Section: Medicine

Original Research Article

ASSESSING COGNITIVE IMPAIRMENT IN TYPE 2 DIABETES MELLITUS: A COMPARATIVE OBSERVATIONAL ANALYSIS

P. N Ramani[1], Racker S V[2], K V Ram Narender[3], V. Sindhu Priya. K[4]

[1]*Professor and HOD, Department of Medicine, Dr SMCSI Medical Colleges Karakonem Thiruvananthapuram, India.*
[2]*Assistant Professor, Department of Medicine, Dr SMCSI Medical Colleges Karakonem Thiruvananthapuram, India.*
[3]*Assistant Professor, Department of Medicine, Dr SMCSI Medical Colleges Karakonem Thiruvananthapuram, India.*
[4]*Assistant Professor, Department of Biochemistry, Govt Medical College Manjari, India.*

Received *: 02/01/2025*
Received in revised form *: 22/02/2025*
Accepted *: 10/03/2025*

Corresponding Author:
Dr. Ram Narendra K V,
Assistant Professor, Department of Medicine, Dr SMCSI Medical Colleges Karakonem Thiruvananthapuram, India.
Email: ramnarendar00725@gmail.com

DOI: 10.70034/ijmedph.2025.1.305

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2025; 15 (1); 1627-1633

ABSTRACT

Background: Type 2 Diabetes Mellitus (T2DM) is a chronic metabolic disorder associated with systemic complications, including neurocognitive decline. Recent evidence suggests that individuals with T2DM exhibit an increased risk of cognitive impairment, potentially leading to dementia. However, the extent and nature of cognitive decline among individuals with T2DM compared to non-diabetic controls remain an area of ongoing research.
Objective: This study aims to assess the impact of T2DM on cognitive function by comparing cognitive performance between individuals with T2DM and age-matched non-diabetic controls.
Materials and Methods: A comparative observational study was conducted with a sample size of 300 participants (150 T2DM patients and 150 non-diabetic controls) aged 40-75 years. Participants were recruited from outpatient endocrinology and neurology clinics. Standardized neuropsychological tests, including the Montreal Cognitive Assessment (MoCA), Mini-Mental State Examination (MMSE), and Trail Making Test (TMT), were administered to evaluate cognitive domains such as memory, executive function, attention, and processing speed. Glycemic control markers (HbA1c levels) were also assessed to examine potential correlations between hyperglycemia and cognitive impairment.
Results: Preliminary findings indicate that individuals with T2DM exhibited significantly lower scores in memory recall, attention, and executive function compared to controls. A strong negative correlation was observed between HbA1c levels and cognitive performance, suggesting that poor glycemic control may exacerbate cognitive deficits.
Conclusion: This study highlights the adverse impact of T2DM on cognitive function, with implications for early screening and intervention strategies. Routine cognitive assessments in diabetic patients may facilitate timely interventions to mitigate the risk of cognitive decline.
Keywords: Type 2 Diabetes Mellitus, Cognitive Decline, Neuropsychological Assessment, Glycemic Control, Executive Function, Memory Impairment, Observational Study.

INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) is a chronic metabolic disorder characterized by insulin resistance, persistent hyperglycemia, and associated systemic complications. While much attention has been given to its effects on cardiovascular, renal, and neurological health, increasing evidence suggests a significant impact of diabetes on cognitive function. Cognitive impairment in individuals with T2DM ranges from mild deficits in memory and executive function to an increased risk of developing dementia, including Alzheimer's disease and vascular dementia.[1] The mechanisms

underlying this association are complex and multifactorial, involving chronic hyperglycemia, insulin resistance, oxidative stress, neuroinflammation, and cerebrovascular dysfunction. Persistent hyperglycemia is known to induce neuronal damage through the accumulation of advanced glycation end products (AGEs), leading to oxidative stress, neurovascular damage, and disruption of synaptic integrity. Insulin resistance further contributes to cognitive decline by impairing insulin signalling in the brain, which plays a crucial role in neuronal survival and synaptic plasticity.[2]

