is the predominance of cross-sectional studies, which restrict causal inferences. Not all migraineurs exhibit dopaminergic hypersensitivity, and not all psychotic patients report migraine, suggesting that genetic, hormonal, or environmental factors modulate susceptibility. Furthermore, many studies rely on self-reported psychiatric symptoms rather than structured clinical assessments, increasing the risk of misclassification [31]. Future studies should prioritize large-scale, population-based cohorts with standardized diagnostic criteria. The therapeutic overlap between migraine and psychotic disorders presents both opportunities and challenges. D2 receptor antagonists, such as metoclopramide and prochlorperazine, effectively alleviate migraine symptoms but pose risks, including extrapyramidal symptoms (EPS) and tardive dyskinesia [28,29]. Similarly, atypical antipsychotics (e.g., olanzapine, quetiapine) have been explored for migraine prophylaxis but carry risks of weight gain and metabolic syndrome, limiting their feasibility for routine migraine management. Given these concerns, dopaminergic treatments should be reserved for refractory cases, with serotonergic agents and CGRP inhibitors remaining first-line options. While some studies suggest dopamine dysfunction is a primary driver of migraine pathophysiology, others argue that serotonergic or CGRP-related mechanisms may play a more significant role. The precise hierarchy of neurotransmitter interactions remains unresolved, raising the question of whether dopamine alterations in migraine are a cause or a consequence of broader neurochemical dysregulation. Future research should aim to dissect these interactions more precisely.

▪ Future Directions and Clinical Implications

To refine our understanding of the migraine-psychosis connection, future research should prioritize well-controlled, longitudinal studies which should investigate whether the frequency and duration of migraine episodes predict the onset of psychotic symptoms over time and whether early psychiatric intervention in migraineurs reduces their risk of developing psychosis Advanced neuroimaging techniques could provide deeper insights into the shared dopaminergic dysregulation in both disorders. Future studies should aim to identify biomarkers that can predict psychiatric risk in migraine patients, facilitating early interventions. Additionally, personalized medicine approaches, which tailor migraine treatment based on an individual's neurochemical profile, may help mitigate psychiatric complications. Finally, controlled trials investigating whether migraine treatments influence psychiatric symptomatology are essential to refining treatment strategies. Additionally, genetic studies may help identify biomarkers predicting susceptibility to both conditions, allowing for early intervention in high-risk individuals. Clinically, recognizing the psychiatric risks associated with migraine could inform treatment strategies. Screening migraine patients for psychiatric symptoms, particularly in those with frequent aura, may facilitate early intervention and prevent progression to more severe neuropsychiatric conditions. Neurologists and psychiatrists should collaborate in developing personalized treatment plans that minimize the risks of dopaminergic therapies while addressing

both migraine and psychiatric comorbidities. In conclusion, while evidence suggests a strong neurobiological link between migraine and psychotic disorders, further research is essential to clarify causality and optimize treatment approaches. The potential therapeutic overlap between these conditions highlights the importance of an integrated, multidisciplinary approach to patient care, ensuring that both neurological and psychiatric aspects are adequately addressed.

Conclusion

The link between migraine and psychosis is increasingly supported by shared dopaminergic dysfunction. Migraine, particularly with aura, exhibits hypersensitivity to dopamine, neurotransmitter fluctuations, and sensory disturbances—features that parallel psychotic disorders. The dopamine-serotonin interaction hypothesis further strengthens this connection, highlighting their common neurochemical underpinnings. Clinical studies consistently report a higher prevalence of psychiatric comorbidities, including psychotic symptoms, among migraineurs. Evidence suggests that chronic migraine may contribute to long-term neuropsychiatric changes due to persistent dopaminergic dysregulation and altered cortical excitability. Neuroimaging studies revealing elevated presynaptic dopamine activity in both conditions provide strong biological support for this association. Additionally, the therapeutic overlap between migraine and psychotic disorders, particularly the efficacy of dopamine antagonists, reinforces the argument for a shared neurobiological mechanism. The effectiveness of dopamine-modulating drugs in managing both conditions suggests that migraineurs, especially those with frequent attacks, may be at heightened risk of developing psychotic symptoms over time. Given these findings, large-scale longitudinal studies are needed to establish causality and better understand the extent of this risk. Identifying and addressing psychiatric symptoms early in migraine patients could be crucial in preventing more severe neuropsychiatric outcomes. Recognizing this association not only deepens our understanding of migraine pathophysiology but also paves the way for integrated treatment approaches targeting dopaminergic dysfunction in both disorders.

Disclosures

This study did not involve any human or animal subjects; therefore, IRB approval and informed consent were not needed.

Conflicts of interest

The authors declare that they have no conflicts of interest related to this work. No financial support, services, or third-party payments were received for any aspect of the submitted work. Additionally, the authors have no relevant financial activities outside of this work, patents, or other relationships that could be considered broadly relevant to the manuscript.

Consent for publication

Not applicable

Availability of data and materials

This article is based on previously published data and literature, all appropriately cited in the manuscript. No new datasets were generated or analyzed during the current study.

Authors' contributions

R.J.A.R. and R.E. jointly conceptualized the study, conducted the literature review, and drafted the manuscript. R.J.A.R. synthesized data from neuroimaging, pharmacological, and clinical studies, critically analysed research gaps, and integrated key discussions on dopaminergic dysfunction in migraine and psychosis. R.E. contributed to the organization of the manuscript, ensured coherence between sections, and provided critical insights into the pathophysiological mechanisms discussed. Both authors refined the structure, ensured scientific rigor, and aligned the manuscript with journal guidelines. The final version was reviewed and approved by both authors.

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Declaration of interests

The authors have no competing interests

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