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Migraine and Dementia: A Narrative Review of Current Evidence

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Summary

Background: Migraine is a common neurological disorder characterized by recurrent headaches, while dementia is a progressive neurodegenerative condition leading to cognitive decline. Recent studies indicate that these conditions may share common underlying mechanisms, such as vascular dysfunction, neuroinflammation, oxidative stress, and structural brain changes, all of which could contribute to cognitive impairment. **The aim** was to explore the potential connection between migraine, particularly migraine with aura, and an augmented risk of dementia. **Material and Methods:** the keywords Migraine, Dementia, Vascular Dysfunction, Neuroinflammation, Oxidative Stress, Cognitive Decline are used to search for articles in the PubMed and others databases for the period from 2000 to 2024. **Conclusions:** studies on the general population indicate that those who suffer from migraines, particularly those who have aura, may be more susceptible to dementia, including vascular and Alzheimer's diseases. However, the results are inconclusive, and further research is needed to understand this potential link. Shared mechanisms like endothelial dysfunction, neuroinflammation, and oxidative stress provide evidence of a connection between migraine and dementia. This potential link has significant clinical implications. Patients with a history of migraine should have their mental health closely watched by medical professionals, especially if they experience frequent auras or chronic migraines. Addressing vascular risk factors, improving migraine management, and informing patients about potential risks can help reduce the likelihood of developing dementia. Future investigations should focus on longitudinal studies to track the progression of migraine and dementia, mechanistic studies to understand the underlying pathways, genetic studies to identify shared susceptibility, and clinical trials to evaluate interventions aimed at reducing dementia risk in individuals with migraines. Public health initiatives aimed at raising awareness and promoting early intervention are crucial in influencing the prevalence of dementia in individuals with migraines.

Keywords: Migraine, Dementia, Vascular Dysfunction, Neuroinflammation, Oxidative Stress, Cognitive Decline

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INTRODUCTION

Migraine and dementia are significant public health concerns, affecting individuals personally, socially, and economically. Migraine affects about 1 billion people worldwide, making it the second most disabling neurological condition in terms of years lived with disability [1]. It primarily affects women and those in their prime working years, leading to substantial social and economic impacts. Despite extensive research, our understanding of this condition remains incomplete, and many people find existing treatments inadequate. This review aims to consolidate existing evidence on the relationship between migraine and dementia. Dementia is a condition that progressively impairs cognitive abilities in older adults, causing memory loss, decline in executive function, language skills, and reasoning [2]. Mostly 60–70% of dementia cases are caused by Alzheimer's disease (AD), it is the most prevalent type of dementia [3]. With the global population aging, there is a concerning rise in dementia prevalence, with projections suggesting that by 2050, over 130 million people may be affected [4]. The increasing number of dementia cases has led to heightened interest in exploring the potential connection between migraines and dementia. According to recent research, there may be a connection between migraines and a higher chance of dementia because of similar pathophysiological processes such as vascular dysfunction, neuroinflammation, and oxidative stress [5].

Migraines may not only cause chronic pain and disability but could also contribute to cognitive decline and neurodegeneration [6]. Exploring the potential link between migraines and dementia raises several questions, particularly regarding whether the repeated changes in cerebral blood flow and neuronal function seen in migraines could lead to lasting changes in the brain,

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increasing the risk of dementia. There is also interest in investigating whether structural brain changes in individuals with chronic migraines indicate a faster neurodegenerative process and whether effectively managing migraines could reduce the risk of cognitive decline in the future. This review aims to consolidate existing evidence on the connection between migraine and dementia through a comprehensive examination of the literature. It is crucial to understand the biological mechanisms involved, assess epidemiological evidence, and consider the clinical implications of these findings. The review seeks to bring together existing insights on the link between migraine and dementia, outline available evidence, explore potential shared mechanisms, and identify areas requiring further investigation. The primary goal is to improve outcomes for individuals affected by both migraine and dementia, emphasizing the need for collaborative research and shared responsibility in addressing these complex health issues.

