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Review Article

Assessing the Connection Between Migraine and Ischemic Stroke

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Abstract

Migraine and ischemic stroke are clinically distinct neurological disorders that share overlapping vascular and pathophysiological mechanisms. Migraine, especially with aura, has been observed to correlate with an elevated risk of ischemic stroke, a relationship that holds significant clinical relevance, especially in younger patients and women with additional vascular risk factors such as smoking or estrogen-containing contraceptive use.

The pathogenesis of this connection is multifactorial and not fully described. Some of the suggested mechanisms include cortical spreading depression (CSD), which results in transient cerebral hypoperfusion; endothelial dysfunction that contributes to a prothrombotic state; different cerebrovascular reactivity components; and the possible contribution of paradoxical embolism in patients with a patent foramen ovale (PFO). White matter hyperintensities evident on neuroimaging among migraineurs are additional structural changes that indicate a microvascular component that is chronic.

Methods: This narrative review synthesizes the existing literature on the relationship between migraine and ischemic stroke, focusing on epidemiological data, proposed mechanism, and clinical management strategies, including migrainous infarction. This article is intended to assist evidence-based, clinical decision-making that is informed by neurovascular.

Keywords: Migraine and Ischemic Stroke; Ischemic stroke; Migraine; Mechanism of Ischemic Stroke; A Neurovascular Perspective; Connection Between Migraine and Ischemic Stroke

Abbreviations

AAN: American Academy of Neurology; AHA: American Heart Association; AJNR: American Journal of Neuroradiology; ASA: American Stroke Association; BMJ: British Medical Journal; CDC: Centers for Disease Control and Prevention; CGRP: Calcitonin Gene-Related Peptide; CSD: Cortical Spreading Depression; CT: Computed Tomography; CTA: Computed Tomography Angiography; DTI: Diffusion Tensor Imaging; DWI: Diffusion-Weighted Imaging; FLAIR: Fluid-Attenuated Inversion Recovery; HR: Hazard Ratio; ICHD: International Classification of Headache Disorders; JAMA: Journal of the American Medical Association; MA: Migraine with Aura; MAI: Migrainous Arterial Infarction (note: inferred abbreviation, confirm usage); MO: Migraine without Aura; MRA: Magnetic Resonance Angiography; MRI: Magnetic Resonance Imaging; OC: Oral Contraceptives; PFO: Patent Foramen Ovale; TCD: Transcranial Doppler; TIA: Transient Ischemic Attack; TOAST: Trial of ORG 10172 in Acute Stroke Treatment Classification

Introduction

The manuscript is a narrative review that aims to explore the relationship between migraine and ischemic stroke, providing a comprehensive overview of the current evidence and clinical implications.

Migraine and ischemic stroke are prevalent and disabling neurological conditions encountered across various clinical settings. Although the two disorders have traditionally been regarded as independent pathophysiological processes, and these disorders remain clinically and epidemiologically allied, accumulating clinical and epidemiological data suggest that these disorders have a considerable interface, especially in patients with migraine with aura, a subtype that has been found to have a persistent and independent association with the severity of ischemic stroke.

The prevalence of migraine affects approximately 15% of the global population, with a higher incidence in females of reproductive age, compared to ischemic stroke, which is one of the major causes of morbidity and permanent disability all over the world, especially among older adults [1]. But stroke in younger groups, usually cryptogenic, has led to more attention to the possibility of migraine as an indicator of potential risk or mechanism. Notably, such a connection does not confine itself to the peri-ictal phase of migraine because the subclinical cerebrovascular changes, such as white matter hyperintensities (WMHs) and silent infarcts, were proven by studies even in migraineurs without the direct participation of an acute neurological event.

The interrelation of the pathophysiological pathways linking Migraine and ischemic stroke remains multifactorial. Such are cortical spreading depression (CSD), endothelium cell dysfunction, platelet aggregation dysfunctions, Arrhythmias (Atrial Fibrillation, AF), and right-to-left shunting of the heart, which most typically occurs through a patent foramen ovale (PFO). Moreover, some migraine-specific pharmacotherapeutics, specifically vasoconstrictive drugs (e.g., ergotamines and triptans), have the potential to worsen existing vascular risk in susceptible people [2].

In this review, we set out to break down the migraine stroke connection with clinical panache by outlining the neurovascular and hematologic intersection, appraising the prognostic and interventionist correlations, and providing a recommendation on risk stratification and prevention measures at a routine neurologic and primary care practice setting.

