Original Article

Association Between Metabolic Syndrome Severity and Cognitive Function in Adults: A Cross-Sectional Study

Jyothula Tripura Lakshmi[1], Shashikant Verma[2], Malini Kulshrestha[3], Rakesh Yaduvanshi[4]

[1] PhD Scholar, Department of Physiology, Rohilkhand Medical College and Hospital, Bareilly, Uttar Pradesh, India. [2]Professor & Head, Department of Physiology, Rohilkhand Medical College and Hospital, Bareilly, Uttar Pradesh, India. [3]Professor, Department of General Medicine, Rohilkhand Medical College and Hospital, Bareilly, Uttar Pradesh, India. [4]Professor, Department of Psychiatry, Rohilkhand Medical College and Hospital, Bareilly, Uttar Pradesh, India.

Abstract

Background: Metabolic syndrome (MetS) is a cluster of metabolic abnormalities, including central obesity, hypertension, hyperglycaemia, hypertriglyceridaemia and low level of high-density lipoprotein cholesterol (HDL-C) that are linked to higher cardiovascular and metabolic risk. Recent studies have shown that metabolic syndrome could have detrimental effects on cognition via vascular, inflammatory and metabolic pathways. The aim of the present study was therefore to assess if there was any relationship between worsening metabolic syndrome and cognitive function among adults. **Material and Methods:** The present study was cross sectional and observational study among adults aged 25-65 years having metabolic syndrome in a tertiary care centre of Bareilly, Uttar Pradesh, India. The criteria used for the diagnosis of metabolic syndrome were modified NCEP ATP III criteria. All the anthropometric measurements, blood pressure, fasting blood glucose, triglycerides, and HDL-C were evaluated according to standard methods. The Hindi version of Mini Mental State Examination (HMSE) was used to assess cognitive function. Spearman correlation was used to determine the relationship of components of metabolic syndrome with MMSE score and Kruskal–Wallis test was used for comparison between groups. **Results:** Waist circumference, fasting blood glucose, triglycerides, and systolic blood pressure demonstrated significant negative correlations with MMSE score, whereas HDL-C showed a significant positive correlation. A progressive decline in MMSE score was observed with increasing number of metabolic syndrome components (24.10 ± 1.85 in subjects with three components, 22.55 ± 2.46 with four components, and 20.76 ± 3.10 with five components; $p < 0.001$). Mild to moderate cognitive impairment was observed in 49.7% of participants, while severe cognitive impairment was present in 4.7% of subjects. **Conclusion:** Increasing severity of metabolic syndrome was significantly associated with worsening cognitive function. The findings suggest that cumulative metabolic abnormalities may adversely affect cognition and highlight the importance of early identification and management of metabolic risk factors to reduce cognitive decline.

Keywords: MetS – Metabolic Syndrome; MMSE – Mini-Mental State Examination; HMSE – Hindi Mental State Examination; BMI – Body Mass Index; HDL-C – High Density Lipoprotein Cholesterol; NCEP ATP III – National Cholesterol Education Program Adult Treatment Panel III.

Received: 01 June 2026 **Revised: 20 June 2026** **Accepted: 13 July 2026** **Published: 17 July 2026**

Introduction

Metabolic syndrome (MetS) is a constellation of interrelated metabolic abnormalities characterized by central obesity, hypertension, hyperglycemia, hypertriglyceridemia, and reduced high-density lipoprotein cholesterol (HDL-C), which collectively increase the risk of cardiovascular disease, type 2 diabetes mellitus, and premature mortality.[1] Studies have found insulin resistance to be the primary pathogenic mechanisms of MetS, and the secondary mechanisms of chronic low-grade inflammation, oxidative stress, dysfunction of endothelial cells, and alteration of adipokines play important roles in the pathogenesis and progression of metabolic abnormalities associated with MetS.[2]

