# Migraine Linked to Increased Vascular Dementia Odds in Observational Data

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Migraine, a debilitating neurological disorder affecting millions globally, extends beyond acute headache attacks. Its long-term neurological implications have been a subject of increasing scrutiny, particularly concerning cerebrovascular health. Clinicians have long observed a correlation between migraine and various cerebrovascular events, prompting questions about its role in neurodegenerative processes.

**KEY TAKEAWAYS**

- The Pivot: Migraine is not merely a headache disorder; it is a risk factor for vascular dementia, requiring vigilance in long-term patient management.
- The Data: Patients with migraine show a consistently increased risk for vascular dementia, with some studies reporting hazard ratios exceeding 1.5.
- The Action: Clinicians should consider migraine a potential early indicator of cerebrovascular vulnerability, prompting closer monitoring of vascular risk factors in these patients.

Migraine is a complex neurological condition, characterised by recurrent headaches often accompanied by sensory disturbances, nausea, and photophobia. Beyond the acute suffering, a growing body of evidence implicates migraine in a broader spectrum of neurological sequelae, particularly those affecting cerebrovascular integrity. The question of whether migraine predisposes individuals to neurodegenerative conditions, specifically vascular dementia, has moved from speculative to a focus of epidemiological inquiry. This concern stems from observations linking migraine, especially migraine with aura, to an elevated risk of stroke and white matter lesions, which are themselves risk factors for vascular cognitive impairment.

Vascular dementia, the second most common form of dementia after Alzheimer's disease, results from impaired blood flow to the brain, leading to cognitive decline. Its pathogenesis is intrinsically linked to cerebrovascular health, making any condition that impacts cerebral vasculature a potential contributor. The hypothesis connecting migraine to vascular dementia posits that recurrent migraine attacks, particularly those involving cortical spreading depression or vasospasm, may induce subtle, cumulative cerebrovascular damage over decades. This damage, while perhaps subclinical in younger patients, could accelerate or exacerbate the development of vascular pathology in later life, culminating in cognitive impairment.

## Understanding the association

Epidemiological studies, primarily large-scale cohort and case-control designs, have consistently identified an association between a history of migraine and an increased risk of vascular dementia. These studies typically leverage

national health registries, electronic medical records, or population-based cohorts, tracking individuals over many years to observe incident dementia diagnoses. The methodology often involves identifying cohorts of patients with a documented migraine diagnosis and comparing their subsequent incidence of vascular dementia to that of matched controls without migraine, adjusting for known confounders such as hypertension, diabetes, hyperlipidaemia, and smoking.

One such approach involves retrospective cohort analyses, where researchers identify patients diagnosed with migraine at baseline and follow them forward in time through medical records. They then ascertain the incidence of vascular dementia, often using diagnostic codes from hospital admissions or specialist neurology clinics. A key challenge in these studies is the accurate diagnosis of both migraine and vascular dementia, which can vary across healthcare systems and over time. Another common design is the case-control study, where individuals with vascular dementia are matched with controls without dementia, and their migraine history is retrospectively assessed. This design is efficient for rare outcomes but is susceptible to recall bias regarding migraine diagnosis.

The observed association is not always uniform across migraine subtypes. Some analyses suggest that migraine with aura carries a stronger association with vascular events and, consequently, vascular dementia, compared to migraine without aura. This distinction is clinically relevant, as migraine with aura is mechanistically linked to transient focal neurological symptoms, often attributed to cortical spreading depression, a wave of neuronal and glial depolarisation that propagates across the cerebral cortex. This phenomenon is thought to transiently reduce cerebral blood flow, potentially contributing to microvascular damage over time. The cumulative effect of these microvascular insults, even if individually subclinical, could contribute to the white matter changes and lacunar infarcts characteristic of vascular dementia.

Specific data points from meta-analyses of observational studies have shown that individuals with migraine have a significantly increased risk of vascular dementia. For example, some pooled analyses report a hazard ratio (HR) for vascular dementia in migraineurs ranging from 1.5 to 2.0, even after adjusting for traditional cardiovascular risk factors. This suggests that migraine itself, independent of other comorbidities, contributes to the risk. The confidence intervals around these estimates are typically tight, indicating a statistically robust association (e.g., 95% CI, 1.3-2.3; P<.001). The absolute risk increase, while modest in younger populations, becomes more pronounced with advancing age, aligning with the typical onset of vascular dementia.

The mechanisms underpinning this association are complex and likely multifactorial. Beyond cortical spreading depression, other proposed pathways include endothelial dysfunction, a prothrombotic state, and chronic inflammation, all of which are implicated in both migraine pathophysiology and cerebrovascular disease. Migraineurs, particularly those with frequent attacks, may exhibit subtle but persistent alterations in cerebral autoregulation, making their brains more vulnerable to fluctuations in blood pressure and cerebral perfusion. These changes could predispose them to silent brain infarcts and white matter hyperintensities, which are recognised precursors to vascular cognitive impairment.

The role of genetic predispositions also warrants consideration. Certain genetic variants associated with migraine susceptibility may also influence cerebrovascular health, creating a shared vulnerability to both conditions. For instance, genes involved in vascular tone regulation or inflammatory pathways could contribute to both migraine severity and the risk of vascular damage. However, specific genetic links directly connecting migraine to vascular dementia remain an active area of research, with no definitive causal genes identified to date.

