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CLINICAL AND BIOCHEMICAL CHARACTERISTICS OF COGNITIVE DYSFUNCTION IN TYPE 2 DIABETES MELLITUS

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Abstract. Cognitive dysfunction is an increasingly recognized complication of type 2 diabetes mellitus (T2DM), significantly affecting patients' functional status and disease management. This study aimed to evaluate the clinical manifestations and biochemical characteristics associated with cognitive impairment in patients with T2DM. A cross-sectional observational study was conducted among 98 patients with T2DM treated at the Fergana Branch of the Republican Scientific Center of Emergency Medicine. Cognitive function was assessed using standardized screening tools, and biochemical parameters reflecting glycemic control, lipid metabolism, and insulin resistance were analyzed. Cognitive dysfunction was identified in 58.2% of patients and was significantly associated with poor glycemic control, higher insulin resistance, and dyslipidemia. Elevated HbA1c and fasting plasma glucose levels showed a strong correlation with the severity of cognitive impairment.

Keywords: type 2 diabetes mellitus, cognitive dysfunction, hyperglycemia, insulin resistance, dyslipidemia, HbA1c, metabolic control

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by persistent hyperglycemia, insulin resistance, and progressive β-cell dysfunction. Beyond its well-established microvascular and macrovascular complications, increasing attention has been directed toward its effects on the central nervous system. Cognitive dysfunction has emerged as a significant but often underrecognized complication of T2DM, affecting domains such as memory, attention, executive function, and information processing speed. These impairments may adversely influence treatment adherence, self-management, quality of life, and overall prognosis [1-5].

The pathophysiological mechanisms underlying cognitive impairment in T2DM are multifactorial and complex. Chronic hyperglycemia promotes oxidative stress, low-grade inflammation, and advanced glycation end-product formation, which together contribute to neuronal injury and cerebral microangiopathy. Insulin resistance at the level of the brain may disrupt insulin-mediated neuromodulation and synaptic plasticity, while recurrent hypoglycemic episodes can induce direct neuronal damage [6-8]. Additionally, dyslipidemia and endothelial dysfunction may compromise cerebral perfusion, further exacerbating cognitive decline.

Epidemiological studies indicate that patients with T2DM have a higher risk of mild cognitive impairment and dementia compared with non-diabetic individuals. However, the severity and clinical presentation of cognitive dysfunction vary widely, suggesting an important role for metabolic control, disease duration, and associated biochemical disturbances [9-12]. Despite growing global interest, data from Central Asian populations remain limited, and regional clinical-biochemical characteristics have not been sufficiently explored.

The present study was designed to investigate the clinical manifestations and biochemical correlates of cognitive dysfunction in patients with T2DM treated at a tertiary emergency medical center. By analyzing cognitive symptoms alongside metabolic and biochemical

parameters, this research aims to contribute to a more comprehensive understanding of diabetes-associated cognitive impairment and to provide evidence relevant for early identification and targeted intervention.

MATERIALS AND METHODS:

This observational cross-sectional study was conducted at the Fergana Branch of the Republican Scientific Center of Emergency Medicine. A total of 98 patients diagnosed with type 2 diabetes mellitus were enrolled between January and September 2024. The diagnosis of T2DM was established according to the criteria of the World Health Organization, based on fasting plasma glucose, glycated hemoglobin levels, and clinical history. Patients aged between 45 and 75 years with a disease duration of at least five years were included. Individuals with a prior diagnosis of neurodegenerative disease, major psychiatric disorders, acute cerebrovascular events, severe visual or hearing impairment, or chronic alcohol abuse were excluded to minimize confounding effects on cognitive assessment.

RESULTS

Among the 98 patients included in the study, cognitive dysfunction was identified in 57 individuals, corresponding to 58.2% of the cohort. Patients with cognitive impairment were generally older and had a longer duration of diabetes compared with those without detectable cognitive deficits. Clinically, cognitive dysfunction most frequently manifested as impaired short-term memory, reduced attention span, and slowed mental processing, often accompanied by subjective complaints of forgetfulness and decreased mental efficiency.

Table 1 presents a comparative analysis of the most common clinical symptoms observed in patients with and without cognitive dysfunction. Memory complaints and attention deficits were significantly more prevalent in the cognitively impaired group, while symptoms such as chronic fatigue and sleep disturbances were also more frequently reported.

