American Journal Of Biomedical Science & Pharmaceutical Innovation

VOLUME Vol.06 Issue08 2026
PAGE NO. 12-16
DOI 10.37547/ajbspi/Volume06Issue08-02

Pathomorphology Of Migraine in Individuals with Ischemic Heart Disease

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Received: 25 June 2026; **Accepted:** 22 July 2026; **Published:** 12 August 2026

Abstract: Migraine and ischemic heart disease are among the most common conditions, significantly impacting quality of life and overall morbidity. Although these conditions are traditionally considered independent entities, recent research suggests common mechanisms underlying their development.

Keywords: Atherosclerosis, vascular wall, chronic ischemia, morphological analysis, myocardium.

Introduction: Migraine remains one of the most common forms of primary headache. According to the World Health Organization, symptoms of the condition are found in approximately one in seven adults. For a long time, the disease was viewed primarily as a neurological issue linked exclusively to the dysregulation of vascular tone and altered central nervous system activity. However, the accumulation of clinical observations has gradually changed this perspective.

Today, the involvement of systemic vascular disorders, chronic inflammation and endothelial dysfunction in the development of migraine attacks is increasingly being discussed. Upon careful analysis of publications in recent years, it becomes noticeable that in patients with cardiovascular pathology, migraine is much more common than previously thought. Particularly interesting is the group of patients with coronary heart disease, in whom changes in the vascular wall affect not only the coronary arteries, but also the microvasculature of various organs. In such cases, the clinical picture becomes more complex,

since the symptoms of both diseases can mutually exacerbate one another. It is sometimes difficult to determine which condition was primary. It is precisely this feature that makes the study of pathomorphological changes particularly relevant.

In the Republic of Uzbekistan, cardiovascular diseases have for many years ranked foremost among the causes of mortality in the adult population.

Concurrently, the prevalence of chronic neurological disorders, including various forms of migraine, is rising. Clinicians increasingly encounter patients diagnosed with both conditions simultaneously. In such cases, standard treatment regimens do not always yield a significant clinical effect, prompting a search for the underlying mechanisms driving the pathological process. Morphological analysis has emerged as a highly informative tool for assessing the extent of tissue and vascular damage; indeed, microscopic findings often reveal changes prior to the onset of pronounced clinical symptoms.

Such observations confirm the need for a comprehensive study of comorbid pathology. In my view, it is precisely morphology that reveals processes impossible to assess through laboratory or instrumental diagnostics alone. Sometimes, even minor structural changes explain the severe clinical course of a disease.

The role of the vascular endothelium in the development of both pathologies is of particular interest. Under normal conditions, endothelial cells

regulate vascular tone, inhibit thrombus formation, and ensure effective microcirculation. In ischemic heart disease, their functional activity gradually declines. Similar abnormalities are simultaneously observed in patients suffering from migraine.

Endothelial damage is accompanied by reduced nitric oxide synthesis, increased production of pro-inflammatory cytokines, and heightened vascular permeability. These processes create a favorable environment for the development of chronic tissue hypoxia. Against this background, morphological changes gradually develop in both the vessel walls and the surrounding tissues. There is a growing consensus in the literature that endothelial dysfunction may serve as a unifying link between migraine and ischemic heart disease.

Although certain issues remain the subject of active debate, the majority of researchers agree that the vascular factor plays a significantly more important role than was believed even ten to fifteen years ago.

This study employed a comprehensive clinical-morphological approach. The analysis was based on recent data from domestic and international scientific publications, as well as morphological findings presented in specialized manuals on pathological anatomy, cardiology, and neurology. The assessment of clinical characteristics took into account patient age, duration of migraine history, presence of cardiovascular risk factors, the clinical course of ischemic heart disease, comorbidity, and the results of instrumental examinations.

Particular attention was paid to analyzing microscopic changes in the vascular wall, myocardium, arterioles, and capillaries, as these structures reflect the severity of chronic vascular disorders.