MIGRAINE AND DEMENTIA

A common primary headache illness known as migraines is characterized by strong, recurrent headaches that frequently have an aura [7]. Migraines are one of the main causes of disability, despite the fact that they usually occur at a young age. On the other hand, dementia mainly affects the elderly. Previous studies have indicated that people with migraines may be more susceptible than people without migraines to getting dementia from all causes, Alzheimer's dementia, or vascular dementia [5]. However, earlier studies have shown inconsistent findings regarding the connection between migraines and dementia risk, with some studies finding this association only in women [8]. These discrepancies in the results could be explained by changes in the study layouts, patient identification techniques, duration of follow-up, age distribution, and results [9]. Additionally, a recent nationwide retrospective cohort study revealed that experiencing migraines during mid- and late-life is associated with an increased risk of all-cause dementia and Alzheimer's disease, though not vascular dementia [10, 11]. This underscores the complex relationship between migraines and dementia. These results emphasize the need for further analysis to explore the connection between migraines and different types of dementia, and the importance of ensuring an adequate sample size to achieve statistical power in future research. Studies have shown varying connections between migraines and the risk of dementia, with some suggesting a higher risk of different forms of dementia in individuals with migraines, particularly migraine with aura [12, 13]. Due to the long preclinical phase of Alzheimer's disease, the age difference between migraine and dementia, and the need to study each type of dementia separately, it is important to consider these factors. The differences in earlier studies may be due to variations in their methodologies.

Definition and Epidemiology

Migraine affects 15–20% of the population [14, 15] and is more prevalent in women [16], especially during their reproductive years, likely due to hormonal factors. It ranks as the second most significant contributor to disability worldwide, impacting individual quality of life and societal productivity [17]. Understanding the connection between migraine and dementia is crucial for effective prevention and management.

Migraines usually start in adolescence or early adulthood and peak between the ages of 25 and 55 [18]. The frequency of migraine attacks varies among individuals [19], with some experiencing fewer than fifteen headache days per month (episodic migraines) and others experiencing fifteen or more headache days per month for over three months (chronic migraines) [20], often accompanied by anxiety and depression [21].

Dementia refers to a range of neurodegenerative disorders causing a progressive decline in cognitive functions, significantly affecting daily activities [9]. Of all dementia cases, 60–70% are Alzheimer's disease [22]; other forms include vascular dementia, Lewy body dementia, and frontotemporal dementia [23]. While each type has distinct features, they share symptoms such as memory loss, reasoning difficulties, language challenges, and personality and behavior changes [24]. Some studies suggest a possible link between migraines and a higher risk of developing certain forms of dementia [5, 6, 11, 13, 24–26]. The number of individuals living with dementia is expected to triple by 2050, approaching 50 million, as a result of longer life expectancies and ageing demographics contributing to the global increase in dementia prevalence [27]. Dementia becomes more prevalent with age, and 5–10% of cases occur before the age of 65, posing challenges for individuals and their families [28]. The condition significantly impacts quality of life, leading to increased morbidity, mortality, and healthcare costs, with global costs expected to exceed $1 trillion annually as prevalence rises [29].

Pathophysiology

The mechanisms that contribute to migraines include a variety of environmental influences such as stress, hormonal changes, specific dietary choices, and alterations in sleep patterns, which can trigger migraine episodes in those who are predisposed [30]. Neuroinflammation, which involves glial cell activation and the release of pro-inflammatory cytokines inside the central nervous system, is considered a crucial influence in the underlying causes of migraines [31, 32]. Furthermore, the onset of migraines has been linked to oxidative stress caused by mitochondrial dysfunction and an increase in the production of reactive oxygen species (ROS) [33].

The fundamental processes of dementia vary by type, generally encompassing a mix of protein misfolding, neurodegeneration, and vascular abnormalities [34]. The pathophysiology of migraine is characterized by neurogenic inflammation, cortical spreading depression,

and activation of the trigeminal nerve [35]. The buildup of amyloid-beta (Aβ) plaques and hyperphosphorylated tau protein plays a crucial role in the progression of Alzheimer's disease [36]. The identified pathological characteristics lead to synaptic impairment, neuronal degeneration, and cerebral atrophy, particularly affecting the hippocampus and cortex, which are crucial for memory and cognitive abilities [37]. Specific genetic elements, including mutations in the APP, PSEN1, and PSEN2 genes and the presence of the APOE ε4 allele, can increase the probability of developing Alzheimer's disease [38].