Table 1

Clinical symptoms in patients with type 2 diabetes mellitus with and without cognitive dysfunction

Clinical symptom	Cognitive dysfunction (n = 57), %	No cognitive dysfunction (n = 41), %	p-value
Memory impairment	73.7	29.3	<0.001
Attention deficit	68.4	26.8	<0.001
Slowed thinking	54.4	19.5	0.002
Chronic fatigue	61.4	36.6	0.021
Sleep disturbances	49.1	31.7	0.048

Biochemical analysis revealed significant differences between the two groups. Patients with cognitive dysfunction demonstrated poorer glycemic control, as evidenced by higher fasting plasma glucose and HbA1c levels. Dyslipidemia was also more pronounced, particularly elevated triglyceride levels and reduced high-density lipoprotein cholesterol. Insulin resistance indices were significantly higher among cognitively impaired patients.

Table 2 summarizes the key biochemical parameters and the results of statistical comparisons between groups.

Table 2

Biochemical parameters in patients with type 2 diabetes mellitus with and without cognitive dysfunction

Parameter	Cognitive dysfunction	No cognitive dysfunction	p-

	(mean ± SD)	(mean ± SD)	value
Fasting plasma glucose (mmol/L)	9.8 ± 1.6	8.4 ± 1.3	<0.001
HbA1c (%)	8.9 ± 1.1	7.6 ± 0.9	<0.001
Triglycerides (mmol/L)	2.3 ± 0.7	1.8 ± 0.5	0.004
HDL-cholesterol (mmol/L)	0.96 ± 0.18	1.12 ± 0.21	0.001
HOMA-IR	4.6 ± 1.3	3.2 ± 1.1	<0.001

Correlation analysis demonstrated a significant association between HbA1c levels and the severity of cognitive impairment, suggesting that chronic hyperglycemia plays a central role in cognitive decline among patients with T2DM.

DISCUSSION

The findings of this study indicate that cognitive dysfunction is highly prevalent among patients with type 2 diabetes mellitus and is closely associated with poor metabolic control and adverse biochemical profiles [13]. More than half of the examined patients exhibited measurable cognitive deficits, predominantly affecting memory and attention, which is consistent with data reported in international literature.

Chronic hyperglycemia appears to be a key determinant of cognitive impairment, as reflected by significantly higher HbA1c levels in affected patients. Sustained elevation of blood glucose may contribute to neuronal damage through oxidative stress, mitochondrial dysfunction, and microvascular injury [15]. The observed association between insulin resistance and cognitive dysfunction further supports the concept of impaired insulin signaling within the brain, which has been implicated in reduced synaptic plasticity and neurodegeneration.

Dyslipidemia, particularly elevated triglycerides and reduced HDL-cholesterol, was more pronounced in patients with cognitive impairment [16]. These abnormalities may exacerbate endothelial dysfunction and cerebral atherosclerosis, leading to compromised cerebral blood flow and subsequent cognitive decline. The coexistence of multiple metabolic disturbances likely amplifies their detrimental effects on brain structure and function.

The clinical implications of these findings are substantial. Cognitive dysfunction may interfere with a patient's ability to adhere to complex therapeutic regimens, recognize hypoglycemic symptoms, and engage in effective self-care [17-19]. Early identification of cognitive impairment in patients with T2DM is therefore essential, particularly in those with long disease duration and suboptimal metabolic control. Routine cognitive screening, combined with aggressive management of glycemia and lipid abnormalities, may help mitigate the progression of cognitive decline.

CONCLUSION

Cognitive dysfunction represents a frequent and clinically significant complication of type 2 diabetes mellitus, closely linked to long-standing disease and inadequate metabolic control. The present study demonstrates that patients with T2DM and cognitive impairment exhibit significantly worse glycemic indices, higher insulin resistance, and more pronounced lipid abnormalities compared with cognitively preserved individuals. These metabolic disturbances likely contribute synergistically to neuronal injury and cerebral microvascular dysfunction, accelerating cognitive decline. The high prevalence of cognitive impairment observed emphasizes the need for routine cognitive screening in patients with T2DM, particularly in those with poor glycemic control and prolonged disease duration. Integrating cognitive assessment into

standard diabetes care and optimizing metabolic parameters may improve clinical outcomes, treatment adherence, and quality of life.

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