Metabolic syndrome is a high public health problem in India as its prevalence ranges from 10% to 56% of the population studied and diagnostic criteria applied.[3] There is a tendency for higher prevalence to be seen in the urban area, female and middle aged. The trend of Indian population to obesity and diabetes mellitus have also added up to the growing incidence of metabolic syndrome and its related complications.[4]

The cardiovascular and metabolic effects of MetS are known, but there is growing evidence that metabolic syndrome can have a negative impact on cognitive functioning as well. Cognitive function is composed of various higher mental processes including memory, attention, language, orientation, executive function, calculation and visuospatial ability that are necessary for normal day to day life and quality of life. Cognitive

Address for correspondence: Dr. Jyothula Tripura Lakshmi, PhD Scholar, Department of Physiology, Rohilkhand Medical College and Hospital, Bareilly, Uttar Pradesh, India.
E-mail: tripurajyothula123@gmail.com

DOI:
10.21276/amit.2026.v13.i2.835

How to cite this article: Lakshmi JT, Verma S, Kulshrestha M, Yaduvanshi R. Association Between Metabolic Syndrome Severity and Cognitive Function in Adults: A Cross-Sectional Study. Acta Med Int. 2026;13(2):1121-1125.

dysfunction related to metabolic syndrome has been identified as an emerging health disorder as subtle cognitive decline may happen even prior to the presence of dementia.[5] Several pathophysiological mechanisms have been associated with the individual components of metabolic syndrome and likely are involved in the brain dysfunction associated with metabolic syndrome. Advanced glycation end-product accumulation, oxidative neuronal damage, diminished insulin action, hippocampal dysfunction and cerebral microangiopathy may be associated with chronic hyperglycemia.[6] Endothelial dysfunction, decreased cerebral blood flow, white matter ischemic change and impaired autoregulation of cerebral blood flow are all related to hypertension.[7] Dyslipidemia, especially high triglycerides and low HDL cholesterol, can fuel inflammation and oxidative stress in the vessels and result in neurodegeneration and reduced synaptic plasticity.[8] Central obesity is also a metabolically active inflammatory condition and it has been linked to higher level of production of pro-inflammatory cytokines and adipokines, which may have negative effects on neuronal integrity and cognition.[9]

The metabolic abnormalities may act independently in causing cognitive dysfunction; however, the impact when multiple abnormalities exist may be more deleterious because of cumulative metabolic burden. It is likely that the joint effect of several metabolic abnormalities has a synergistic negative effect on cognition.[10] Increasing number of metabolic syndrome components has been associated with worsening metabolic abnormalities and vascular dysfunction, which may accelerate cognitive decline. Early cognitive impairment associated with MetS may remain clinically unnoticed for prolonged periods, thereby increasing the future risk of dementia and reduced quality of life.[6]

Despite increasing recognition of this association, studies evaluating the relationship between metabolic syndrome severity and cognitive function in the Indian population remain limited. Most available studies have focused primarily on the presence or absence of metabolic syndrome rather than assessing the effect of increasing number of metabolic syndrome components on cognitive function. Understanding this relationship may help in early identification of individuals at higher risk of cognitive decline and facilitate timely preventive interventions. Therefore, the present study was undertaken to evaluate cognitive function in adults with metabolic syndrome and to assess its association with increasing severity of metabolic syndrome.

Materials and Methods

Study design, study setting and sample size: The present cross-sectional observational study was conducted in a tertiary care centre, Bareilly, Uttar Pradesh, India. The study was carried out after obtaining approval from the Institutional Ethics Committee, and written informed consent was obtained from all participants prior to inclusion in the study in accordance with the ethical principles of the Declaration of Helsinki. The study included 380 adults (The sample size was calculated using mean and standard deviation values obtained from a previous similar study) including 179 (47.1%) males and 201 (52.9%) females aged between 25 and 65 years who attended the Medicine Outpatient Department and fulfilled the diagnostic criteria for metabolic syndrome according to the modified National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) criteria.[11]