Still, these observational studies, while compelling, do not establish causality. Confounding by indication is a persistent

concern; patients with migraine may be more likely to seek medical attention, leading to earlier or more frequent diagnoses of other conditions, including vascular risk factors, which then contribute to dementia. Residual confounding, where unmeasured or inadequately adjusted factors influence both migraine and dementia risk, also remains a possibility. For example, lifestyle factors such as stress, sleep patterns, and physical activity are known to influence both conditions but are often difficult to capture comprehensively in large datasets.

The diagnostic criteria for vascular dementia also present a challenge. Unlike Alzheimer's disease, which has relatively well-defined pathological hallmarks, vascular dementia is a heterogeneous condition, encompassing a spectrum of cognitive impairments resulting from various cerebrovascular pathologies. The reliance on clinical diagnostic codes, rather than post-mortem examination, introduces a degree of diagnostic imprecision. This imprecision could either dilute or inflate the observed association, depending on the specific biases inherent in clinical practice.

Another limitation in many of these studies is the lack of detailed information on migraine characteristics, such as attack frequency, severity, duration, and specific aura symptoms. These granular details could provide valuable insights into whether certain migraine phenotypes carry a higher risk for vascular dementia. For example, patients with chronic migraine or those experiencing frequent, prolonged aura might exhibit a different risk profile compared to those with episodic migraine without aura. Future research needs to incorporate more detailed phenotyping of migraine to refine these associations.

The long latency period between migraine onset, typically in adolescence or early adulthood, and the development of vascular dementia, usually in late life, also complicates research. This extended timeline makes it difficult to conduct prospective studies that capture the full trajectory of risk. Most studies rely on retrospective ascertainment of migraine history, which can be subject to recall bias, particularly for diagnoses made many years prior. Furthermore, changes in migraine diagnosis and management over decades could influence outcomes, but these historical variations are rarely accounted for in current analyses.

The clinical implications of this association are significant. If migraine is indeed an independent risk factor for vascular dementia, it underscores the importance of comprehensive vascular risk factor management in migraine patients, even those without overt cardiovascular disease. Early intervention to control hypertension, diabetes, and hyperlipidaemia, alongside lifestyle modifications, could potentially mitigate the long-term cerebrovascular consequences of migraine. However, whether aggressive migraine treatment itself, beyond symptomatic relief, can reduce the risk of vascular dementia remains an unanswered question, requiring dedicated prospective intervention trials.

The field needs more robust, prospectively designed studies with detailed neuroimaging and cognitive assessments to fully elucidate the causal pathways and identify specific migraine characteristics that confer the highest risk. Such studies would ideally follow large cohorts of migraineurs from a young age, tracking their cerebrovascular health and cognitive function over decades, while meticulously controlling for all known confounders. Only then can clinicians move beyond association to develop targeted preventive strategies.

> **CLINICAL IMPLICATIONS**
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> The consistent epidemiological link between migraine and an increased risk of vascular dementia demands a shift in how clinicians perceive and manage migraine. It is no longer merely a headache disorder; it is a potential marker of cerebrovascular vulnerability that warrants proactive, long-term risk reduction strategies.

European GPs and specialists should view a migraine diagnosis, particularly with aura, as an early warning sign for future cognitive decline.
This means a more aggressive approach to managing traditional vascular risk factors in migraine patients. Blood pressure, lipid profiles, and glucose levels should be monitored closely and treated to target, even in younger individuals. The evidence suggests migraine itself contributes to risk, so simply treating the headache is insufficient for long-term neuroprotection. This requires a broader, more integrated approach to patient care.
For the pharmaceutical industry, this association opens avenues for research into whether existing or novel migraine prophylactic therapies might offer neuroprotective benefits beyond headache reduction. A trial powered to detect a reduction in vascular dementia incidence in migraineurs would be a monumental undertaking, but one that could redefine the value proposition of these treatments. The current data, while observational, provides a strong rationale for such investigations.
Patients with migraine, especially those with a long history or aura, should be counselled about their elevated risk for vascular dementia. This education empowers them to engage more actively in lifestyle modifications and adherence to vascular risk factor management. While the news may be concerning, it provides a tangible reason for vigilance and proactive health management, shifting the narrative from passive suffering to active prevention.

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## REFERENCES

1. 1. Shin H, Ha WS, Kim J, Park SH, Han K, Baek MS, et al. Association between migraine and the risk of vascular dementia: A nationwide longitudinal study in South Korea. PLoS One. 2024;19(4):e0300379. doi:10.1371/journal.pone.0300379
2. 2. Hurh K, Jeong SH, Kim SH, Jang SY, Park EC, Jang SI, et al. Increased risk of all-cause, Alzheimer's, and vascular dementia in adults with migraine in Korea: a population-based cohort study. J Headache Pain. 2022;23(1):107. doi:10.1186/s10194-022-01484-y
3. 3. Jiang W, Liang GH, Li JA, Yu P, Dong M, et al. Migraine and the risk of dementia: a meta-analysis and systematic review. Aging Clin Exp Res. 2022;34(6):1237-1246. doi:10.1007/s40520-021-02065-w
4. 4. Morton RE, St John PD, Tyas SL. Migraine and the risk of all-cause dementia, Alzheimer's disease, and vascular dementia: A prospective cohort study in community-dwelling older adults. Int J Geriatr Psychiatry. 2019;34(11):1667-1676. doi:10.1002/gps.5180
5. 5. Wang L, Wu JC, Wang FY, Chen X, Wang Y. Meta-analysis of association between migraine and risk of dementia. Acta Neurol Scand. 2022;145(2):146-154. doi:10.1111/ane.13528

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