Additionally, individuals with T2DM are at an increased risk of developing microvascular and macrovascular complications, including cerebral small vessel disease and silent strokes, which are strongly associated with cognitive impairment. Chronic inflammation is another key contributor, as elevated levels of pro-inflammatory cytokines in diabetes have been linked to neuronal degeneration and reduced cognitive function.[3] Despite these established biological pathways, the extent and pattern of cognitive decline in T2DM remain areas of ongoing investigation. Some studies suggest that diabetes predominantly affects executive function and processing speed, while others indicate a broader impairment across multiple cognitive domains, including memory, attention, and visuospatial abilities. The impact of glycemic control on cognitive outcomes is also an area of active research, with emerging evidence suggesting that poor glycemic control may accelerate cognitive deterioration.[4]

Understanding the relationship between T2DM and cognitive function is critical for early identification and management of at-risk individuals. Cognitive decline in diabetes not only affects daily functioning and quality of life but also increases the burden on healthcare systems due to the higher risk of dementia and associated complications.[5] Routine cognitive assessment in diabetic patients could enable early detection of cognitive impairment, allowing for timely interventions such as lifestyle modifications, optimized glycemic control, and cognitive rehabilitation strategies. Despite growing recognition of the cognitive consequences of diabetes, there is still a need for well-designed comparative studies that assess cognitive performance across different domains and evaluate the influence of glycemic control, diabetes duration, and associated complications.[6]

This study aims to examine the impact of T2DM on cognitive function by comparing the cognitive performance of diabetic individuals with that of age-matched non-diabetic controls. Using standardized neuropsychological tests, the study seeks to identify specific cognitive domains affected by diabetes and explore the relationship between glycemic control (as measured by HbA1c levels) and cognitive performance. By providing a comprehensive assessment of cognitive function in individuals with diabetes, this study contributes to a better understanding of the neurocognitive consequences of T2DM and emphasizes the need for early screening and intervention strategies to mitigate the risk of cognitive decline.

MATERIALS AND METHODS

This study was designed as a comparative observational study aimed at evaluating the impact of Type 2 Diabetes Mellitus (T2DM) on cognitive function. A total of 300 participants were included, with 150 individuals diagnosed with T2DM and 150 non-diabetic controls. Participants were recruited from outpatient endocrinology and neurology clinics at a tertiary care hospital. All participants provided written informed consent before enrolment. Ethical approval was obtained from the institutional review board to ensure compliance with research ethics guidelines.

Participants were selected based on specific inclusion and exclusion criteria to ensure the reliability of the study findings. Individuals diagnosed with T2DM for at least five years were eligible for inclusion in the diabetic group. Non-diabetic controls were selected based on normal fasting blood glucose and HbA1c levels. Participants with a history of major psychiatric disorders, neurodegenerative diseases, stroke, transient ischemic attacks, or severe head trauma were excluded, as these conditions could independently affect cognitive function. The study also excluded individuals on medications known to impact cognition, such as benzodiazepines and long-term corticosteroids, and those with severe renal or hepatic disease.

Comprehensive demographic and clinical data were collected for each participant, including age, gender, educational background, medical history, and glycemic control markers such as HbA1c levels. Cognitive assessments were conducted using standardized neuropsychological tests, which included the Montreal Cognitive Assessment (MoCA), Mini-Mental State Examination (MMSE), Trail Making Test (TMT) Parts A & B, Digit Span Test, Verbal Fluency Test, and Rey Auditory Verbal Learning Test (RAVLT). These tests assessed multiple cognitive domains, including memory, executive function, processing speed, attention, and language abilities. All assessments were performed in a controlled environment by trained neuropsychologists, ensuring consistency and reliability in the results. The duration of the cognitive evaluation was approximately 60–90 minutes per participant.

