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Abstract

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Intracranial atherosclerosis is a significant contributor to ischemic stroke, a leading cause of morbidity and mortality worldwide. This 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 flow and subsequent ischemic events. The pathogenesis of intracranial atherosclerosis shares common features with atherosclerosis in other vascular beds but exhibits distinct characteristics due to its unique anatomical and hemodynamic properties of intracranial arteries. Plaque formation typically involves endothelial dysfunction, lipid accumulation, inflammatory processes, and smooth muscle cell proliferation within the arterial wall. The 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. These modalities enable clinicians to stratify stroke risk and tailor management strategies, ranging from medical management to endovascular interventions. This 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. This dysfunction allows for the infiltration of lipoproteins, particularly low-density lipoprotein (LDL), into the arterial wall. These lipoproteins undergo modifications, such as

oxidation, leading to their retention in the intima. Subsequently, this is an inflammatory response, attracting monocytes and T lymphocytes to the site. These cells further contribute to the uptake of lipids and release of cytokines, promoting a chronic inflammatory state within the artery wall.

Inflammatory processes and smooth muscle cell proliferation:

The inflammatory 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 fibrous cap. This fibrous cap stabilizes the plaque. However, these cells also undergo changes that increase its vulnerability to rupture or erosion, leading to thrombosis and subsequent ischemic events.

Plaque formation and progression:

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

Clinical Implications

Ischemic stroke risk:

Intracranial atherosclerosis poses a substantial risk factor for ischemic stroke, particularly when the plaque leads to significant stenosis.

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Received: 01/12/2023
Revised: 15/01/2024
Published: 20/01/2024

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Keywords: Intracranial atherosclerosis, Endothelial dysfunction, Lipid accumulation, Ischemic stroke, Intracranial stenosis.

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:

The 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 affects 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 findings 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 findings, 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. The scope of the review

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