

FUNCTIONAL AND STRUCTURAL CHANGES OF THE CENTRAL NERVOUS SYSTEM DEPENDING ON BODY MASS INDEX IN TYPE 2 DIABETES MELLITUS

Mamadinova Lolakhon Xomidovna

Assistant of the Department of Neurology,
Andijan State Medical Institute,

Doctor of Philosophy in Medical Sciences (PhD)

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Abstract

Type 2 diabetes mellitus is one of the most common metabolic diseases associated with chronic hyperglycemia and multiple systemic complications. Obesity and increased body mass index are considered important risk factors contributing to the progression of neurological complications in diabetic patients. The aim of this article is to study functional and structural changes of the central nervous system depending on body mass index in patients with type 2 diabetes mellitus. The analysis demonstrates that increased body mass index aggravates metabolic disturbances, vascular dysfunction, neuroinflammation, and neuronal damage, leading to cognitive impairment and structural brain alterations. Early diagnosis and комплексный management of obesity in diabetic patients may reduce the progression of central nervous system dysfunction.

Keywords: type 2 diabetes mellitus, body mass index, central nervous system, obesity, cognitive impairment, neurodegeneration, brain structure, metabolic disorders

Introduction

Type 2 diabetes mellitus is a chronic metabolic disorder characterized by insulin resistance, impaired glucose metabolism, and persistent hyperglycemia. The disease is associated with numerous complications affecting the cardiovascular, renal, peripheral nervous, and central nervous systems. In recent years, increasing attention has been paid to the impact of diabetes on the brain because chronic metabolic disturbances may lead to cognitive dysfunction, neurodegeneration, and structural brain changes.

Obesity is one of the main risk factors for the development and progression of type 2 diabetes mellitus. Increased body mass index contributes to insulin resistance, chronic low-grade inflammation, oxidative stress, endothelial dysfunction, and microvascular impairment. These pathological mechanisms negatively influence cerebral circulation and neuronal metabolism, increasing the risk of central nervous system damage.

Studies show that patients with type 2 diabetes and obesity have a higher incidence of cognitive decline, memory impairment, decreased attention, and reduced executive function compared with patients having normal body mass index. Structural changes may include cerebral atrophy, white matter lesions, reduced hippocampal volume, and impaired neuronal connectivity.

The relationship between body mass index and central nervous system alterations in diabetes remains an important scientific and clinical problem. Understanding these mechanisms is essential for early diagnosis, prevention, and development of effective treatment strategies.

Aim of the Study

The aim of this article is to analyze functional and structural changes of the central nervous system depending on body mass index in patients with type 2 diabetes mellitus.

Materials and Methods

This article is based on the analysis of scientific literature and clinical data related to neurological complications of type 2 diabetes mellitus. The study reviewed publications

concerning obesity, body mass index, neurodegenerative changes, cognitive dysfunction, and structural brain abnormalities in diabetic patients.

Clinical and diagnostic approaches used in the assessment of central nervous system involvement were analyzed, including neurological examination, cognitive assessment, magnetic resonance imaging, laboratory evaluation of metabolic parameters, and body mass index measurement.

Patients were conditionally divided according to body mass index categories to evaluate the relationship between obesity severity and neurological manifestations. Functional changes such as cognitive impairment, memory disorders, attention deficits, and emotional disturbances were compared with structural changes identified by neuroimaging methods.

Results

The analysis showed that patients with type 2 diabetes mellitus and elevated body mass index demonstrated more pronounced functional and structural changes of the central nervous system compared with patients with normal body weight.

Functional neurological disturbances included decreased memory, impaired concentration, reduced cognitive flexibility, emotional instability, and slower psychomotor reactions. Cognitive impairment was more frequent in patients with obesity and prolonged duration of diabetes.

Structural brain changes included cortical atrophy, enlargement of cerebral ventricles, white matter lesions, decreased hippocampal volume, and impaired microvascular circulation. Magnetic resonance imaging demonstrated that these changes were more severe in patients with higher body mass index.

The study also showed that obesity contributes to chronic neuroinflammation and oxidative stress. Increased levels of inflammatory mediators negatively affected neuronal metabolism and cerebral blood flow. Insulin resistance and vascular endothelial dysfunction were identified as important mechanisms leading to neuronal damage.

Hyperglycemia combined with obesity aggravated microangiopathy and reduced oxygen supply to brain tissues. As a result, chronic cerebral hypoxia contributed to progressive neurodegenerative changes and cognitive dysfunction.

Discussion

The findings confirm that body mass index significantly influences the severity of central nervous system dysfunction in patients with type 2 diabetes mellitus. Obesity not only worsens metabolic control but also accelerates neurodegenerative and vascular changes in the brain.

One of the key pathogenetic mechanisms is insulin resistance. In the central nervous system, insulin plays an important role in neuronal metabolism, synaptic plasticity, and cognitive processes. Impaired insulin signaling contributes to neuronal dysfunction and cognitive decline.

Another important factor is chronic inflammation associated with obesity. Adipose tissue produces inflammatory cytokines that may penetrate the blood-brain barrier and activate neuroinflammatory processes. Long-term inflammation damages neurons and contributes to cerebral atrophy.

Microvascular disorders also play a significant role. Diabetic angiopathy impairs cerebral circulation, resulting in ischemic changes and reduced oxygen delivery to neural tissue. White matter lesions and microstructural brain damage are common consequences of chronic vascular insufficiency.

The results suggest that management of body weight should be considered an important component in preventing neurological complications of diabetes. Lifestyle modification, glycemic control, physical activity, balanced nutrition, and treatment of obesity may help reduce the progression of central nervous system dysfunction.

Conclusion

Type 2 diabetes mellitus is associated with significant functional and structural changes of the central nervous system, especially in patients with increased body mass index. Obesity aggravates insulin resistance, chronic inflammation, oxidative stress, vascular dysfunction, and neurodegenerative processes.

Patients with elevated body mass index demonstrate more severe cognitive impairment and structural brain abnormalities compared with diabetic patients of normal weight. Early diagnosis of neurological complications and effective control of body mass index are essential for preventing progression of central nervous system damage.

Comprehensive management of diabetes and obesity may improve neurological outcomes, preserve cognitive function, and enhance quality of life in patients with type 2 diabetes mellitus.

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