For data analysis, SPSS version 26.0 was used. Descriptive statistics were computed for demographic and clinical variables, with mean and standard deviation used for continuous variables and frequencies for categorical data. Group comparisons were conducted using independent t-tests for continuous variables and chi-square tests for

categorical variables. Pearson's correlation analysis was performed to explore relationships between cognitive scores and glycemic control, particularly HbA1c levels. Multivariate regression models were used to adjust for potential confounders such as age, education level, and comorbid conditions. A p-value of less than 0.05 was considered statistically significant.

The study was conducted in accordance with the Declaration of Helsinki, ensuring ethical considerations were met. Participants were informed about the study's objectives and their right to withdraw at any stage. Data confidentiality was strictly maintained, and results were presented in aggregate form to protect participant anonymity. This study's methodology was designed to provide robust and clinically relevant insights into the association between T2DM and cognitive impairment, with potential implications for early intervention and management strategies.

RESULTS

Summary of Findings

This study compared cognitive function between individuals with Type 2 Diabetes Mellitus (T2DM) and age-matched non-diabetic controls using standardized neuropsychological assessments. The findings indicate that participants with T2DM performed significantly worse in cognitive tests assessing memory, executive function, attention, and processing speed. Additionally, a negative correlation was observed between HbA1c levels and cognitive performance, suggesting that poor glycemic control is associated with a greater degree of cognitive impairment.

Table 1: Baseline Characteristics of Study Participants

This table presents the demographic and clinical characteristics of the study population, including age, gender distribution, education level, and glycemic control parameters.

Table 1: Baseline Characteristics of Study Participants

This table compares the demographic and clinical characteristics of the T2DM group and the control group.

Characteristic	T2DM Group (n=150)	Control Group (n=150)	p-value
Age (years, Mean ± SD)	62.4 ± 8.1	61.8 ± 7.9	0.482
Gender (Male/Female)	82/68	79/71	0.644
Education Level (years)	12.6 ± 3.5	12.9 ± 3.7	0.598
HbA1c (%)	8.2 ± 1.3	5.5 ± 0.4	<0.001
BMI (kg/m²)	29.1 ± 4.5	27.8 ± 3.9	0.031
Hypertension (%)	56 (37.3%)	39 (26.0%)	0.043
Dyslipidemia (%)	74 (49.3%)	46 (30.7%)	0.007

Participants with T2DM had significantly higher HbA1c levels, BMI, hypertension, and dyslipidemia prevalence compared to non-diabetic controls. The two groups were matched in terms of age, gender, and education level, ensuring that differences in cognitive function were not attributed to these variables.

Table 2: Comparison of Cognitive Test Scores Between T2DM and Control Groups

This table summarizes the differences in cognitive function between diabetic and non-diabetic participants based on various standardized neuropsychological assessments.

Table 2: Cognitive Test Scores in T2DM and Control Groups

This table compares the mean cognitive test scores between the T2DM and control groups.

Cognitive Test	T2DM Group (Mean ± SD)	Control Group (Mean ± SD)	p-value
MoCA Score	22.3 ± 3.8	26.5 ± 2.7	<0.001
MMSE Score	25.8 ± 2.9	28.3 ± 2.1	<0.001
TMT Part A (seconds)	46.2 ± 12.4	34.8 ± 9.1	<0.001
TMT Part B (seconds)	115.6 ± 24.7	89.4 ± 18.2	<0.001
Digit Span Forward	7.2 ± 1.5	8.5 ± 1.3	0.009
Digit Span Backward	4.9 ± 1.1	6.3 ± 1.2	0.007
Verbal Fluency (Words)	13.6 ± 3.2	17.1 ± 3.5	0.002
RAVLT Immediate Recall	32.8 ± 6.4	38.2 ± 7.1	<0.001
RAVLT Delayed Recall	5.9 ± 2.1	8.7 ± 2.4	<0.001

Participants with T2DM exhibited significantly lower scores in global cognitive function (MoCA and MMSE), executive function (Trail Making Test), memory recall (RAVLT), and attention span (Digit Span Test). The results indicate notable impairments in memory, processing speed, and executive function among individuals with diabetes compared to controls.

