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PECULIAR FEATURES OF CEREBRAL PERFUSION IN YOUNG PATIENTS WITH METABOLIC SYNDROME

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Abstract: Metabolic syndrome (MetS) is a multifactorial clinical condition characterized by abdominal obesity, insulin resistance, arterial hypertension, dyslipidemia, and impaired glucose metabolism. Although traditionally associated with middle-aged and elderly populations, the prevalence of MetS among young adults has increased significantly over recent decades. Early vascular dysfunction caused by MetS may adversely affect cerebral blood circulation before the appearance of clinically evident cerebrovascular diseases. Cerebral perfusion abnormalities represent one of the earliest manifestations of vascular impairment and may contribute to cognitive decline and increased risk of ischemic stroke. This article reviews the specific characteristics of cerebral perfusion in young patients with metabolic syndrome and discusses the underlying pathophysiological mechanisms, diagnostic approaches, and clinical significance.

Keywords: Metabolic syndrome, cerebral perfusion, young adults, cerebral blood flow, insulin resistance, endothelial dysfunction, MRI perfusion, cerebrovascular disease.

Introduction

Metabolic syndrome (MetS) represents a complex metabolic disorder characterized by the coexistence of several cardiovascular risk factors that collectively increase morbidity and mortality worldwide. Although metabolic syndrome was previously considered a disease affecting middle-aged and elderly individuals, its prevalence among adolescents and young adults has risen considerably due to sedentary lifestyles, unhealthy dietary habits, obesity, and reduced physical activity. According to international epidemiological studies, approximately one-quarter of the adult population worldwide meets the diagnostic criteria for metabolic syndrome, while the prevalence among young adults continues to increase annually [1,2]. This trend has become a serious concern because vascular complications may begin developing long before clinical symptoms become apparent. The central nervous system is particularly sensitive to disturbances in blood circulation because the human brain requires approximately 20% of the body's oxygen despite constituting only about 2% of total body weight. Cerebral blood flow must remain relatively constant through highly regulated autoregulatory mechanisms that maintain adequate oxygen and nutrient delivery to neuronal tissue. Even slight reductions in cerebral perfusion may impair neuronal metabolism, decrease synaptic activity, and eventually contribute to structural brain damage [4]. Consequently, understanding cerebral hemodynamic alterations during the early stages of metabolic syndrome has become an important area of neurological and cardiovascular research.

The aim of this study was to analyze the peculiar features of cerebral perfusion in young patients with metabolic syndrome and to summarize the current evidence regarding the underlying pathophysiological mechanisms and their clinical significance.

Materials and Methods. This study was designed as a narrative literature review based on publications retrieved from international scientific databases, including PubMed, Scopus, Web of Science, and Google Scholar. Original articles, systematic reviews, clinical guidelines, and observational studies published in English between 2009 and 2024 were analyzed. The search strategy included the keywords metabolic syndrome, cerebral perfusion, cerebral blood flow, insulin resistance, endothelial dysfunction, young adults, arterial spin labeling, and magnetic resonance imaging. Studies involving young adults with metabolic syndrome that evaluated cerebral perfusion using neuroimaging techniques such as arterial spin labeling MRI, perfusion MRI, CT perfusion, or transcranial Doppler ultrasonography were included. Data concerning cerebral blood flow, cerebrovascular reactivity, endothelial function, and cognitive outcomes were synthesized and compared.

Results. Analysis of the reviewed studies demonstrated that young patients with metabolic syndrome exhibit early disturbances in cerebral perfusion despite the absence of overt neurological symptoms. Most neuroimaging investigations reported significantly reduced cerebral blood flow, particularly in the frontal cortex, hippocampus, temporal lobes, anterior cingulate cortex, and basal ganglia [4,7]. These regions are responsible for executive function, attention, learning, and memory.

The reviewed studies consistently identified endothelial dysfunction as one of the earliest pathological mechanisms contributing to impaired cerebral perfusion. Insulin resistance reduced nitric oxide bioavailability and increased oxidative stress, resulting in diminished vasodilation and vascular stiffness [2]. Chronic inflammation, characterized by elevated concentrations of TNF-α, IL-6, and C-reactive protein, further aggravated endothelial injury and microvascular dysfunction [2,4].

Hypertension and dyslipidemia were also associated with reduced cerebral perfusion through vascular remodeling, increased cerebrovascular resistance, and early atherosclerotic changes [6]. Several studies demonstrated impaired cerebrovascular reactivity and microvascular dysfunction, leading to decreased oxygen delivery despite relatively preserved blood flow in major cerebral arteries [4].

Furthermore, neuropsychological assessments revealed subtle cognitive impairment, including reduced attention, slower information processing, impaired working memory, and decreased executive function in young adults with metabolic syndrome [4,5].

Discussion. The findings indicate that cerebral perfusion abnormalities develop at an early stage of metabolic syndrome and may precede clinically apparent cerebrovascular disease. The combination of endothelial dysfunction, insulin resistance, chronic inflammation, hypertension, and dyslipidemia creates a multifactorial mechanism responsible for impaired cerebral circulation. Similar observations have been reported by Iadecola [4], who emphasized the importance of vascular dysfunction in early neurological impairment.

The predominance of hypoperfusion within the frontal cortex and hippocampus explains the cognitive deficits frequently observed in young adults with metabolic syndrome. These results are consistent with previous investigations demonstrating that reduced cerebral blood flow contributes to impaired executive function and memory performance [5].

Advances in neuroimaging, particularly arterial spin labeling MRI, have enabled non-invasive quantitative assessment of cerebral perfusion before structural brain lesions become detectable [7]. Early identification of cerebral hypoperfusion may therefore facilitate timely preventive interventions aimed at improving endothelial function and reducing long-term cerebrovascular risk.

Lifestyle modification, including weight reduction, regular physical exercise, healthy dietary habits, and pharmacological management of metabolic abnormalities, remains the most effective strategy for preserving cerebral circulation and preventing future neurological complications [2,3].

Conclusion. Young patients with metabolic syndrome demonstrate significant alterations in cerebral perfusion even in the absence of neurological symptoms. Endothelial dysfunction, insulin resistance, chronic inflammation, hypertension, and dyslipidemia collectively impair cerebral blood flow and cerebrovascular reactivity. Modern neuroimaging techniques allow early detection of these vascular abnormalities and may contribute to improved prevention of cognitive impairment and cerebrovascular disease. Further prospective clinical studies involving larger populations are required to establish the prognostic value of cerebral perfusion abnormalities and to optimize early therapeutic strategies.

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