Mechanism and clinical characteristics of Ischemic stroke

Mechanism and clinical characteristics of Ischemic Stroke Ischemic stroke is defined as an acute neurological deficit caused by a focal cerebral, spinal, or retinal infarction secondary to vascular occlusion. It accounts for approximately 87% of all stroke cases globally, with the remainder attributed to intracerebral or subarachnoid hemorrhage [3,4]. The most common etiologies include large artery atherosclerosis, cardioembolism, and small vessel occlusion (lacunar infarcts), as stratified by the TOAST classification system. It has been estimated that the annual number of ischemic stroke cases amounts to 12.2 million patients globally (World Stroke Organization, 2023), and approximately 7 million deaths are caused by stroke aftermath. Recent information according to the Centers for Disease Control and Prevention (CDC.gov) of 2022 indicates that stroke continues as one of the top chronic disabilities in the U.S and has severe repercussions on cognitive and physical performance [5,6]. Pathophysiologically, the pathogenesis of ischemic stroke starts with a sudden loss of blood circulation to the brain, leading to energy dysfunction, excitatory and oxidative stress, and cell death. The ischemic cascade embraces the liberation of glutamate, calcium toxicity, mitochondrial dysfunction, and inflammation processes, which result in irreversible infarction unless reperfusion is attained within the therapeutic time range. Ischemic stroke Clinically, the most common presentation of ischemic stroke entails sudden onset of hemiparesis, aphasia, facial droop, or visual field deficits; however, posterior circulation strokes can be characterized by presenting with dizziness, ataxia, or isolated cranial nerve palsies. Diagnosis is time-sensitive, and non-contrast CT brain is the initial imaging modality to exclude or to make hemorrhage diagnosis, and then MRI-DWI, CTA/MRA, or CT perfusion is used to evaluate salvageable penumbra [7]. Notably, cases such as stroke imitations or unusual stroke pathologies, such as migraine-related pathologies, should be noted in younger patients who lack traditional risk factors. In a 2020 review, it was stressed that approximately 15% of strokes among patients younger than 50 are cryptogenic, as there is a need to consider the possibility of cardioembolism and vasculopathy in the former and migraine-related mechanisms (cortical spreading depression or paradoxical embolism via PFO) in the latter [8]. Early identification and classifica-

tion of stroke subtype are essential not only for acute reperfusion therapy, but also for targeted secondary prevention strategies, including antithrombotic therapy, risk factor control, and, in selected cases, structural interventions such as PFO closure.

5-Pathophysiological Mechanisms Linking Migraine and Ischemic Stroke The connection between migraine, specifically migraine with aura, and ischemic stroke is no longer an accident. Numerous epidemiological, imaging, and pathophysiologic studies have validated this two-way relationship and have indicated that migraine might represent not only an indicator of cerebrovascular susceptibility but also a frequent cause of ischemic events [16,17]. For hemorrhagic stroke, evidence is less consistent; some analyses suggest a slightly increased risk, particularly in women <45 years with aura, but absolute event rates remain low. Meta-analyses and large cohorts consistently show ~1.2-2.0x higher risk of ischemic stroke in people with migraine overall, with higher risk for migraine with aura than for migraine without aura. Representative estimates: HR 1.20 (overall migraine), HR 1.44 (MA), HR 1.13 (MO). In a groundbreaking 2016 meta-analysis report on BMJ, patients were observed to be at a 2.4-fold increased risk of ischemic stroke when migraine with aura was present, especially in women less than 45 years of age who smoked or used birth control pills that contained estrogen [18]. These findings were further affirmed by a 2020 review on AHA Journals (Stroke), which mentioned that as many as 20% of cryptogenic strokes might be mechanistically or associated with migraine in young adults [19]. Some hypothetical pathophysiological mechanisms are proposed to explain this relationship: Cortical Spreading Depression (CSD): Indeed, CSD, which is the neurophysiological correlate of aura, causes a temporary cerebral hypoperfusion, a pro-inflammatory cascade, and a rise in platelet aggregation. This can give rise to a system of microvascular ischemia, especially in patients with compromised vascular autoregulation. Endothelial Dysfunction: Changes in endothelial intima-media vasodilation, higher arterial irrigations, and prothrombotic markers of von Willebrand factor and homocysteine are evident among migraineurs. These alterations predispose the formation of microthrombi and vascular blockage.

