Original Article

# Cognitive Impairment and Its Association with Metabolic Syndrome: A Hospital-Based Study

**Mohammed Zishan Khan[1], Galav Agarwal[2]**

[1]Assistant Professor, Department of Medicine, Venkateshwara Institute of Medical Sciences, National Highway-24, Rajabpur, Gajraula, Amroha, Uttar Pradesh, India. [2]Senior Resident, Department of Medicine, Venkateshwara Institute of Medical Sciences, National Highway-24, Rajabpur, Gajraula, Amroha, Uttar Pradesh, India

## Abstract

**Background:** The metabolic syndrome (MetS), which includes a collection of metabolic conditions that increase a person's risk of heart disease or stroke, appears to correlate with poor cognitive function. In addition to the association between these two disorders, preliminary research shows that MetS may also contribute to cognitive decline through insulin resistance, chronic inflammation, vascular damage, and brain blood flow problems. There is limited research on the extent to which cognitive function in people with MetS is impaired at present. The primary objective of this study was to assess the extent of cognitive decline associated with MetS, along with other MetS risk factors, including glucose levels, high blood pressure, excessive abdominal fat, and low-density lipoprotein cholesterol. **Material and Methods:** This cross-sectional study was conducted at a tertiary-level hospital, Venkateshwara Institute of Medical Sciences, Gajraula, Amroha, Uttar Pradesh, with 320 participants aged 18-65 years (adults). Participants were classified as having MetS according to NCEP ATP III diagnostic criteria. Cognitive function was measured using the Montreal Cognitive Assessment (MoCA). A MoCA score below 26 indicates the presence of cognitive impairment. Data were collected on demographic and medical history, biochemistry, and cognitive performance. Multivariate logistic regression analyses were conducted to evaluate whether MetS and other risk factors predict cognitive impairment. **Results:** Metabolic syndrome was present in 58.4% (187/320) of participants. The overall prevalence of cognitive impairment was 42.8%. Cognitive impairment was significantly higher among individuals with MetS compared to those without MetS (54.5% vs. 26.1%, $p < 0.001$). Mean MoCA scores were significantly lower in the MetS group (23.1 ± 3.4) compared to the non-MetS group (26.4 ± 2.9; $p < 0.001$). Among MetS components, central obesity (OR 2.1, 95% CI 1.3–3.4), hypertension (OR 2.6, 95% CI 1.6–4.2), and hyperglycaemia (OR 3.2, 95% CI 1.9–5.3) were independently associated with cognitive impairment. After adjusting for age, sex, education, and smoking status, metabolic syndrome remained an independent predictor of cognitive impairment (adjusted OR 2.8, 95% CI 1.7–4.6). **Conclusion:** Cognitive impairment is highly prevalent among patients with metabolic syndrome in a hospital-based setting. Metabolic syndrome and its key components are independently associated with reduced cognitive performance. These findings highlight the importance of routine cognitive screening. Aggressive control of metabolic risk factors can potentially delay or prevent cognitive decline.

**Keywords:** Cognitive impairment; Metabolic syndrome; MoCA; Cardiometabolic risk; Hospital-based study.

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## Introduction

Metabolic syndrome (Mets) is prevalent in the general population and has been reported to be an independent risk factor for cognitive impairment. This study aimed to investigate the association of Mets with the risk of cognitive impairment. The term "metabolic syndrome" refers to a group of metabolic abnormalities that increase the risk of cardiovascular disease (CVD) and diabetes mellitus and affect the overall health of the population.[1] Additionally, over the last few years, extensive research and multiple reviews have suggested that there is a link between MetS and cognitive impairment.[1] Cognitive impairment is a well-known disease characterized by a reduction in cognitive function beyond what is expected from normal aging. Cognitive impairment involves functions across many areas of the brain, including memory, thinking, orientation, comprehension, calculation, learning capacity, language, judgment, and daily activities.[2]