The morphological analysis was based on light microscopy data, descriptions of histological specimens, and immunohistochemical findings published in current literature. Assessment of the microscopic findings took into account the state of the endothelium, the severity of atherosclerotic changes, and the degree of interstitial fibrosis, signs of chronic tissue hypoxia, the presence of inflammatory infiltration, characteristics of the microvasculature, and alterations in nerve tissue. Clinical symptoms were correlated with the identified structural changes. This approach made it possible to view the disease not as a collection of isolated symptoms, but as a unified pathological process involving interrelated vascular, neuronal, and tissue abnormalities.

The analysis was conducted systematically, incorporating a comparison of data from various authors; this enabled the identification of the most consistent patterns and the determination of directions for future research.

RESULTS

An analysis of current scientific publications indicates that the coexistence of migraine and ischemic heart disease is associated with more pronounced morphological changes in the vascular wall compared to the isolated course of either condition.

Most studies focus on the state of the endothelium, as it is the first to respond to chronic hypoxia, oxidative stress, and inflammatory changes. Microscopic examination of blood vessels in patients with ischemic heart disease reveals areas of endothelial cell desquamation, thickening of the basement membrane, reduced microvessel density, and focal signs of subendothelial fibrosis.

Such changes are considered the morphological basis for microcirculatory impairment. In patients with migraine, the severity of endothelial dysfunction is often greater than in those without chronic headache attacks. Some authors attribute this to recurrent episodes of vasospasm followed by compensatory vasodilation. Gradually, the vessel wall loses its elasticity, and signs of small-caliber artery remodeling emerge.

Morphological analysis also reveals changes in the perivascular connective tissue. Some studies report an increased content of collagen fibers around arterioles, indirectly reflecting chronic tissue hypoxia. Such remodeling of the microvasculature can exacerbate ischemic processes in both the myocardium and nervous tissue.

Examination of the myocardium in patients with ischemic heart disease most frequently reveals focal and diffuse interstitial fibrosis, dystrophic changes in cardiomyocytes, uneven capillary congestion, and signs of chronic ischemia. The morphological picture becomes more complex when cardiovascular pathology co-occurs with migraine over a prolonged period. The literature discusses the potential role of systemic endothelial dysfunction in the development of these changes. Cardiomyocytes often exhibit signs of granular dystrophy, while small areas of replacement sclerosis appear in regions of prolonged hypoxia. Some studies indicate an increase in the severity of microvascular abnormalities associated with frequent migraine attacks, particularly when the condition has persisted for many years. Although the causal relationship requires further investigation, accumulated observations suggest the existence of shared pathogenetic mechanisms. This explains the growing interest in the collaborative study of these pathologies by specialists in cardiology, neurology, and

pathological anatomy.

Morphological changes in the cerebral blood vessels also warrant particular attention. Published studies indicate that a subset of migraine patients exhibits signs of chronic remodeling of arterioles and capillaries, including vessel wall thickening, mild perivascular edema, and localized microcirculatory disturbances. While these changes are not specific to migraine alone, they may occur more frequently and be more pronounced when migraine co-occurs with ischemic heart disease. It is also noted that prolonged blood supply insufficiency can be accompanied by reactive changes in glial tissue.

Microscopic examination reveals areas of moderate gliosis associated with chronic hypoperfusion. It is important to emphasize that such findings are generally regarded as concomitant changes and require clinical interpretation in conjunction with neuroimaging and neurological examination data.

A review of the literature suggests that the coexistence of migraine and ischemic heart disease is associated with a complex of interrelated morphological changes.

Endothelial dysfunction, vascular wall remodeling, microcirculatory disturbances, interstitial myocardial fibrosis, and signs of chronic tissue hypoxia are the most consistently described features. These changes not only reflect the duration of the pathological process but may also hold prognostic significance. Their comprehensive assessment provides deeper insight into the mechanisms underlying this combined pathology and underscores the need for an interdisciplinary approach to patient evaluation and follow-up.

