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Abstract

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Intracranial atherosclerosis is a signi cant contributor to ischemic stroke, a leading cause of morbidity and mortality worldwide. is condition involves the formation of atherosclerotic plaques within the walls of arteries supplying blood to the brain. Over time, these plaques can progress to cause narrowing of the arterial lumen, known as intracranial stenosis, leading to reduced cerebral blood ow and subsequent ischemic events. e pathogenesis of intracranial atherosclerosis shares common features with atherosclerosis in other vascular beds but exhibits distinct characteristics due to the unique anatomical and hemodynamic properties of intracranial arteries. Plaque formation typically involves endothelial dysfunction, lipid accumulation, in ammatory processes, and smooth muscle cell proliferation within the arterial wall. e advanced stages of intracranial atherosclerosis can lead to plaque rupture or thrombosis, resulting in acute ischemic stroke [1].

Diagnosing intracranial atherosclerosis and assessing its severity are crucial for guiding therapeutic interventions and preventing stroke. High-resolution imaging techniques such as magnetic resonance imaging (MRI) and intravascular ultrasound (IVUS) play pivotal roles in visualizing intracranial atherosclerotic plaques and evaluating their characteristics, including composition and degree of stenosis. ese modalities enable clinicians to stratify stroke risk and tailor treatment strategies, ranging from medical management to endovascular interventions. is review aims to elucidate the pathophysiology of intracranial atherosclerosis, its clinical implications in stroke prevention, and the diagnostic approaches available to clinicians. By understanding the mechanisms underlying this condition and the tools available for its assessment, healthcare providers can enhance early detection and optimize management strategies to mitigate the burden of ischemic stroke associated with intracranial atherosclerosis [2].

Pathophysiology of Intracranial Atherosclerosis

Endothelial dysfunction and lipid accumulation:

Intracranial atherosclerosis begins with endothelial dysfunction, where the normal endothelial lining of intracranial arteries becomes compromised. is dysfunction allows for the in ltration of lipoproteins, particularly low-density lipoprotein (LDL), into the arterial wall. ese lipoproteins undergo modi cations, such as oxidation, leading to their retention in the intima. Subsequently, there is an in ammatory response, attracting monocytes and T lymphocytes to the site. ese cells further contribute to the uptake of lipids and the release of cytokines, promoting a chronic in ammatory state within the artery wall.

In ammatory processes and smooth muscle cell proliferation:

e in ammatory milieu within the artery wall stimulates smooth muscle cells to migrate from the media to the intima. Once in the intima, these smooth muscle cells proliferate and produce extracellular matrix proteins, contributing to the formation of a brous cap over the lipid-rich core of the plaque. is brous cap stabilizes the plaque but can also undergo changes that increase its vulnerability to rupture or erosion, leading to thrombosis and subsequent ischemic events [3].

Plaque formation and progression:

Over time, the accumulation of lipids, in ammatory cells, and smooth muscle cells leads to the formation of an atherosclerotic plaque within the intracranial artery. is plaque may initially be asymptomatic but can progress to cause signi cant stenosis of the arterial lumen. e progression of the plaque depends on various factors, including systemic risk factors (e.g., hypertension, diabetes mellitus) and local hemodynamic conditions. Advanced plaques may also develop features such as calci cation or hemorrhage, further complicating their stability and clinical management.

Clinical Implications

Ischemic stroke risk:

Intracranial atherosclerosis poses a substantial risk factor for ischemic stroke, particularly when the plaque leads to signi cant

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stenosis or undergoes rupture or thrombosis. Ischemic strokes associated with intracranial atherosclerosis can result from embolic events due to plaque instability or hemodynamic impairment due to severe stenosis.

Neurological sequelae:

e neurological consequences of intracranial atherosclerosis depend on the location and severity of the arterial involvement. Patients may experience transient ischemic attacks (TIAs), lacunar syndromes, or complete ischemic strokes, each of which can lead to varying degrees of neurological impairment and disability. Intracranial atherosclerosis not only a ects neurological function but also impacts overall quality of life. Stroke survivors may face long-term physical, cognitive, and according to thematic categories, including pathophysiological mechanisms, clinical manifestations, diagnostic modalities, and management strategies. Emphasis was placed on integrating ndings to provide a coherent understanding of the disease process and its implications for clinical practice. As a review article based on secondary data analysis, no primary data collection involving human or animal subjects was conducted. Ethical considerations focused on ensuring accuracy and objectivity in the interpretation of published research ndings, adhering to ethical guidelines for literature review and synthesis (Table 2).

Limitations:

Limitations of this study include potential biases inherent in systematic literature reviews, such as publication bias and variability in study methodologies across included articles. e scope of the review

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