Understanding migraine: A neurovascular perspective

Migraine is a complex neurovascular disorder characterized by recurrent episodes of moderate to severe headache, often accompanied by nausea, photophobia, phonophobia, and in some cases, focal neurological symptoms known as aura. The disorder exhibits a clear female predominance, with a global prevalence estimated at 14% in women and 7% in men, peaking between ages 25 and 55 (Global Burden of Disease Study, 2021, Lancet Neurology) [10].

The International Classification of Headache Disorders, 3rd edition (ICHD-3) divides migraine into

- Common migraine (migraine without aura).
- Classic migraine (Migraine with aura).
- Chronic migraine (15 days or more of headaches/month (more than 3 months) with at least 8 of them characterized by migraine, defining chronic migraine as a headache that occurs at least in part because of migraine and that lasts more than 3 months) [11].

Symptoms of aura, which are normally visual but on occasion sensory, language, or motor in nature, are thought to be a demonstration of the effect of cortical spreading depression (CSD): a slowly advancing wave of neuronal and glial depolarization followed by brain inhibition. The pathological conditions caused by CSD include transient oligoemia, the excretion of inflammatory mediators, and the destruction of blood-brain barriers, which are considered the roots of the vascular component of migraine to ischemic stroke.

The migraine is progressively being understood as a systemic melody, rather than a vascular action [12]. It has been demonstrated in advanced neuroimaging exploration (entailing ultrasophisticated MRI-FLAIR and T2-weighted-based) that various indications reported prevalence of white-matter hyperintense lesions in up to 30-40% of migraine-with-aura patients, including certain regions of the posterior circulation (Radiology Review, 2020, AJNR.org) [14]. The following changes are normally clinically silent but may indicate chronic microvascular compromise.

Clinically, the role of migraine diagnosis remains clinical, although whenever there are red flags (e.g., late-onset aura, hemiplegic symptoms, change of headache pattern), the utilization of neuroimaging is appropriate [13]. In 2022, AAN proposed to distinguish between migraine with aura and TIA since it is critical in older people or individuals at risk of cardiovascular events [15].

Notably, the pharmacologic-specific agents for migraine, such as triptans, ergot-derived drugs, and eventually the CGRP-pathway antagonists, exhibit decreasing degrees of vasculature. Cerebrovascular disease patients require triptans that are to be avoided due to their vasoconstrictor effect, but newer compounds, gepants and monoclonal anti-CGRP antibodies are safer to use in high-risk patients.

Given that the paradigm of migraine is changing beyond the study of pain pathways and into the consideration of neurovascular regulation such that it must be considered as a form of stroke risk phenotype in clinical practice, it is understandable why migraine, especially in the case of migraine with aura, is a prime candidate to assess as a form of stroke risk.

Bridging the two - exploring the connection between migraine and ischemic stroke

Over the past two decades, accumulating evidence has transformed our understanding of migraine from a benign primary headache disorder to a potential neurovascular risk phenotype, particularly in the setting of migraine with aura. Pathophysiological, clinical trial, imaging, and epidemiologic evidence have now demonstrated that there exists a statistically and biologically meaningful relationship between migraine, ischemic stroke, particularly in young to middle-aged women with coincident vascular risk factors.

The growing awareness urges physicians to consider migraine as a chronic pain syndrome and as indicator of cerebrovascular vulnerability, warranting clinical attention.

For instance, individuals who suffer from migraines have been found to have a higher risk of experiencing ischemic strokes during the perioperative period.

Pathophysiological mechanisms linking migraine and ischemic stroke

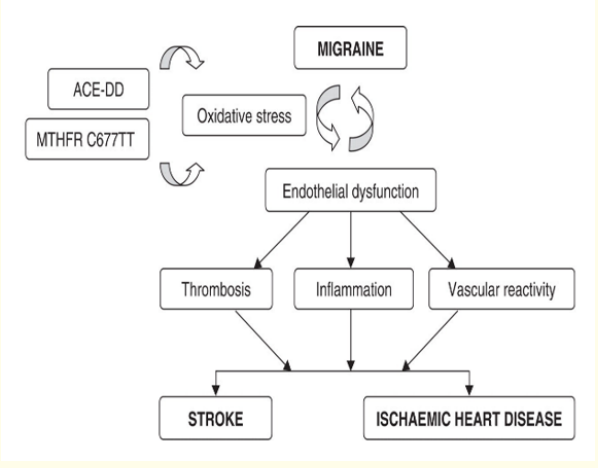

Figure 1: Possible Mechanism of Ischemia in Migraine.