Metabolic syndrome was diagnosed in subjects having any three or more of the following components:

- Central obesity (waist circumference ≥90 cm in men and ≥80 cm in women)
- Serum triglycerides ≥150 mg/dL
- HDL-C <40 mg/dL in men and <50 mg/dL in women
- Blood pressure ≥130/85 mmHg or current antihypertensive treatment
- Fasting blood glucose ≥100 mg/dL or use of antidiabetic medication

Inclusion and Exclusion Criteria:

Both male and female subjects aged 25–65 years diagnosed with metabolic syndrome were included in the study. Subjects with pre-existing chronic respiratory diseases, pregnancy, neurological disorders, psychiatric illness, or history of intake of drugs known to affect cognitive function were excluded from the study.

Anthropometric and Clinical Assessment: Height and weight were measured with subjects wearing light clothing and no footwear. Body mass index (BMI) was calculated as weight in kilograms divided by height in meter square (kg/m²). Waist circumference was measured midway between the lower costal margin and iliac crest at the end of normal expiration, while hip circumference was measured at the level of maximum gluteal prominence. Waist-hip ratio was subsequently calculated. Blood pressure was measured in sitting posture using a calibrated mercury sphygmomanometer after at least 5 minutes of rest. Two readings were recorded at an interval of 5 minutes and the average value was considered for analysis.

Biochemical Parameters Assessment: All participants were instructed to report after overnight fasting of 8–12 hours. Venous blood samples were collected under aseptic precautions for estimation of fasting blood glucose, triglycerides, and HDL cholesterol using standard enzymatic methods in the central laboratory. Fasting blood glucose was estimated by glucose oxidase-peroxidase method, triglycerides by glycerol phosphate oxidase-peroxidase method, and HDL-C by direct enzymatic method.

Assessment of Cognitive Function: Cognitive function was assessed using the Hindi version of the Mini-Mental State Examination (HMSE), a validated 30-point questionnaire designed to evaluate orientation, registration, attention, calculation, recall, language, and visuospatial ability.[12] The assessment was performed in a quiet environment through face-to-face interview. Based on MMSE scores, subjects were categorized as normal cognition (24–30), mild to moderate cognitive impairment (18–23), and severe cognitive impairment (<18).[13]

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using SPSS version 26.0. Spearman correlation analysis was used to assess the association between metabolic syndrome components and MMSE score, and comparison among groups

was performed using the Kruskal–Wallis test. A p-value <0.05 was considered statistically significant.

Results

[Table 1] shows the baseline demographic and anthropometric characteristics of the study participants. The study population predominantly consisted of middle-aged adults with increased body mass index and central obesity, indicating a high prevalence of overweight and abdominal adiposity among subjects with metabolic syndrome.

Table 1: Baseline demographic and anthropometric characteristics of study participants

Parameters/components	Mean ± SD
Age (year)	51.60 ± 9.02
Height (m)	1.61 ± 0.087
Weight (kg)	75.51 ± 10.85
Body Mass Index (kg/m²)	29.22 ± 4.11
Waist Circumference (cm)	97.92 ± 9.60
Hip Circumference (cm)	97.56 ± 8.49

Table 2: Metabolic syndrome components data of study participants

Parameters/components	Mean ± SD
Waist Circumference (cm)	97.92 ± 9.60
Systolic Blood Pressure (mmHg)	140.34 ± 15.36
Diastolic Blood Pressure (mmHg)	89.82 ± 9.51
Fasting Blood Glucose (mg/dL)	131.17 ± 31.47
Triglycerides (mg/dL)	176.60 ± 50.52
HDL-C (mg/dL)	45.76 ± 6.58

[Table 2] depicts the metabolic profile of study participants. Elevated fasting blood glucose, triglyceride levels, blood pressure, and increased waist circumference were observed among study subjects, reflecting the presence of significant metabolic abnormalities.

