SPECIFIC FEATURES OF COGNITIVE IMPAIRMENTS IN TYPE 2 DIABETES MELLITUS

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Annotation

Type 2 diabetes mellitus demonstrates significant associations with cognitive deterioration across multiple neuropsychological domains. This investigation examined specific patterns of cognitive dysfunction in diabetic populations through comprehensive neuropsychological assessment and metabolic parameter analysis. The study evaluated 127 patients with established type 2 diabetes and 85 matched controls, revealing distinct executive function deficits, working memory impairment, and processing speed reduction in the diabetic cohort. Glycemic control quality correlated inversely with cognitive performance metrics, while disease duration predicted progressive cognitive decline trajectories. These findings underscore the clinical imperative for systematic cognitive screening and metabolic optimization strategies in diabetes management protocols.

Keywords: diabetes mellitus, cognitive impairment, executive dysfunction, glycemic control, neurodegeneration, insulin resistance, hippocampal atrophy

The global prevalence of type 2 diabetes mellitus continues its ascending trajectory, with current epidemiological data indicating approximately 537 million affected individuals worldwide. While traditional diabetic complications including retinopathy, nephropathy, and peripheral neuropathy receive considerable clinical attention, the cognitive consequences of chronic hyperglycemia remain insufficiently recognized in routine practice. Emerging neurobiological evidence demonstrates that prolonged metabolic dysregulation initiates cascade mechanisms that compromise neural integrity, synaptic plasticity, and neurotransmitter systems. Population studies reveal that diabetic individuals face substantially elevated risks for both mild cognitive impairment and frank dementia compared with metabolically healthy populations. Despite this concerning association, the precise phenomenological characteristics of diabetes-associated cognitive dysfunction, its temporal evolution patterns, and the specific neuropsychological domains most vulnerable to metabolic perturbation require further elucidation. This investigation aimed to characterize the distinctive cognitive profile manifested in type 2 diabetes and to examine relationships between metabolic parameters and neuropsychological performance across multiple cognitive domains.

Literature review

Previous investigations have established foundational knowledge regarding diabetic cognitive complications, though significant gaps persist in understanding specific mechanisms and clinical presentations. Biessels and colleagues documented accelerated cognitive aging in diabetic populations, with longitudinal observations revealing annual decline rates exceeding those in age-matched controls. Reijmer's neuroimaging analyses identified structural brain alterations including hippocampal volume reduction and white matter integrity compromise in diabetic cohorts. However, these structural findings required correlation with detailed neuropsychological phenotyping. Yaffe's large cohort study demonstrated hypoglycemic episode frequency as an independent predictor of cognitive deterioration, yet the differential vulnerability of specific cognitive domains remained unclear. Russian investigators led by Strokov examined diabetic polyneuropathy relationships with central nervous system dysfunction, establishing peripheral-central correlations but not addressing isolated cognitive profiles. Recent work by Mansur explored inflammatory mediators in diabetic cognitive impairment, though sample sizes limited generalizability. The existing literature predominantly focuses on global cognitive metrics rather than domain-specific patterns, and few studies have systematically examined the interaction between glycemic variability, disease chronicity, and discrete neuropsychological functions. This research gap necessitates comprehensive domain-specific cognitive assessment in well-characterized diabetic populations.

Methodology

This cross-sectional analytical study was conducted at the Regional Endocrinology Center between March 2022 and November 2023, following approval from the institutional ethics committee and adherence to Declaration of Helsinki principles. The investigation enrolled 127 patients with established type 2 diabetes mellitus diagnosed according to American Diabetes Association criteria, with disease duration ranging from 3 to 18 years. A control group comprising 85 metabolically healthy individuals matched for age, educational level, and socioeconomic status underwent identical assessment procedures. Inclusion criteria specified age between 45 and 70 years, documented diabetes diagnosis exceeding three years, current metabolic management with oral hypoglycemic agents or insulin therapy, and willingness to complete extensive neuropsychological evaluation. Exclusion parameters encompassed prior cerebrovascular events, documented psychiatric disorders, current psychotropic medication use, alcohol dependence history, severe hepatic or renal dysfunction, thyroid disorders, and any neurological conditions potentially confounding cognitive assessment. All participants provided written informed consent after detailed protocol explanation, and those demonstrating comprehension difficulties during consent procedures were excluded.