Migraine may be linked to issues with the endothelium, which is the inner lining of blood vessels. This connection involves three key aspects: first, oxidative stress plays a significant role in this dysfunction; second, it leads to problems such as thrombosis, inflammation, and changes in how blood vessels react; and third, it is related to broader vascular diseases.

Numerous epidemiological, imaging, and pathophysiological studies have confirmed a connection between migraines, especially those accompanied by aura, and ischemic strokes.

This two-way relationship indicates that migraine may serve not only as a marker of cerebrovascular vulnerability but also as a frequent contributor to ischemic incidents [16,17]. Evidence regarding hemorrhagic stroke is less conclusive; certain studies point towards a slight increase in risk, especially among women under 45 years with aura, though the overall incidence remains low.

Meta-analyses and large cohort studies consistently indicate that individuals with migraine face a 1.2- to 2.0-fold elevated risk

of ischemic stroke compared to those without migraine, with a higher risk associated with migraine with aura than with migraine without aura. Representative hazard ratios include: HR 1.20 (overall migraine), HR 1.44 (with aura), and HR 1.13 (without aura).

A significant 2016 meta-analysis published in the BMJ reported that patients with migraine with aura experienced a 2.4-fold increase in ischemic stroke risk, particularly among women under 45 who smoked or used estrogen-containing contraceptives [18]. These findings were further supported by a 2020 review in the AHA Journals (Stroke), which indicated that approximately 20% of cryptogenic strokes in young adults may be mechanistically linked to migraine [19].

Several potential pathophysiological mechanisms have been proposed to elucidate this relationship:

- **Cortical Spreading Depression (CSD):** CSD, which correlates with migraine aura, leads to temporary cerebral hypoperfusion, initiates inflammatory responses, and increases platelet aggregation. This cascade can result in microvascular ischemia, particularly in patients with compromised vascular autoregulation.
- **Endothelial Dysfunction:** Migraines are associated with changes in endothelial intima-media function, augmented arterial flow, and prothrombotic markers like von Willebrand factor and homocysteine, which increase the risk for microthrombus formation and vascular obstruction.
- **Patent Foramen Ovale (PFO) and Right-to-Left Shunt:** In 2022, the Society for Cardiovascular Angiography and Interventions (SCAI) issued guidelines suggesting that individuals between 18 and 60 years old who have experienced a stroke related to a patent foramen ovale (PFO) should undergo closure of the PFO rather than just receiving antiplatelet medication. In contrast, the European Society of Cardiology (ESC) provided additional recommendations in June 2025, advocating for PFO closure in certain patients aged 18-60 with cryptogenic strokes, particularly those with significant anatomical risk factors or a high Risk of Paradoxical Embolism (RoPE) score. They advise against this procedure for individuals whose strokes are unlikely to be linked to PFO issues. For patients younger than 18 or older than 60, the current evidence does not sufficiently support a definitive recommendation.

It is recommended not to routinely close a patent foramen ovale (PFO) for issues like migraines or decompression sickness in divers, as the evidence supporting such procedures is often limited or inconsistent. However, in severe cases, closure could be an option, but it's essential that this decision is tailored to the individual patient. A detailed conversation regarding the uncertain advantages and possible risks should take place [21]. Ultimately, the choice to proceed with closure should involve a collaborative approach from a "Heart and Brain Team," which should consist of both a neurologist and a cardiologist to carefully evaluate the clinical situation, the anatomy of the PFO, and the overall risks and benefits involved.