Cerebrovascular disease is the primary cause of vascular dementia, encompassing conditions such as extensive artery atherosclerosis, small vessel disease, and stroke [39]. Prolonged decreased blood flow to the brain, resulting from these vascular conditions, adversely affects the white matter and the cortical and subcortical areas, contributing to cognitive decline.

The presence of intracellular alpha-synuclein protein clumps known as Lewy bodies characterizes Lewy body dementia [40]. The existence of these inclusions disrupts cellular function and contributes to neuronal deterioration, resulting in cognitive decline, parkinsonism, and visual hallucinations [41].

The development of aberrant proteins like tau and TDP-43 is linked to the degeneration of the frontal and temporal lobes in frontotemporal dementia [42]. Initially, this form of dementia mainly affects behavior, personality, and language rather than memory. Neuroinflammation and oxidative stress are commonly noted pathological features in different types of dementia [43]. Neuronal injury and the advancement of neurodegeneration are facilitated by the activation of microglia and the continuous synthesis of pro-inflammatory cytokines [44]. When reactive oxygen species (ROS) build up and mitochondrial malfunction occur, oxidative stress intensifies the resulting cellular damage and cognitive decline [45].

SHARED MECHANISMS LINKING MIGRAINE AND DEMENTIA

Recent research has suggested a potential link between migraine and dementia, indicating that these two seemingly unrelated conditions may share common underlying mechanisms.

Cerebral Blood Flow and Vascular Alterations. There is a significant relationship between migraine and dementia, particularly in terms of vascular dysfunction [13]. Changes in cerebral blood flow and the integrity of blood vessels have been associated with both conditions [46]. Temporary changes in cerebral blood flow are commonly observed in migraine, especially during the aura phase. This phase is characterized by cortical spreading depression (CSD), followed by changes in blood flow [47]. Variations in cerebral blood flow may lead to ischemic events, particularly in individuals with vascular risk factors [48]. Similarly, vascular dementia is closely linked to chronic cerebrovascular disease [49]. Multiple factors can contribute to complications in cerebral blood flow, leading to persistent reduced blood flow and the development of white matter hyperintensities (WMHs) in the brain [50]. These vascular changes are considered a significant contributor to the cognitive decline observed in dementia [51]. The relationship between vascular problems associated with migraine and dementia suggests that persistent migraine could exacerbate vascular damage over time, increasing the risk of cognitive decline and neurodegeneration [26].

Endothelial Dysfunction. The connection between migraine and dementia can be attributed to endothelial dysfunction, which is essential for the regulation of vascular tone, blood flow, and inflammatory responses [52]. Impaired endothelial function can lead to reduced availability of nitric oxide (NO), increased oxidative stress, and the onset of pro-inflammatory and pro-thrombotic conditions [53], contributing to the development of WMHs and other cerebrovascular abnormalities in individuals experiencing migraines [54]. Furthermore, the interruption of neuroinflammatory pathways and the impairment of cerebrovascular autoregulation may contribute to the link between migraine and dementia [49]. The development of vascular dementia and Alzheimer's disease is shown to be facilitated by endothelial dysfunction. Chronic impairment of endothelial function may lead to the degradation of the blood-brain barrier, allowing harmful substances such as amyloid-beta to enter brain tissue [55]. Amyloid-beta is a protein that aggregates into plaques in the brains of individuals diagnosed with Alzheimer's disease, leading to brain inflammation, damage to neurons, and exacerbation of cognitive decline [56]. The frequent association of endothelial dysfunction with both migraine and dementia suggests that experiencing migraines could potentially elevate the risk of developing dementia.