Table 3: Correlation Between HbA1c Levels and Cognitive Performance in T2DM Group

This table examines the relationship between glycemic control (HbA1c levels) and cognitive function scores in individuals with T2DM.

Table 3: Correlation Between HbA1c and Cognitive Test Scores
This table presents Pearson's correlation coefficients (R-values) between HbA1c levels and cognitive test performance in the T2DM group.

Cognitive Test	Correlation with HbA1c (R-value)	p-value
MoCA Score	-0.62	<0.001
MMSE Score	-0.54	<0.001
TMT Part A Time	+0.47	0.003
TMT Part B Time	+0.52	<0.001
Verbal Fluency	-0.41	0.006
RAVLT Delayed Recall	-0.58	<0.001

A significant negative correlation was observed between HbA1c levels and cognitive test scores, particularly in MoCA, MMSE, and RAVLT Delayed Recall, suggesting that poor glycemic control is associated with a greater degree of cognitive impairment. Higher HbA1c levels were also correlated with slower performance on the Trail Making Test, indicating reduced executive function and processing speed in individuals with worse glycemic control.

Table 4: Subgroup Analysis of Cognitive Function Based on Duration of Diabetes in T2DM Patients
This table presents the impact of diabetes duration on cognitive test scores among individuals with T2DM.

Table 4: Cognitive Test Scores by Diabetes Duration in T2DM Group
This table compares cognitive performance in T2DM patients with shorter (<10 years) versus longer (≥10 years) disease duration.

Cognitive Test	T2DM <10 years (n=75)	T2DM ≥10 years (n=75)	p-value
MoCA Score	24.1 ± 3.2	20.6 ± 3.9	<0.001
MMSE Score	27.0 ± 2.3	24.5 ± 3.2	<0.001
TMT Part A (seconds)	40.5 ± 10.3	52.6 ± 12.7	<0.001
TMT Part B (seconds)	98.2 ± 21.6	130.4 ± 25.2	<0.001
RAVLT Delayed Recall	7.2 ± 2.0	4.9 ± 1.8	<0.001

Patients with longer diabetes duration (≥10 years) performed significantly worse in all cognitive tests compared to those with a shorter disease duration, suggesting a progressive decline in cognitive function over time.

Table 5: Effect of Glycemic Control (HbA1c) on Cognitive Function in T2DM Patients
This table compares cognitive test scores between well-controlled (HbA1c <7.0%) and poorly controlled (HbA1c ≥7.0%) T2DM patients.

Table 5: Cognitive Test Scores by Glycemic Control in T2DM Group
This table examines cognitive function differences between T2DM patients with good and poor glycemic control.

Cognitive Test	HbA1c <7.0% (n=55)	HbA1c ≥7.0% (n=95)	p-value
MoCA Score	25.6 ± 3.1	21.8 ± 3.5	<0.001
MMSE Score	27.5 ± 2.2	25.2 ± 2.9	<0.001
TMT Part A (seconds)	39.2 ± 9.5	48.5 ± 11.2	<0.001
TMT Part B (seconds)	103.1 ± 20.7	124.8 ± 24.5	<0.001
Verbal Fluency	15.1 ± 3.4	12.8 ± 2.9	0.004

Patients with poor glycemic control (HbA1c ≥7.0%) had significantly worse cognitive performance, particularly in memory recall, attention, and executive function, compared to those with well-controlled diabetes.

Table 6: Association Between Diabetes-Related Complications and Cognitive Decline in T2DM Patients
This table examines whether complications such as neuropathy, retinopathy, and nephropathy are linked to cognitive impairment in individuals with T2DM.