- **Arrhythmias (Atrial Fibrillation, AF):** Earlier cohorts suggested an association between aura migraine and AF; newer large population-based studies (2024) do not confirm a general increase (some even report lower AF rates ≥55 y). Overall, the link is uncertain and unlikely to fully explain the migraine-stroke connection.
- **White Matter Hyperintensities (WMHs) and Silent Infarcts:** Neuroimaging studies reveal that individuals suffering from migraines, even without experiencing a definitive stroke, tend to exhibit a greater prevalence of white matter hyperintensities (WMHs) as well as silent cerebral infarcts. These infarcts are typically small and located in regions such as deep and juxtacortical areas of the frontal, parietal, and temporal lobes. These findings suggest that there is an ongoing chronic microangiopathy in the brain, which supports the theory of a long-term vascular condition. However, the clinical implications of these observations remain unclear, as research on their progression and impact on cognitive function has produced mixed results.
- **Hormonal and Lifestyle Factors:** The interplay between migraines and strokes has been examined, particularly in patients with a potential cerebrovascular syndrome. A history of migraines should prompt a careful assessment of stroke risk in clinical settings, especially in younger individuals who present atypical stroke symptoms but do not exhibit significant atherosclerotic factors or experience cryptogenic infarcts. The risk escalates for those exposed to exogenous estrogen, particularly in conjunction with smoking. The combination of visual aura and smoking dramatically heightens the risk; research indicates that women with visual aura who also smoke

and use oral contraceptives have approximately 7-10 times greater odds of suffering an ischemic stroke compared to non-smokers and those not using oral contraceptives. While combined hormonal contraceptives containing estrogen are shown to modestly raise the risk of ischemic stroke in the general population, this risk is significantly amplified (between three to six times in certain studies) for those experiencing migraine with aura. In 2021, the American Heart Association/ American Stroke Association (AHA/ASA) cautioned against using combined oral contraceptives in women with migraine accompanied by aura who also possess vascular risk factors. This growing body of evidence indicates that migraine, particularly in its aura form, should be viewed not merely as a primary headache disorder but rather as a significant health concern.

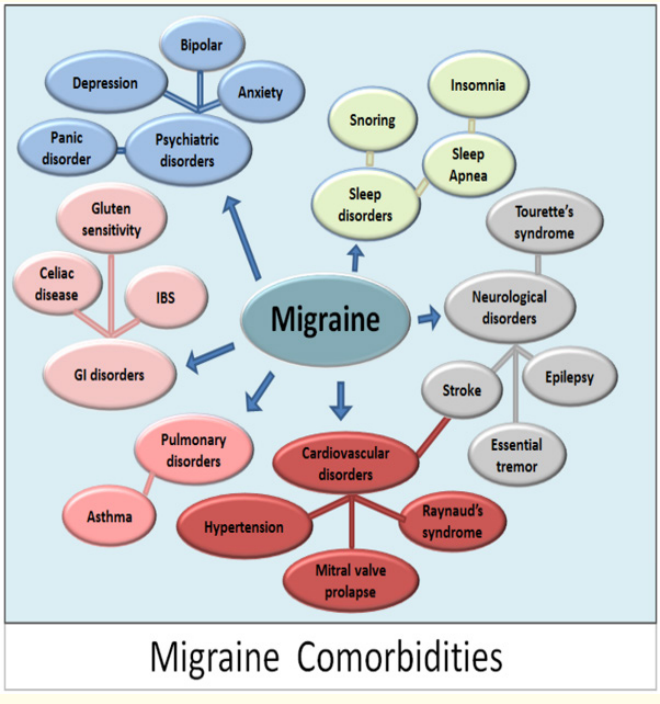

Figure 2: Illustrates various comorbid conditions associated with migraines.

Migrainous infarction and diagnostic considerations

Migrainous infarction is a not common but established clinical concept, characterized by the International Classification of Headache Disorders, 3rd Edition (ICHD-3), as a cerebral infarction that occurs within the framework of a typical migraine attack with aura, that during neuroimaging, the confirmation of ischemic atheroma is accompanied by the symptoms of the aura and the absence of any other cause of the stroke finds out [23].

It is a condition that constitutes about 0.5-1.5% of all ischemic strokes and is mainly associated with young women aged below 50 years of age who have no known vascular risk factors. It is reported that the localization of migrainous infarction is mostly restricted to the posterior circulation, an occurrence that involves occipital and cerebellar territories (Neurology.org (AAN, 2022)), which correspond to the standard aura symptomatology.

Diagnostic criteria (ICHD-3)

To make a diagnosis of migrainous infarction, the following conditions should be fulfilled:

- An ICHD-3 migraine attack that meets the aura given requirements.
- One or more long-lasting aura symptoms (more than 60 minutes).
- Ischemia illustrated by neuroimaging (MRI or CT) in an area related to the aura.
- There was no indication of an alternative infarction cause (e.g., cardioembolism, vasculitis).