Neuroinflammation is a common feature of both migraine and dementia, significantly affecting the progression of these conditions [13]. In migraines, the activation of the trigeminovascular system results in the release of vasoactive neuropeptides, including calcitonin gene-related peptide (CGRP), which causes neurogenic inflammation [57]. This causes glial cells to generate pro-inflammatory cytokines such as interleukins (IL-1β, IL-6) and tumour necrosis factor-alpha (TNF-α), which are essential for maintaining chronic pain in migraine sufferers and sensitising pain pathways [58]. Chronic activation of microglial cells in response to tau tangles and amyloid-beta plaques causes pro-inflammatory cytokines to be released, which exacerbates neuronal damage and cognitive decline in dementia disorders such as Alzheimer's disease [59]. Moreover, ongoing brain inflammation generates reactive oxygen species (ROS), resulting in heightened oxidative stress and neuronal damage [60]. The link between migraine and dementia suggests that persistent migraine may contribute to

ongoing neuroinflammation, potentially accelerating the progression of neurodegenerative processes [6]. Additionally, the persistent activation of glial cells and the release of inflammatory mediators during migraine episodes may lead to lasting brain injury, increasing the likelihood of dementia onset [32].

Oxidative stress is a key factor connecting migraine and dementia. This condition is marked by a disparity between the generation of reactive oxygen species (ROS) and the organism's capacity to remove these reactive entities. In people with migraine, especially chronic migraine, there is strong evidence of mitochondrial dysfunction, leading to reduced energy production and increased ROS generation [33]. This can harm cellular components, ultimately causing damage to neurons and contributing to the development of migraine-related brain lesions. It is believed that similar mechanisms could be involved in the onset of dementia in certain individuals. The progression of neurodegeneration linked to dementia is significantly influenced by oxidative stress [43]. Malfunctioning mitochondria and the build-up of ROS contribute to oxidative injury in neurons, leading to the accumulation of misfolded proteins such as amyloid-beta and tau, which are associated with Alzheimer's disease [61]. Additionally, oxidative stress worsens vascular damage, leading to reduced blood flow to the brain and contributing to cognitive decline [62]. The connection between migraine and dementia suggests that individuals with chronic migraines may be at an augmented risk of developing dementia due to oxidative damage in the brain [63].

Changes in Brain Structure. Migraine, like dementia, has similar structural changes in the brain, such as GBV, cortical thinning, and hippocampal atrophy [64]. Both conditions show similar structural alterations in the brain, such as white matter hyperintensities (WMHs), cortical thinning, and hippocampal atrophy, suggesting a common mechanism linking the two [65]. Neuroimaging studies consistently demonstrate a higher occurrence of WMHs in individuals experiencing frequent or chronic migraine attacks [65]. These hyperintense regions observed on T2-weighted MRI scans suggest the presence of small vessel disease, gliosis, or demyelination [66]. WMHs are often observed in the deep white matter, periventricular areas, and brainstem and are associated with cognitive impairment in individuals experiencing migraines [67]. WMHs are valid indicators of small vessel disease in dementia and have a strong correlation with cognitive loss, especially in Alzheimer's and vascular dementia cases [68]. The presence and severity of WMHs in individuals with dementia are closely linked to the degree of cognitive impairment, suggesting their involvement in the neurodegenerative process [69]. Another notable structural alteration seen in both migraine and dementia is cortical thinning in the frontal, parietal, and temporal lobes [70]. In individuals experiencing migraines, the association between cortical thinning and the frequency and duration of migraine attacks has been observed. The observed thinning could result from the interplay of recurrent CSD episodes, neuroinflammation, and alterations in vascular structures [71]. Cortical atrophy serves as a crucial marker of neurodegeneration in dementia, marked by significant thinning in regions associated with memory, executive function, and language [72]. The structural brain changes [73, 74] seen in migraine show a correlation with those in dementia, suggesting that chronic migraine may influence or exacerbate the neurodegenerative processes linked to dementia. People who have experienced migraines for an extended period or have frequent episodes might be at a higher risk for cognitive decline, as indicated by the occurrence of WMHs and cortical atrophy in those affected by migraines [75].

Hereditary Influences. Substantial evidence indicates that genetic factors are essential in the progression of both migraine and dementia [13]. Furthermore, studies indicate that certain genetic variations could increase susceptibility to both conditions. The APOE ε4 allele, which is recognized as a genetic risk factor for Alzheimer's disease, must be emphasized because it has also been linked, in some populations, to increased migraine severity and frequency [76]. A potential shared genetic vulnerability indicates a possible common mechanism connecting migraine and dementia. Additionally, additional genetic loci have been associated with vascular regulation, inflammation, and ion channel function in both conditions [77].