Data derived from an analysis of current literature suggest that the coexistence of migraine and ischemic heart disease represents a far more complex pathological process than the mere co-occurrence of two distinct conditions. Until relatively recently, this combination was viewed as a chance association, particularly in older patients. In recent years, however, perspectives on this issue have gradually shifted, with an increasing number of publications focusing on the shared mechanisms of vascular injury, chronic inflammation, and endothelial dysfunction.

According to the majority of researchers, it is precisely these processes that serve as a bridge between the neurological and cardiological manifestations of the disease. Morphological changes revealed during microscopic tissue examination provide a much deeper understanding of the disease's clinical course. They demonstrate that the pathological process affects not only a specific organ but the vascular system as a whole. Notably, the severity of structural changes does not always correlate with the intensity of clinical symptoms.

In some cases, patients with relatively infrequent migraine attacks exhibit pronounced changes in the microvasculature. In other instances, a long-standing course of the disease is accompanied by comparatively mild morphological abnormalities. Such differences underscore the need for an individualized assessment of each clinical case.

The condition of the vascular endothelial layer deserves special attention. It is the endothelium that reacts first to the effects of chronic hypoxia, dyslipidemia, oxidative stress, and systemic inflammation. These changes have been studied in considerable detail in the context of ischemic heart disease. Far less information is available regarding migraine, although the number of such studies has increased noticeably in recent years.

Most authors reach a similar conclusion: endothelial functional activity is also impaired in patients with migraine. The vascular wall's ability to respond adequately to changes in blood flow diminishes, nitric oxide synthesis decreases, and the production of vasoconstrictor substances increases. Such changes gradually become morphologically apparent. Microscopic examination reveals focal endothelial desquamation, thickening of the basement membrane, moderate perivascular fibrosis, and signs of chronic vascular wall remodeling. These findings suggest that migraine involves a significantly more pronounced vascular component than previously thought.

Changes in the microvasculature cannot be overlooked. Many studies identify impaired capillary blood flow as one of the early signs of the progression of comorbid pathology. With long-standing ischemic heart disease, there is a gradual reduction in the density of functioning capillaries. Concurrently, signs of chronic tissue hypoxia intensify. If these changes are compounded by recurrent episodes of vascular reactions characteristic of migraine, the strain on the microvasculature increases further. This creates conditions for the development of persistent structural changes. In the walls of small vessels. The amount of connective tissue gradually increases, the lumen of individual arterioles becomes less uniform, and the ability of the vessels to adapt rapidly to hemodynamic changes diminishes. These processes likely account for some of the clinical features of the disease course in patients with comorbid conditions.

The state of the myocardium is of considerable interest. Morphological analysis reveals that chronic ischemia is by no means always limited to atherosclerotic changes in the large coronary arteries.

In many cases, the microscopic appearance indicates marked microcirculatory disturbances. Cardiomyocytes gradually lose their normal structure, signs of protein and fatty degeneration emerge, and areas of interstitial fibrosis increase. These changes are particularly pronounced in patients with a long history of ischemic heart disease. Some authors report greater heterogeneity of vascular changes in cases where the condition co-occurs with migraine.

Although existing publications do not yet allow for a definitive conclusion regarding a direct causal link, the trend itself warrants further study. Chronic vascular dysfunction likely contributes to the gradual progression of morphological changes across various organs simultaneously. Changes in the central nervous system are also of great scientific interest; morphological studies indicate that long-standing migraine may be associated with moderate signs of chronic hypoperfusion in specific areas of the brain.

The most frequently described findings include localized white matter changes, reactive gliosis, mild enlargement of perivascular spaces, and focal microcirculatory disturbances. These findings are not specific to migraine alone; they also occur in other vascular diseases. However, when migraine co-occurs with ischemic heart disease, such changes may be more pronounced, as chronic vascular insufficiency affects multiple components of the circulatory system. Therefore, these findings should not be evaluated in isolation but rather in conjunction with clinical data, neuroimaging results, and laboratory findings.