Differentiating from stroke mimics and TIA

It is important and difficult to differentiate migrainous infarction and TIA or other MAI. The course of migraine aura changes within 5-20 minutes, possesses a positive (e.g., scintillation, paresthesia) component of visual, sensory, etc. nature, and presents a "marching" character along cortical areas [24]. In contrast, TIAs (Transient Ischemic Attacks) present suddenly and are characterized by negative symptoms such as loss of vision or weakness. Unlike migraines, these symptoms do not develop gradually. However, there can be some overlaps with migraine symptoms, so if there is any uncertainty, it is important to treat it as a potential stroke.

The neuroimaging is central. Results which implicate infarction may be confirmed by MRI-DWI, and vascular imaging (CTA, MRA, or TCD) should exclude causes of an underlying arteriopathy, dissection, or embolic sources. Cardiac testing with bubble contrast echocardiography is commonly warranted in young patients to determine the presence of PFO or an aneurysm in the atrial septum.

Key clinical considerations

- It is more likely to develop migrainous infarction when aura is frequent or prolonged, indicating a kind of threshold effect of recurring triggering of cortical spreading depression to reach ischemic thresholds with vulnerable locations.
- The vasoconstrictive migraine drugs (e.g., ergot compounds, triptans) consumed in extended aura are conceptualized to present an unfavorable ischemic risk, but limited information exists.
- Young women with focal neurologic deficits during migraine aura should be met with a high index of suspicion, as it can also occur in cases with co-factors oral contraceptives or smoking.

Despite the rarity, migrainous infarction highlights the importance of vascular risk screening and secondary prevention even among patients with a generally benign history of a headache. Such cases may (and in many cases) be addressed through a multidisciplinary approach, including neurology, cardiology, and vascular medicine.

Clinical implications and preventive strategies for physicians

The reported association between migraine, especially migraine with aura, and ischemic stroke has a large clinical implication for physicians in different fields such as neurology, general practice, vascular medicine, and geriatrics of women. Migraine ceased to be a benign pathology of the nervous system and more and more comes to be regarded as a modifiable risk factor at the end of the continuum of care for the cerebrovascular disorder.

Risk stratification and individualized assessment

Clinicians should be extra careful when dealing with migraineurs and, in particular, the aura-prone migraineurs, warning of vascular comorbidity. Significant features that amplify the risk are as follows:

- Female sex under age 50
- Smoking
- Use of estrogen-containing contraceptives
- History of aura with prolonged or complex features
- Family history of stroke or thrombophilia

Routine examinations of blood pressure, lipids, glycemic, and prothrombotic indicators are vital in such patients [25]. Additional investigation to determine with echocardiography, bubble study, and imaging of vessels (MRA, CTA) is warranted in those with migrainous features that appear suggestive of embolic phenomena.

Hormonal therapy and contraceptive management

It is recommended that combined hormonal contraceptives should be avoided in those women who have migraine with aura and one or more other vascular risk factors American Heart Association, 2021). Instead, progestin-only or non-hormonal approaches may be regarded as safer ones.

Pharmacologic decision-making

Making Vascular risk should also be taken into consideration when choosing migraine-specific therapies

- In hemiplegic migraine, traditional caution applies regarding triptans/ergots due to theoretical vasoconstriction risks.
- Triptans and ergotamines, being vasoconstrictive, are contraindicated in patients with a history of stroke or TIA, and in patients with severe vascular disease.
- For older adults who experience migraines and use triptans, the risk of vascular events (such as strokes or heart attacks) is relatively low but not completely absent.
- Selective agonists of the 5-HT1F receptor (Ditans) and gentle selective antagonists, such as CGRP monoclonal anti-CGRP antibodies, have acceptable safety profiles for populations at high risk and have become part of guideline-based preventive therapy (Neurology.org, 2023).

Lifestyle and behavioral modifications

The counseling must not be limited to pharmacologic approaches. Physicians ought to recommend:

- **Smoking cessation**
- **Regular aerobic exercise**
- **Dietary optimization and hydration**
- **Stress and sleep hygiene**, as both are known migraine triggers and cardiovascular risk factors

Secondary prevention in migrainous infarction

Secondary stroke prevention by the standards should be given to the patients who have experienced migrainous infarction:

- Antiplatelet therapy
- Risk factor modification
- Consideration of PFO closure in select individuals following stroke team consultation

Additionally, follow-up of the neurological cases is vital towards the management of migraine in the long run, and stroke education should be incorporated into the care of the patients to determine early occurrence of the relapses.