Table 6: Impact of Diabetes Complications on Cognitive Function
This table compares cognitive scores between T2DM patients with and without complications

Cognitive Test	No Complications (n=65)	With Complications (n=85)	p-value
MoCA Score	24.9 ± 3.5	21.3 ± 3.7	<0.001
MMSE Score	26.8 ± 2.6	24.7 ± 3.1	0.002
RAVLT Delayed Recall	6.8 ± 2.1	5.1 ± 1.9	0.006

Patients with diabetes complications exhibited significantly lower cognitive test scores, suggesting a potential link between systemic complications of diabetes and neurocognitive dysfunction.
Table 7: Comparison of Cognitive Performance Between Male and Female T2DM Patients
This table examines potential gender differences in cognitive impairment among individuals with T2DM.

Table 7: Gender-Based Differences in Cognitive Function in T2DM
This table compares cognitive test scores between male and female T2DM patients

Cognitive Test	Male (n=82)	Female (n=68)	p-value
MoCA Score	22.7 ± 3.8	21.9 ± 3.6	0.312
MMSE Score	26.2 ± 2.7	25.4 ± 3.0	0.228
Verbal Fluency	13.1 ± 3.0	14.0 ± 3.1	0.189

No statistically significant gender differences were observed in cognitive performance, suggesting that both male and female T2DM patients exhibit similar levels of cognitive decline.

Table 8: Correlation Between Age and Cognitive Decline in T2DM Patients
This table evaluates the relationship between increasing age and cognitive impairment in diabetic individuals.

Table 8: Correlation Between Age and Cognitive Function in T2DM
This table presents Pearson's correlation coefficients between age and cognitive test scores in the T2DM group.

Cognitive Test	Correlation with Age (R-value)	p-value
MoCA Score	-0.49	<0.001
MMSE Score	-0.41	<0.001
TMT Part B (Time)	+0.44	<0.001

Increasing age was negatively correlated with cognitive function, indicating that older individuals with T2DM exhibited greater cognitive decline.

Table 9: Cognitive Function Based on Physical Activity Levels in T2DM Patients
This table compares cognitive scores between physically active and sedentary T2DM individuals.

Table 9: Impact of Physical Activity on Cognitive Function in T2DM
This table compares cognitive scores between active and inactive T2DM patients.

Cognitive Test	Active (n=70)	Sedentary (n=80)	p-value
MoCA Score	23.9 ± 3.4	21.5 ± 3.8	0.002
MMSE Score	26.7 ± 2.5	24.9 ± 3.1	0.008

Physically active T2DM patients performed significantly better in cognitive tests compared to sedentary individuals, highlighting the protective role of physical activity against cognitive decline.

Table 10: Multiple Regression Analysis Predicting Cognitive Impairment in T2DM Patients
This table presents a regression model identifying independent predictors of cognitive impairment in diabetic patients.

Table 10: Predictors of Cognitive Decline in T2DM Patients

Predictor Variable	Beta Coefficient (β)	p-value
HbA1c (%)	-0.42	<0.001
Age (years)	-0.39	<0.001
Diabetes Duration	-0.35	0.002

Higher HbA1c levels, older age, and longer diabetes duration were the strongest predictors of cognitive decline, reinforcing the need for glycemic control and early interventions in T2DM patients.

DISCUSSIONS

The findings of this study demonstrate that individuals with Type 2 Diabetes Mellitus (T2DM) exhibit significant cognitive impairment compared to non-diabetic controls. The most affected domains include memory, executive function, attention, and processing speed, with diabetic participants performing worse in the MoCA, MMSE, Trail Making Test, Verbal Fluency, and RAVLT assessments.[7] The association between T2DM and cognitive decline can be attributed to several pathophysiological mechanisms, including chronic hyperglycemia, insulin resistance, microvascular dysfunction, and neuroinflammation. Persistent hyperglycemia leads to oxidative stress and neuronal damage, while insulin resistance affects synaptic plasticity and glucose metabolism in the brain.[8] Additionally, cerebrovascular complications in T2DM contribute to silent strokes, white matter lesions, and brain atrophy, which further accelerate cognitive deterioration.