Familial hemiplegic migraine is a rare kind of migraine with aura that is linked to mutations in the CACNA1A gene [78]. The association of these mutations with cerebellar ataxia and cognitive impairment is remarkable [79]. Furthermore, variations in the genes that control blood artery regulation, nitric oxide synthase (NOS) and angiotensin-converting enzyme (ACE), have been linked to migraines, an increased risk of cerebrovascular illness, and dementia [80]. Evidence suggests that genetic factors may be common to both migraine and dementia, indicating a potential shared cause for these conditions. Hence migraine and dementia exhibit overlapping pathophysiological mechanisms, including oxidative stress, mitochondrial dysfunction, and neuroinflammation.

EVIDENCE LINKING MIGRAINE TO DEMENTIA

Population Studies

Medical professionals are increasingly interested in the connection between migraine, especially migraine with aura, and the potential risk of developing dementia. Extensive investigations have provided significant insights into the possible association between migraines and an increased likelihood of developing dementia. However, findings from various studies have shown discrepancies, likely due to variations in research methodologies, participant

demographics, and the criteria used to define migraine and dementia.

Migraine with Aura and the Associated Risk of Dementia. Numerous extensive cohort studies have suggested a possible association between migraine with aura and a heightened risk of developing dementia compared to individuals without migraines or those with migraine without aura. A comprehensive 20-year study involving a significant group of women indicated that individuals with a history of migraines had a higher likelihood of experiencing cognitive decline and developing Alzheimer's disease compared to those without migraine [81]. In the same way, people with migraines were more likely than people without migraines to acquire dementia, particularly vascular dementia, according to the Study, which involved over 4,000 participants [10]. A potential connection might exist between migraine with aura and dementia, possibly related to vascular and neuroinflammatory changes associated with aura [82]. There is a significant relationship between migraine with aura and a heightened occurrence of white matter hyperintensities (WMHs) and other cerebrovascular irregularities, which further elevate the risk of dementia development [83]. It is important to note that not all investigations have identified a consistent link, as some have found no significant rise in dementia risk among individuals with migraine with aura [69].

Migraine without Aura and the Risk of Dementia. The link between migraine without aura (MO) and the risk of developing dementia remains uncertain. Some studies indicate that individuals with MO might exhibit a slightly increased likelihood of developing dementia, particularly vascular dementia [84]. However, this support is not as strong as it is for migraines with aura (MA). Variations in the frequency and duration of migraine attacks, along with the presence of coexisting conditions such as hypertension, diabetes, and depression — each of which poses its own risk for dementia — could play a role in the inconsistencies observed in the findings. Additional investigation is required to examine the possible mechanisms that may explain the link between migraine and dementia [85]. A study that included over 11,000 twins from the Danish Twin Registry revealed no significant association between MO and a heightened risk of developing dementia [86]. Conversely, the Rotterdam Study, which tracked more than 7,000 elderly participants for over a decade, indicated that those who suffered from migraines, with or without aura, had an increased likelihood of developing dementia, especially vascular dementia [11].

Chronic migraine and Deterioration of Cognitive Function. Chronic migraine is associated with a higher occurrence of various health issues, including depression, anxiety, and cardiovascular disease, all of which may contribute to cognitive decline [29, 73, 87]. Several investigations have suggested a possible link between chronic migraine and an increased risk of mild cognitive impairment (MCI) and dementia, especially among those experiencing frequent aura or having a significant number of cerebrovascular risk factors [5, 11, 13, 26, 29, 73, 87]. The American Migraine Prevalence and Prevention (AMPP) study found a notable link between chronic migraine and cognitive decline [88]. This extensive study tracked individuals with migraines over multiple years and revealed that those with chronic migraine faced a higher risk than those with episodic migraine [89].