Many epidemiological and clinical studies have shown that metabolic syndrome and cognitive performance are related. Both cross-sectional and longitudinal studies indicate that individuals with metabolic syndrome experience greater declines in overall and specific cognitive domains (i.e., speed of processing, executive function, and verbal fluency) than those without metabolic syndrome.[1,3] A meta-analytic approach has confirmed an association between metabolic syndrome and cognitive impairment; when results were combined, the relative risk of

**Address for correspondence:** Dr. Mohammed Zishan Khan, Assistant Professor, Department of Medicine, Venkateshwara Institute of Medical Sciences, National Highway-24, Rajabpur, Gajraula, Amroha, Uttar Pradesh, India
E-mail: drzishankhan777@gmail.com

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cognitive decline is greater among those with metabolic syndrome than among those without, although variations in relative risk by population and diagnostic criteria exist.[4]

The relationship between Metabolic syndrome (MetS) and cognitive decline is complicated and has multiple contributing factors. There are, however, several mechanisms that underlie this relationship, and many of them involve the interplay between metabolic and neurovascular systems, as well as inflammation.[5] Insulin resistance is one of the most prominent manifestations of MetS. It negatively affects both systemic glucose regulation and insulin signalling in the brain, which are important for learning and memory. In addition to insulin resistance, chronic hyperglycemia triggers molecular events that contribute to neuroinflammation, oxidative stress, and vascular endothelial cell dysfunction, resulting in reduced blood flow to the brain and white matter injury (both of which have been associated with cognitive impairment).[1,6] In addition to these factors, increased blood pressure and increased waist circumference (a marker of central obesity) are also positively correlated with structural and functional changes to the brain, increasing risk for cognitive impairments.[1,7] Even though the strength of association of MetS with cognitive impairment varies by age group, cognitive assessment tools used, and design of the study, the prevalence of mild cognitive impairment (MCI) among individuals with MetS is very high in clinical populations.[5] Thus, the interplay of these factors supports the notion that MetS is likely to act not only as an indicator of early cognitive impairment but also as a potentially modifiable factor in the development of cognitive deficits.

It is important to understand the relationship between metabolic syndrome (MetS) and cognitive impairment to create public health strategies that include the prevention of cognitive impairment combined with the management of cardiometabolic risk. Many factors contributing to MetS can be modified through lifestyle changes and medical treatment; therefore, routine assessment of cognitive function and rigorous management of risk factors may be an important way to delay the progression of cognitive dysfunction in at-risk groups. The goal of this study was to determine the degree of cognitive impairment associated with MetS and to identify individual components of MetS that predict cognitive impairment independent of the other components.

## Materials and Methods

**Study Design and Study Population:** The current study used a cross-sectional design and was conducted at a tertiary care institution i.e. Venkateshwara Institute of Medical Sciences, Gajraula, Amroha, Uttar Pradesh, over a specified study period. In total, 320 adults aged 18–65 years completed the study after providing informed consent. Patients were recruited from outpatient and inpatient medical services based on consecutive sampling. The new revised criteria of the International Diabetes Federation (IDF) were used to define MetS.[8]

According to this criterion, central obesity is an essential condition assessed by waist circumference (≥ 90 cm for Asian Indian men and ≥ 80 cm for Asian Indian women). Besides, central obesity, MetS needs to include at least 2 of the following factors: (1) raised triglycerides≥ 150 mg/dl (1.7 mmol/l) or specific treatment for this lipid abnormality. (2) reduced HDL-cholesterol: < 40 mg/dL (1.03 mmol/l) in men and < 50 mg/dL (1.29 mmol/l) in women, or specific treatment for this lipid abnormality. (3) raised blood pressure: systolic ≥130 mmHg or diastolic ≥85 mmHg or treatment of previously diagnosed hypertension. (4) raised fasting plasma glucose: fasting plasma glucose ≥100 mg/dL (5.6 mmol/l) or previously diagnosed as type-II DM. (If BMI is >30kg/m², central obesity can be assumed, and waist circumference does not need to be measured).