Neuropsychological assessment employed a comprehensive battery administered by trained clinical psychologists in standardized environmental conditions. Global cognitive screening utilized the Montreal Cognitive Assessment, which demonstrates superior sensitivity for detecting mild cognitive impairment compared with alternative brief screening instruments. Executive function evaluation incorporated the Trail Making Test Parts A and B, with completion time differences providing indices of cognitive flexibility and set-shifting capacity. The Digit Span subtest from the Wechsler Adult Intelligence Scale assessed working memory through forward and backward recall trials, while the Symbol Digit Modalities Test quantified processing speed and sustained attention. Verbal memory examination employed the Rey Auditory Verbal Learning Test, measuring immediate recall, learning slope across trials, delayed recall, and recognition discrimination indices. The Stroop Color-Word Interference Test evaluated inhibitory control and response conflict resolution. Visual memory assessment utilized the Brief Visuospatial Memory Test-Revised, examining encoding, consolidation, and retrieval of spatial information. Language functions were probed through phonemic and semantic verbal fluency tasks, reflecting lexical access and strategic retrieval processes. Metabolic characterization included glycated hemoglobin measurement via high-performance liquid chromatography, with sampling conducted within 48 hours of cognitive assessment to ensure temporal relevance. Fasting plasma glucose and postprandial glucose measurements provided additional glycemic indices. Comprehensive lipid panels quantified total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and triglyceride concentrations. Systematic screening for diabetic complications encompassed ophthalmologic examination for retinopathy grading, urinary albumin-to-creatinine ratio determination for nephropathy assessment, and standardized neurological examination with monofilament testing for peripheral neuropathy detection. Blood pressure measurements followed standardized protocols with three separate determinations. Medical record review extracted data regarding diabetes duration, current pharmacological regimen, documented hypoglycemic episodes, and comorbid conditions including hypertension and dyslipidemia.

Results

Demographic and clinical characteristics demonstrated adequate matching between groups, with mean age of 58.3 years in the diabetic cohort compared with 57.6 years in controls, showing no significant difference. Educational attainment distributed similarly across groups, with mean completion of 13.4 years in diabetic patients versus 13.8 years in controls. The diabetic group exhibited mean disease duration of 9.7 years, with glycated hemoglobin averaging 8.2 percent, indicating suboptimal glycemic control across the sample. Microvascular complications prevalence included retinopathy in 43 percent of diabetic participants, nephropathy in 31 percent, and peripheral neuropathy in 52 percent. Hypertension

affected 68 percent of diabetic individuals compared with 34 percent of controls, while dyslipidemia rates reached 71 percent versus 28 percent respectively. Neuropsychological assessment revealed significant cognitive performance disparities between groups across multiple domains. Montreal Cognitive Assessment scores averaged 24.1 in diabetic patients compared with 27.3 in controls, representing a clinically meaningful three-point differential. This global screening difference reflected specific domain vulnerabilities rather than uniform cognitive decline. Executive function evaluation through Trail Making Test Part B demonstrated mean completion times of 98.7 seconds in diabetic individuals versus 72.3 seconds in controls, indicating substantial impairment in cognitive flexibility and task-switching efficiency. The interference score, calculated as the difference between Parts B and A, averaged 52.4 seconds in diabetic patients compared with 34.1 seconds in controls, further emphasizing executive control deficits. Digit Span backward performance, specifically taxing working memory and mental manipulation capacities, showed mean spans of 4.2 in diabetic participants versus 5.6 in controls, while forward span differences proved less pronounced at 6.1 versus 6.7, suggesting that active maintenance and manipulation processes suffer disproportionately compared with passive storage. Processing speed assessment via the Symbol Digit Modalities Test yielded mean scores of 38.4 correctly completed items in diabetic patients compared with 48.9 in controls, representing approximately 21 percent performance decrement. This psychomotor slowing reflected both perceptual processing efficiency reduction and motor output speed compromise. Stroop Color-Word Interference Test performance revealed prolonged response latencies in the incongruent condition for diabetic individuals, with mean completion times of 67.8 seconds versus 52.1 seconds in controls, indicating impaired inhibitory control mechanisms and increased vulnerability to response conflict. Memory domain assessment demonstrated nuanced patterns of impairment. Rey Auditory Verbal Learning Test immediate recall averaged 6.2 words per trial in diabetic patients compared with 7.8 words in controls, while the learning slope across five trials showed attenuated acquisition in the diabetic group. Critically, delayed recall after 20 minutes revealed more pronounced deficits, with diabetic participants recalling mean 7.4 words versus 10.2 words in controls, suggesting consolidation or retrieval difficulties beyond initial encoding problems. Recognition discrimination indices remained relatively preserved, with diabetic individuals achieving 87 percent accuracy compared with 93 percent in controls, implying that retrieval cues substantially improved performance.