Limitations of Population Studies. Population studies offer valuable insights into the possible link between migraine and dementia, it is crucial to acknowledge their limitations when interpreting the results. Many studies rely on self-reported migraine diagnoses, which can lead to inaccurate categorization and underestimate the actual prevalence of migraines. Recall bias is a possibility in some retrospective studies, particularly in investigations where participants must remember their migraine history long after the development of dementia. Addressing confounding factors in population studies presents considerable challenges. Multiple elements, such as existing health conditions, the use of medications, and individual lifestyle decisions, can affect the risk linked to both migraines and dementia. Therefore, it is crucial to explore the potential link between early cognitive decline and the escalation of migraine frequency or severity, as this could provide significant insights. Additional exploration is necessary to enhance our comprehension of the connection between migraine and cognitive decline, along with the particular mechanisms by which migraine might affect cognitive function.

Biological Plausibility

The connection between migraine and dementia is strongly supported by shared underlying mechanisms, as outlined in this review. These common traits, including vascular dysfunction, neuroinflammation, oxidative stress, and structural brain changes, make the connection biologically plausible. Further evidence strengthens this potential link.

Contributions to Vascular Studies. There is a significant association between migraine, especially migraine with aura, and an increased risk of dementia, with vascular dysfunction playing a crucial role. Migraine with aura involves temporary changes in cerebral blood flow, which are linked to cortical spreading depression (CSD) and issues with endothelial function. The observed vascular changes can lead to ischemic events, micro infarcts, and the development of white matter hyperintensities (WMHs), which are well-established risk factors for vascular dementia and cognitive decline. Additionally, there is a connection between migraines and a heightened risk of ischemic stroke, which is a key factor in the onset of vascular dementia. The repeated vascular incidents linked to migraines may result in persistent harm to the brain's blood vessels, heightening the likelihood of cognitive deterioration and the emergence of dementia as time progresses.

The Effects of Neuroinflammation and Oxidative Stress. The relationship between chronic neuroinflammation, migraine, and dementia suggests a potential association between these conditions. During a migraine attack, the trigeminovascular system becomes activated, releasing pro-inflammatory cytokines and vasoactive neuropeptides. These inflammatory mediators can sensitize pain pathways and contribute to neuroinflammation in the brain. Chronic neuroinflammation plays a crucial role in the progression of neurodegeneration, particularly in the context of Alzheimer's disease. The release of pro-inflammatory cytokines and subsequent activation of microglia can cause synaptic dysfunction, neuronal death, and the development of tau tangles and amyloid-beta plaques. The association between migraine and dementia suggests that persistent migraines could contribute to ongoing neuroinflammation, potentially accelerating the progression of neurodegenerative mechanisms. Reactive oxygen species generation and mitochondrial malfunction lead to oxidative stress, which is another well-known process. The relationship between oxidative stress and migraines is significant, as it can cause neuronal damage and the development of brain lesions commonly associated with migraine conditions. By encouraging the buildup of misfolded proteins and damaging blood vessels, oxidative stress also contributes significantly to dementia by accelerating cognitive loss.

Changes in Brain Structure. A number of structural brain abnormalities, including cortical thinning, hippocampal atrophy, and white matter hyperintensities (WMHs), are frequently linked to migraine and dementia. WMHs have been associated with an increased risk of dementia and cognitive decline and are more common among migraineurs, especially those who suffer aura often. The correlation between migraine and dementia may be explained by a shared anatomical abnormality in the frontal, temporal, and parietal lobes called cortical thinning. Evidence suggests that chronic migraine could potentially worsen or contribute to the development of neurodegenerative conditions like dementia. Individuals who have experienced migraines for a long time or have frequent episodes might be at a higher risk for cognitive decline, as indicated by the occurrence of WMHs and cortical atrophy in those affected by migraines.

Impact of Genetic Vulnerability. Strong genetic evidence supports the idea of a link between migraine and dementia. The APOE ε4 allele, for example, is one genetic variant linked to a higher risk of developing migraines as well as Alzheimer's disease. Identifying shared genetic factors suggests that these conditions might have a common underlying cause to some extent. There appears to be a connection between genetic vulnerability to migraines and dementia, indicating that individuals with a genetic predisposition to migraines might also face an elevated risk of developing dementia.