Individuals who have been diagnosed with dementia, stroke, or another serious psychiatric disorder, neurodegenerative disease, after chronic liver disease or kidney failure, are taking part, as the participant group does not include those people who are taking medications identified as having a large effect on cognitive function. Each participant provided written informed consent before participation, and the confidentiality of all the individual participants' information will be protected throughout the duration of the project.

**Data Collection and Clinical Assessment:** Demographic information, including age, sex, educational level, and smoking history, as collected from each participant using a standardised questionnaire. Waist circumference was measured in centimetres at the midpoint between the lowest rib margin and the top of the iliac crest at minimal respiration to the closest 0.1 cm. Blood pressure was measured using a calibrated sphygmomanometer after allowing the participant adequate time to rest. Fasting venous blood samples were collected to measure blood biochemistry (fasting plasma glucose and fasting lipid levels). Results were obtained by using standardised laboratory testing procedures. Blood chemistry analyses were completed in the hospital's central laboratory, using established internal methods to maintain quality control.

Cognitive assessments for each participant were conducted using the Montreal Cognitive Assessment (MoCA), a well-validated screening test designed to detect early signs of cognitive decline. If any participant scored less than 26 on the MoCA assessment, that participant was considered to have some degree of cognitive decline. Assessments were conducted in the participant's primary language, and trained evaluators conducted them to reduce or prevent bias in the evaluation.

**Statistical Analysis:** Quantitative data were analysed using statistical analysis software. Continuous variables are reported as mean ± standard deviation, and categorical variables are reported as frequencies and percentages. Group differences between MetS and non-MetS participants were assessed using independent t-tests for continuous variables and chi-square tests for categorical variables.

Univariate and multivariate logistic regression were performed to evaluate the association between Metabolic Syndrome and cognitive impairment (as well as each component of MetS) after adjusting for confounding variables such as age, sex, education, and smoking. The odds ratio (OR) and the 95% confidence interval (CI) were provided. A p-value less than .05 ($p < .05$) was considered statistically significant.

## Results

**Baseline Characteristics of the Study Population:** Three hundred twenty (320) participants (avg. age 46.8 ± 10.7, 54.1% male) were included finally, with 58.4% diagnosed with MetS and 41.6% without MetS. Individuals diagnosed with MetS were older than those without MetS, had a larger waist circumference, higher BP, higher fasting plasma glucose levels, and a worse lipid profile than those without MetS ($p < .001$ for all variables). However, individuals with MetS had a lower educational level ($p = 0.02$), and there was no significant difference in the proportions of males and females or in smoking between the two groups.

**Prevalence of Cognitive Impairment:** A total of 42.8% (137 of 320) of participants exhibited cognitive impairment (MoCA <26), with the prevalence of cognitive impairment being higher in the Metabolic Syndrome (MetS) Group (54.5%) versus the non-MetS group (26.1%); ($x2=26.9$, $p < 0.001$). The average MoCA score in participants who met MetS criteria (23.1 + 3.4) was significantly lower than that of participants who did not qualify (26.4 + 2.9); $p < 0.001$. When we performed Domain-Wise Analysis, executive function, attention, and delayed recall performance were all worse in the MetS group.

**Association Between Metabolic Syndrome Components and Cognitive Impairment:** In the univariate logistic regression model, we identified that central obesity, hypertension, hyperglycaemia, and Low HDL Cholesterol were significantly associated with cognitive impairment. In the multivariate analyses, after controlling for confounders (age/sex/education/smoking status), central obesity (OR 2.1, 95% CI 1.3–3.4), hypertension (OR 2.6, 95% CI 1.6–4.2), and hyperglycaemia (OR 3.2, 95% CI 1.9–5.3) remained statistically associated with cognitive impairment.