Table 3: Correlation between Metabolic Syndrome Components and MMSE Score

Variable	MMSE (r)	p value
Waist Circumference (cm)	-0.150	0.003
Fasting Blood Glucose (mg/dL)	-0.106	0.039
Triglycerides (mg/dL)	-0.130	0.011
HDL-C (mg/dL)	0.185	<0.001
Systolic Blood Pressure (mmHg)	-0.107	0.037
Diastolic Blood Pressure (mmHg)	-0.082	0.11

* Spearman correlation, p <0.05 (statistically significant); Negative correlation coefficient indicates inverse association with MMSE score.

[Table 3] demonstrates the correlation between metabolic syndrome components and MMSE score. Waist circumference, fasting blood glucose, triglycerides, and systolic blood pressure showed significant negative correlations with MMSE score, whereas HDL-C demonstrated a significant positive correlation, indicating worsening cognitive performance with increasing metabolic abnormalities.

[Table 4 and Figure 1] shows comparison of MMSE scores according to metabolic syndrome severity. A significant progressive decline in MMSE score was observed with increasing number of metabolic syndrome components, suggesting a dose-dependent adverse effect of metabolic burden on cognitive function.

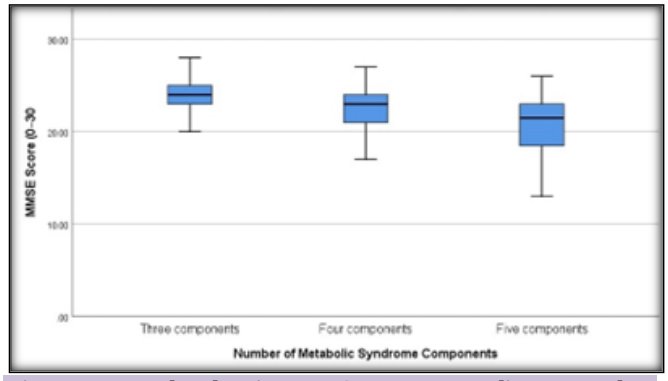

Figure 1: Box plot showing MMSE score according to number of metabolic syndrome components

Table 4: Comparison of MMSE scores according to metabolic syndrome severity

Parameter	Three components	Four components	Five components	P Value
MMSE Score (0–30)	24.10 ± 1.85	22.55 ± 2.46	20.76 ± 3.10	<0.001

*Kruskal–Wallis test, p < 0.05 (statistically significant), data was presented as mean ± SD

Table 5: Distribution of Cognitive Function (MMSE Score) among Study Participants

		Frequency	Percent
Cognitive function (MMSE Score)	Mean ± SD, Range	22.98 ± 2.63 (13 – 28)	
	Normal	173	45.5
	Mild to moderate Impairment	189	49.7

	Severe Impairment	18	4.7
Total		380	100.0

[Table 5] shows the distribution of cognitive function among study participants. Mild to moderate cognitive impairment was observed in the majority of subjects, while severe cognitive impairment was present in a smaller proportion of participants.

Discussion

The present study was conducted to evaluate cognitive function in adults with metabolic syndrome and to assess its association with increasing severity of metabolic syndrome. The findings of the study demonstrated a significant association between metabolic abnormalities and cognitive performance. MMSE score decreased in proportion to the number of metabolic syndrome components, which indicated that higher metabolic syndrome levels may have a negative impact on mental performance.

In the current study, the majority of the study population were middle aged adults with elevated BMI and central obesity which inherently represents the high prevalence of metabolic syndrome-related metabolic abnormalities among patients with metabolic syndrome. Gupta et al. (2004) showed high rates of obesity and abdominal adiposity among Indian adults with metabolic syndrome.[4] The central obesity has been associated with insulin resistance, systemic inflammation, endothelial dysfunction and changes in adipokine secretion which could negatively affect the functioning of neurons and brain metabolism.[14] Panza et al. (2010) also hypothesized that inflammatory and vascular conditions associated with obesity can speed up the neurodegenerative process leading to a decrease in cognitive function.[15]