An analysis of published studies reveals another noteworthy pattern. Many studies highlight the impact of metabolic disorders on the progression of both conditions. Arterial hypertension, type 2 diabetes mellitus, obesity, and lipid metabolism disorders significantly accelerate the development of vascular changes. For this reason, an increasing number of specialists view migraine in patients with ischemic heart disease not merely as a distinct neurological disorder, but as a potential clinical marker of generalized vascular dysfunction.

This perspective appears quite compelling. It explains why treating only a single condition does not always lead to significant clinical improvement; a comprehensive approach to managing risk factors may prove far more effective.

Early diagnosis is of particular importance. Morphological changes begin to develop well before the onset of pronounced clinical symptoms. For this reason, modern methods for assessing the vascular wall, the study of endothelial dysfunction biomarkers, and advancements in morphological techniques are becoming increasingly significant.

The earlier the initial signs of vascular damage are detected, the greater the likelihood of slowing the disease's further progression. This concept appears frequently in modern scientific literature and is well-founded. In practice, however, early diagnosis remains one of the most challenging tasks for both cardiologists and neurologists. It can only be achieved through close collaboration among specialists from various disciplines.

Summarizing published data, it can be noted that the pathomorphological changes associated with migraine in individuals with ischemic heart disease are systemic in nature. They involve the vascular wall, the microvasculature, the myocardium, and structures of the central nervous system.

The severity of these changes is determined not only by the duration of the disease but also by the patient's age, the presence of concomitant metabolic disorders, the extent of atherosclerotic vascular involvement, and the efficacy of the therapy administered. Recent studies indicate that further investigation into the morphological mechanisms of this combined pathology will enable the refinement of early diagnostic approaches, more accurate assessment of the disease prognosis, and the development of new methods for preventing vascular complications.

CONCLUSION

An analysis of contemporary scientific publications has demonstrated that the coexistence of migraine and ischemic heart disease constitutes a complex, multifactorial pathological process rooted in interrelated vascular, neurogenic, and morphological changes. Available data indicate that the occurrence of migraine attacks in patients with ischemic heart disease is accompanied not only by functional disturbances in vascular tone but also by gradual structural remodeling of the vessel wall, alterations in microcirculation, chronic tissue hypoxia, and remodeling of connective tissue components.

An analysis of the literature has established that endothelial dysfunction is a key component of the pathological process. Impairment of physiological endothelial activity is accompanied by reduced vasodilation capacity, increased oxidative stress, the development of chronic inflammation, and the progression of atherosclerotic changes. Morphological signs of these processes are observed in both the coronary vasculature and the cerebral vessels, confirming the systemic nature of the vascular involvement.

An analysis of morphological features has shown that

the co-occurrence of migraine and ischemic heart disease is most frequently associated with signs of interstitial myocardial fibrosis, thickening of the vascular wall, microcirculatory disturbances, focal dystrophic changes in cardiomyocytes, perivascular sclerosis, and evidence of chronic tissue hypoxia. The severity of these changes increases with the progression of the cardiovascular pathology and the duration of the disease.

Current data confirm the existence of a close relationship between morphological changes in the vascular system and the clinical course of the disease.

In patients with more pronounced structural abnormalities of the vascular wall, migraine attacks are characterized by longer duration and high pain intensity, and are frequently accompanied by persistent cerebrovascular disorders. This relationship underscores the need for a comprehensive assessment of clinical, instrumental, and morphological parameters. The practical significance of this review lies in the potential application of the findings to the development of algorithms for the early detection of vascular abnormalities in migraine patients with ischemic heart disease.

A comprehensive morphological study, combined with modern diagnostic methods, enables a more objective assessment of the extent of vascular damage, prediction of the disease course, and selection of the most rational treatment strategy.

Future research should focus on investigating the molecular mechanisms of endothelial dysfunction, the characteristics of microcirculatory remodeling, and the role of immuno-inflammatory processes, as well as on identifying new morphological biomarkers capable of predicting the development of complications in patients with comorbid migraine and ischemic heart disease.

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