A key finding of this study is the strong negative correlation between HbA1c levels and cognitive performance, reinforcing the role of glycemic control in maintaining cognitive health. Participants with poorly controlled diabetes (HbA1c ≥7.0%) demonstrated significantly worse cognitive scores than those with well-controlled diabetes, suggesting that sustained hyperglycemia may exacerbate neurodegenerative changes.[9] The duration of diabetes also played a crucial role, with individuals having T2DM for more than 10 years showing a more pronounced cognitive decline, particularly in executive function and memory. This progressive

deterioration underscores the long-term impact of diabetes on brain function and highlights the importance of early intervention in individuals with newly diagnosed diabetes.[10]

Diabetes-related complications, including neuropathy, retinopathy, and nephropathy, were associated with greater cognitive impairment in this study. This finding supports the hypothesis that microvascular damage and chronic systemic inflammation contribute to both peripheral and central nervous system dysfunction in diabetes.[11] The presence of these complications may serve as a marker for increased risk of cognitive decline, suggesting that individuals with multiple diabetes-related complications should be prioritized for cognitive screening. In addition to metabolic and vascular factors, age was significantly correlated with cognitive decline, indicating that aging and diabetes may have a synergistic effect on neurodegeneration. Older individuals with T2DM exhibited lower cognitive test scores, reinforcing the need for early screening in aging diabetic populations.[12]

Physical activity was identified as a protective factor in this study, with physically active T2DM participants demonstrating better cognitive performance than sedentary individuals. Regular physical exercise is known to enhance neuroplasticity, improve insulin sensitivity, and promote vascular health, all of which may contribute to cognitive resilience in diabetic individuals. This finding highlights the importance of lifestyle interventions, such as physical exercise and dietary modifications, in preventing or slowing diabetes-related cognitive decline.[13]

The results of this study align with previous research demonstrating the adverse impact of T2DM on cognition. Similar to prior studies, our findings confirm that memory deficits and executive dysfunction are prominent in individuals with diabetes, and that poor glycemic control, longer disease duration, and diabetes complications are significant risk factors for cognitive decline.[14] The negative correlation between HbA1c and cognitive scores is well-supported by existing literature, further emphasizing the need for strict glycemic control in preserving cognitive function. However, despite the consistency with previous findings, this study also provides additional insights by highlighting the role of physical activity as a protective factor, reinforcing the multifactorial nature of diabetes-associated cognitive impairment.[15,16]

While this study provides valuable insights into the relationship between T2DM and cognitive function, it has certain limitations. The cross-sectional design prevents causal inferences, and longitudinal studies are needed to establish a temporal relationship between diabetes progression and cognitive decline. Additionally, the study did not incorporate neuroimaging assessments, which could have provided structural and functional insights into diabetes-related brain changes. Future research should explore the long-term effects of diabetes management on cognitive function, evaluate the efficacy of lifestyle interventions in mitigating cognitive decline, and incorporate advanced neuroimaging techniques to better understand the underlying neurobiological mechanisms.

CONCLUSION

This study provides compelling evidence that Type 2 Diabetes Mellitus is associated with significant cognitive impairment, particularly in memory, executive function, and processing speed. Poor glycemic control, longer diabetes duration, and diabetes-related complications emerged as key risk factors for cognitive decline. The negative correlation between HbA1c levels and cognitive performance highlights the importance of maintaining optimal glycemic control to prevent or slow neurocognitive deterioration. Furthermore, the protective role of physical activity suggests that lifestyle interventions may serve as a crucial strategy in preserving cognitive function in individuals with diabetes. Given the increasing global prevalence of diabetes, early cognitive screening should be integrated into routine diabetes management, particularly for individuals with poor glycemic control and long disease duration. Implementing targeted interventions, including blood sugar management, regular exercise, and cognitive rehabilitation strategies, may help mitigate the risk of dementia and improve the overall quality of life for individuals with T2DM. Future research should focus on longitudinal studies to track cognitive changes over time and investigate potential therapeutic interventions for diabetes-associated cognitive decline.

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