In the present study, MMSE score was observed to have significant negative correlations with waist circumference, fasting blood glucose, triglycerides, and systolic blood pressure, and significant positive correlation with HDL-C, which showed strong correlation with cognitive performance. The same results were seen in the study conducted by Yates et al., 2012, which showed a negative effect of obesity, dyslipidemia, hypertension, and insulin resistance on cognitive function via vascular and inflammatory mechanisms.[9] Frisardi et al. (2010) also found that oxidative stress and endothelial dysfunction are related to metabolic syndrome-related metabolic disturbances and their effects on brain structure and cognition.[5]

The inverse relationship of fasting blood glucose and MMSE score found in the present study is also consistent with the results of Biessels and Reagan (2015), who showed that chronic hyperglycemia and insulin resistance have a negative effect on hippocampal function and neuronal signaling, as well as cerebral glucose metabolism, which all lead to memory impairment and cognitive decline.[6] Craft S (2006) also proposed that insulin resistance and impaired cerebral insulin signaling may significantly contribute to neurodegeneration and memory dysfunction.[16]

The significant negative association between waist circumference and MMSE score further supports the role of central obesity in cognitive dysfunction. Similar findings were observed by Kiliaan et al. (2014), who reported that visceral adiposity and adipokine-mediated inflammation may adversely affect neuronal integrity and cerebral function.[8]

The present study also demonstrated significant inverse association between triglyceride levels and MMSE score, whereas HDL-C showed a positive association with cognitive decline. Dyslipidemia may contribute to vascular inflammation, oxidative stress, endothelial dysfunction, and impaired neuronal signaling, thereby adversely affecting cognitive performance. Similar findings were reported by Yaffe et al. (2009), who observed increased cognitive decline among individuals with clustering of dyslipidemia and metabolic abnormalities.[10]

Systolic blood pressure showed a significant negative correlation with MMSE score, whereas diastolic blood pressure did not demonstrate statistically significant association. Similar observations were reported by Iadecola C and Davisson RL (2008), who demonstrated that chronic hypertension contributes to cerebrovascular dysfunction, white matter ischemic changes, and impaired cerebral autoregulation affecting cognitive performance.[7] However, Raffaitin et al. (2009) did not observe significant association between certain metabolic syndrome components and incident dementia after adjustment for vascular risk factors and educational status.[17]

One of the major findings of the present study was the significant progressive decline in MMSE scores with increasing number of metabolic syndrome components. Subjects having five components of metabolic syndrome showed a lower MMSE score than subjects having three and four components of metabolic syndrome, suggesting that the more severe the metabolic syndrome, the worse the cognitive test score. Increasing clustering of metabolic abnormalities has been shown to be related to higher risks of cognitive impairment and accelerated cognitive decline, as reported by Yaffe et al. (2009).[10] In addition, Ng et al., (2016) reported that metabolic syndrome, as a factor was found to be significantly associated with MCI and dementia among older adults.[18]

The present study highlighted the fact that almost half of the study population had mild to moderate cognitive impairment, and a smaller portion of the subjects had severe cognitive impairment. The same finding was reported by Forti et al. (2010) who reported significantly lower cognitive scores among people with metabolic syndrome, especially in those with abdominal obesity and hyperglycemia.[19]

In general, the current study results indicated that the degree of metabolic syndrome is correlated with worsening cognitive function. These co-existing comorbidities of obesity, hyperglycemia, dyslipidaemia, and hypertension have been shown to have interrelated inflammatory, vascular, and metabolic effects on cognition that could be harmful to cognitive function. Early detection of cognitive dysfunction in both individuals with metabolic syndrome and healthy individuals could then enable early preventive and therapeutic measures to be taken.