Future directions and research gaps

While the correlation between migraine—especially migraine with aura—and ischemic stroke is becoming increasingly recognized, numerous questions regarding this connection remain unanswered. These unanswered issues necessitate further research and could lead to enhanced risk stratification, methods for preventing harm, and the development of migraine-specific strategies to mitigate stroke risks.

Unresolved mechanistic pathways

Although various factors have been implicated, including cortical spreading depression (CSD), alterations in the endothelium post-transplantation, endothelial dysfunction, increased platelet reactivity, and paradoxical embolism linked to patent foramen ovale (PFO), the distinction between causal and associative roles of these mechanisms remains uncertain. It is still ambiguous whether migraine truly represents a pro-ischemic condition or if it merely exists as an epiphenomenon in patients with underlying cerebrovascular vulnerabilities. Recent findings reported in Neurology (2023) suggest that microvascular reactivity defects and an imbalance in endothelial nitric oxide may contribute to the risk of aura-associated strokes, though these results are not yet definitive.

Biomarkers and imaging indicators

There is an urgent need for reliable biomarkers that can identify migraine sufferers at the highest risk of experiencing cerebrovascular events. Advanced neuroimaging techniques, such as high-resolution vessel wall MRI and functional perfusion imaging, may offer valuable insights, but they are not widely utilized in current clinical practice. Additionally, methods like diffusion tensor imaging (DTI) and resting-state fMRI have shown alterations in functional connectivity and white matter integrity among migraine patients, particularly those who experience aura. However, these findings have not yet been linked to significant clinical outcomes.

Long-term effects of cgrp-targeted treatments

The long-term effects associated with the widespread use of CGRP monoclonal antibodies and gepants remain uncertain. Given that CGRP is known to promote vasodilation, chronic inhibition of this protein may result in changes to cerebrovascular function that are not yet fully understood.

Migraine in children and adolescents

Much of the research conducted so far has focused on adults, but migraines often begin during adolescence. It remains unclear whether early-onset aura signifies an increased risk for cerebrovascular issues later in life. There is currently a lack of prospective cohort studies in younger populations, which is a significant gap that needs to be addressed.

Sex-specific risks beyond hormonal treatment

Although it is well-established that female sex increases the risk of stroke, the exploration of sex-specific vascular mechanisms and effects beyond hormonal influences is still limited. Current research has primarily focused on potential vascular endothelial estrogen receptor polymorphisms and the expression of genes linked to sex chromosomes. Migraine is now recognized as a prevalent and often overlooked factor in cerebrovascular risk; however, there is still a need for personalized and mechanism-focused risk profiling to tailor preventive strategies. Filling these knowledge gaps will require future research that includes translational imaging and sex-specific studies.

Conclusion

With the changing association between migraine and ischemic stroke, it is essential to consider migraine as a potential cerebrovascular risk factor. This highlights the need for personalized preventive measures that include medication choice, lifestyle advice, and cardiovascular evaluation.

In particular, individuals experiencing migraine with aura exhibit a significantly heightened risk of ischemic events stemming from a variety of complex mechanisms. These include cortical spreading depression, endothelial dysfunction, prothrombotic conditions, and paradoxical embolism through a patent foramen ovale (PFO). Furthermore, neuroimaging studies reveal migraines may serve as an indicator of underlying cerebrovascular compromise, as evident from findings such as white matter hyperintensities and silent infarcts. Although migrainous infarction is rare, it occupies a unique position at the intersection of migraine and direct ischemic pathology. This emphasizes the importance of thorough risk assessment, especially for younger women who may have additional vascular risk factors, such as smoking and hormone therapy. Clinicians should recognize that migraines could be part of a broader spectrum of stroke risk. Tailored preventive strategies should be integrated into routine clinical practice, encompassing decisions on medication, lifestyle adjustments, and cardiovascular assessments. Future research should address the unresolved mechanistic questions, explore sex differences in susceptibility, and investigate the long-term effects of new migraine treatments. Ultimately, applying this knowledge in a practical setting will lead to improved outcomes by facilitating early identification, prevention, and intervention for migraine patients at risk of ischemic events.

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