Visual memory assessment through Brief Visuospatial Memory Test-Revised demonstrated similar patterns, with immediate recall totaling 18.3 correct items across three trials in diabetic patients versus 22.7 in controls, while delayed recall showed 6.8 versus 9.1 items respectively. Verbal fluency examination revealed phonemic fluency means of 11.4 words per minute in diabetic individuals compared with 14.8 in controls, while semantic fluency averaged 16.2

versus 19.7 words, suggesting that strategic search processes mediated by executive systems suffered more than semantic network access per se. Correlation analyses demonstrated significant relationships between metabolic parameters and cognitive outcomes. Glycated hemoglobin levels correlated inversely with multiple cognitive measures, showing particularly strong associations with Trail Making Test Part B performance at r equals negative 0.52, Symbol Digit Modalities Test scores at r equals negative 0.48, and delayed verbal recall at r equals negative 0.44, all achieving statistical significance at p less than 0.001. Diabetes duration exhibited similar inverse relationships, with correlations of r equals negative 0.39 for executive function measures and r equals negative 0.36 for memory performance. Microvascular complication presence associated with worse cognitive outcomes across domains, with patients demonstrating two or more complications showing mean Montreal Cognitive Assessment scores of 22.8 compared with 25.1 in those without complications. Multiple regression analyses controlling for age, education, hypertension, and lipid parameters confirmed that glycated hemoglobin and disease duration independently predicted cognitive performance, with standardized beta coefficients of negative 0.34 and negative 0.28 respectively.

Discussion

The present findings corroborate and extend existing knowledge regarding cognitive manifestations of type 2 diabetes mellitus, while providing novel insights into domain-specific vulnerability patterns and their metabolic determinants. The documented executive function and working memory deficits align with neurobiological models implicating prefrontal-subcortical circuit dysfunction in diabetic cognitive impairment. Chronic hyperglycemia generates multiple pathophysiological cascades that compromise neural integrity, including advanced glycation end-product accumulation, oxidative stress amplification, inflammatory pathway activation, and microvascular endothelial dysfunction. These processes preferentially affect brain regions with high metabolic demands and dense vascular networks, particularly the hippocampus and prefrontal cortex, explaining the observed memory and executive function vulnerabilities. The documented processing speed reduction likely reflects white matter microstructural alterations, as diffusion tensor imaging studies have demonstrated reduced fractional anisotropy in diabetic populations, indicating myelin integrity compromise and axonal disruption. Such white matter pathology impairs the rapid information transfer necessary for efficient cognitive processing.

The strong inverse correlation between glycated hemoglobin and cognitive performance underscores the clinical importance of sustained glycemic control. Hemoglobin A1c represents integrated glycemic exposure over preceding months, providing a more stable metabolic index than point-in-time glucose measurements. The observed relationship suggests that cumulative

hyperglycemic burden drives cognitive deterioration through mechanisms including neuronal insulin resistance, impaired glucose transporter expression, and disrupted brain insulin signaling cascades. Insulin functions not merely as a metabolic hormone but also as a neuromodulator influencing synaptic plasticity, neurotransmitter release, and neuronal survival pathways. Chronic peripheral hyperinsulinemia combined with cerebral insulin resistance creates a paradoxical state where neurons experience functional insulin deficiency despite systemic insulin excess, compromising cognitive function maintenance. The documented association between microvascular complications and cognitive impairment suggests shared pathogenic mechanisms, with small vessel disease affecting both peripheral organs and cerebral microvasculature. Retinopathy and nephropathy presence may serve as clinically accessible biomarkers for cerebrovascular compromise and heightened cognitive risk. The relatively preserved recognition memory despite impaired free recall suggests that retrieval processes suffer more than encoding or storage mechanisms in diabetic cognitive impairment. This pattern differs from Alzheimer disease, where recognition typically deteriorates in parallel with recall, indicating distinct underlying pathophysiology. The diabetic cognitive profile more closely resembles vascular cognitive impairment patterns, consistent with the significant microvascular and macrovascular disease burden in this population. The disproportionate working memory impairment compared with simple span performance indicates that active information manipulation and executive control systems bear greater vulnerability than passive storage buffers, again implicating frontal-subcortical network dysfunction. These findings carry important clinical implications for diabetes management. Standard care protocols emphasize peripheral complication screening but rarely incorporate systematic cognitive assessment. The documented cognitive deficits, particularly in executive function domains, may significantly impair diabetes self-management capacity, creating a vicious cycle where cognitive impairment compromises treatment adherence, leading to worse glycemic control and accelerated cognitive decline.

Type 2 diabetes mellitus associates with a distinctive cognitive impairment profile characterized by executive dysfunction, working memory deficits, and processing speed reduction, reflecting frontal-subcortical circuit compromise. Glycemic control quality and disease chronicity emerge as modifiable determinants of cognitive outcomes, emphasizing the neurological benefits of metabolic optimization. These findings support incorporating routine cognitive screening into diabetes care protocols and prioritizing sustained glycemic control as a neuroprotective strategy.

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