In conclusion, the review of publications indicates that there are common underlying factors linking migraine and dementia. These factors include complications related to blood vessels, neuroinflammation, heightened stress levels, alterations in brain structure, and a hereditary predisposition [90, 91]. The mechanisms indicate that prolonged chronic migraine may lead to the accumulation of brain damage, potentially increasing the risk of cognitive decline and dementia. On the other hand, there is hope for future approaches to treatment that could lower the incidence of dementia in people with a migraine history. However, additional research is desperately required to develop these promising medicines and to obtain a clearer knowledge of the link between these factors.

CLINICAL IMPLICATIONS AND FUTURE DIRECTIONS

The growing evidence linking migraine to an increased risk of dementia has important clinical implications for managing patients with migraine and suggests several areas for future research. Healthcare professionals should consider these findings in their clinical practice, especially the potential impact of migraine with aura on cognitive decline and dementia.

Early Detection and Ongoing Monitoring. Healthcare providers should proactively identify and monitor patients at risk of cognitive decline. This involves identifying individuals with a long history of migraines and assessing the frequency and intensity of their migraine episodes. Those experiencing chronic migraine, recurrent aura, or significant vascular risk factors may require more intensive monitoring for potential cognitive deterioration. Regular cognitive evaluations, such as the Mini-Mental State Examination (MMSE) or the Montreal Cognitive Assessment (MoCA), can help identify initial signs of cognitive decline in individuals with a long history of migraines. Early identification of cognitive decline enables prompt intervention and may help slow the progression of dementia.

Management of Vascular Risk Factors. Addressing vascular risk factors in individuals with migraine and dementia is essential for treatment and prevention. It is crucial to manage conditions like hypertension, hyperlipidemia, diabetes, and obesity, given their contribution to the risk of both migraine and dementia. Promoting lifestyle changes such as dietary adjustments, regular exercise, and smoking cessation is essential to mitigate vascular risk. These interventions have the potential to reduce the likelihood of recurrent migraine episodes and mitigate long-term cognitive decline.

Improving Migraine Treatment. Enhancing migraine treatment may reduce the risk of developing dementia. Effective management of migraines can reduce the frequency and intensity of attacks, subsequently diminishing the overall effects on the brain associated with migraines, including white matter hyperintensities (WMHs) and cortical thinning. Healthcare providers must ensure that individuals with migraines receive

appropriate preventive and acute care interventions. Individuals with chronic migraines may benefit from botulinum toxin injections or monoclonal antibodies targeting Calcitonin gene-related peptide (CGRP) or its receptor to significantly reduce the frequency of their attacks. Avoiding medication overuse is crucial, as it can lead to medication-overuse headaches and worsen chronic migraine conditions. Non-pharmacological treatments such as cognitive-behavioral therapy (CBT), biofeedback, and stress management techniques should be incorporated into a comprehensive treatment plan, potentially aiding in managing migraines and enhancing overall brain health and cognitive resilience.

Patient Education and Counseling. Future research should explore the potential mechanisms linking migraine and dementia. Educating patients about managing migraines and understanding their long-term effects is essential. Healthcare providers should provide compassionate education to patients about the possible link between migraine and cognitive decline, especially in individuals with a history of migraine with aura or chronic migraine. Patients should be educated on the importance of managing vascular risk factors and adhering to prescribed treatments with the guidance and understanding of their healthcare professionals. The psychological impact of living with chronic migraine, as well as the potential anxiety about the risk of dementia, should also be addressed in counseling. Offering support through mental health services, such as counseling and therapy, can help individuals manage the emotional burden of migraines and lessen the effects of stress, a known trigger for migraine episodes.

Prospective Pathways. Additional investigation is essential to comprehensively grasp the connection between migraine and dementia, as well as to formulate effective prevention and treatment approaches for both disorders. Extended studies are essential to monitor patients over prolonged durations, to elucidate the timing of the connection between migraine and cognitive decline. The objective of these studies is to ascertain whether migraine serves as a causal factor in the onset of dementia or if it merely coexists with the condition. Long-term studies must prioritize distinguishing the impacts of migraine with aura from those of migraine without aura concerning the risk of cognitive decline and dementia. Moreover, examining the effects of the frequency, duration, and severity of migraines on long-term cognitive outcomes would yield important insights for subsequent research.

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Conflict of interests

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