Conclusion

In the present study, cognitive function was significantly associated with the severity of MS. The number of metabolic syndrome components significantly decreased scores of the MMSE, suggesting that the cognitive performance worsened considerably as the number of components increased. The need for early recognition and treatment of metabolic abnormalities to prevent cognitive decline is highlighted by these results.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Alberti KGMM, Zimmet P, Shaw J. The metabolic syndrome—a new worldwide definition. Lancet. 2005;366(9491):1059-62.
2. Rochlani Y, Pothineni NVK, Kovelamudi S, Mehta JL. Metabolic syndrome: pathophysiology, management, and modulation by natural compounds. TherAdvCardiovasc Dis. 2017;11(8):215-25.
3. Prasad DS, Kabir Z, Dash AK, Das BC. Prevalence and risk factors for metabolic syndrome in Asian Indians: a community study. J Cardiovasc Dis Res. 2012;3(3):204-10.
4. Gupta R, Deedwania PC, Gupta A, Rastogi S, Panwar RB, Kothari K. Prevalence of metabolic syndrome in an Indian urban population. Int J Cardiol. 2004;97(2):257-61.
5. Frisardi V, Solfrizzi V, Seripa D, Capurso C, Santamato A, Sancarlo D, et al. Metabolic-cognitive syndrome: a cross-talk between metabolic syndrome and Alzheimer's disease. Ageing Res Rev. 2010;9(4):399-417.
6. Biessels GJ, Reagan LP. Hippocampal insulin resistance and cognitive dysfunction. Nat Rev Neurosci. 2015;16(11):660-71.
7. Iadecola C, Davisson RL. Hypertension and cerebrovascular dysfunction. Cell Metab. 2008;7(6):476-84.
8. Kiliaan AJ, Arnoldussen IA, Gustafson DR. Adipokines: a link between obesity and dementia? Lancet Neurol. 2014;13(9):913-23.
9. Yates KF, Sweat V, Yau PL, Turchiano MM, Convit A. Impact of metabolic syndrome on cognition and brain: a selected review of the literature. ArteriosclerThrombVasc Biol. 2012;32(9):2060-7.
10. Yaffe K, Weston AL, Blackwell T, Krueger KA. The metabolic syndrome and Development of cognitive impairment among older women. Arch Neurol. 2009;66(3):324-8.
11. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. Circulation. 2002;106(25):3143-421.
12. Tiwari SC, Tripathi RK, Kumar A. Applicability of the Mini Mental State Examination (MMSE) and the Hindi Mental State Examination for the urban elderly in India. IntPsychogeriatr. 2009;21(1):123-8.
13. Folstein MF, Folstein SE, McHugh PR. "Mini-mental state": a practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res. 1975;12(3):189-98.
14. Hotamisligil GS. Inflammation and metabolic disorders. Nature. 2006;444(7121):860-7.
15. Panza F, Frisardi V, Capurso C, Imbimbo BP, Vendemiale G, Santamato A, et al. Metabolic syndrome and cognitive impairment: current epidemiology and possible underlying mechanisms. J Alzheimers Dis. 2010;21(3):691-724.
16. Craft S. Insulin resistance syndrome and Alzheimer disease: pathophysiologic mechanisms and therapeutic implications. Alzheimer Dis AssocDisord. 2006;20(4):298-301.
17. Raffaitin C, Gin H, Empana JP, Helmer C, Berr C, Tzourio C, et al. Metabolic syndrome and risk for incident Alzheimer's disease or vascular dementia: the Three-City Study. Diabetes Care. 2009;32(1):169-74.
18. Ng TP, Feng L, Nyunt MSZ, Gao Q, Lim ML, Collinson SL, et al. Metabolic syndrome and the risk of mild cognitive impairment and progression to dementia: follow-up of the Singapore Longitudinal Ageing Study cohort. JAMA Neurol. 2016;73(4):456-63.
19. Forti P, Pisacane N, Rietti E, Ravaglia G, Dalmonte E, Bianchin M, et al. Metabolic syndrome and risk of cognitive impairment in older adults. J Am Geriatr Soc. 2010;58(3):487-95.