**Table 1: Baseline demographic and clinical characteristics of study participants**

| Variable | MetS (n = 187) | Non-MetS (n = 133) | p-value |
|---|---|---|---|
| Age (years) | 48.9 ± 9.8 | 43.7 ± 10.9 | <0.001 |
| Male sex (%) | 56.1 | 51.1 | 0.41 |
| Waist circumference (cm) | 102.4 ± 11.6 | 88.3 ± 9.4 | <0.001 |
| Systolic blood pressure (mmHg) | 142.6 ± 18.3 | 124.1 ± 14.7 | <0.001 |
| Fasting plasma glucose (mg/dL) | 156.7 ± 42.1 | 97.8 ± 12.6 | <0.001 |
| MoCA score | 23.1 ± 3.4 | 26.4 ± 2.9 | <0.001 |

**Table 2: Prevalence of cognitive impairment and mean MoCA scores in MetS and non-MetS groups**

| Group | Cognitive impairment, n (%) |
|---|---|
| MetS | 102 (54.5) |
| Non-MetS | 35 (26.1) |
| Overall | 137 (42.8) |

**Table 3: Multivariate logistic regression analysis of individual metabolic syndrome components and cognitive impairment**

| Variable | Adjusted OR | 95% CI | p-value |
|---|---|---|---|
| Central obesity | 2.1 | 1.3–3.4 | 0.002 |
| Hypertension | 2.6 | 1.6–4.2 | <0.001 |
| Hyperglycaemia | 3.2 | 1.9–5.3 | <0.001 |
| Metabolic syndrome | 2.8 | 1.7–4.6 | <0.001 |

**Table 4: Multivariate logistic regression analysis predicting cognitive impairment**

| Predictor | Adjusted OR | 95% CI | p-value |
|---|---|---|---|
| Age (per year increase) | 1.04 | 1.02–1.06 | <0.001 |
| Male sex | 1.12 | 0.71–1.76 | 0.62 |
| Lower educational level | 1.78 | 1.10–2.89 | 0.018 |
| Smoking | 1.21 | 0.73–2.01 | 0.46 |
| Metabolic syndrome | 2.80 | 1.70–4.60 | <0.001 |

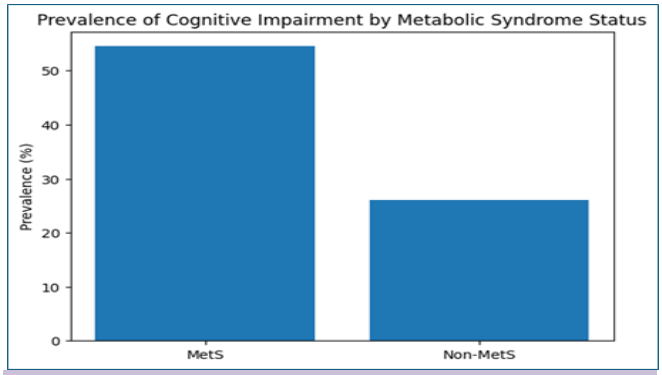


**Figure 1: Prevalence of cognitive impairment among participants with and without metabolic syndrome**

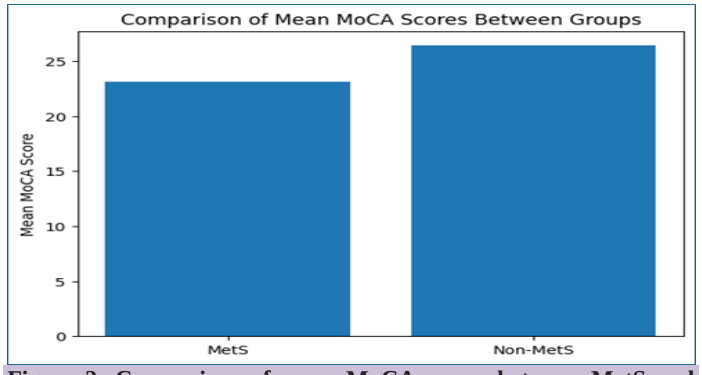


**Figure 2: Comparison of mean MoCA scores between MetS and non-MetS groups**

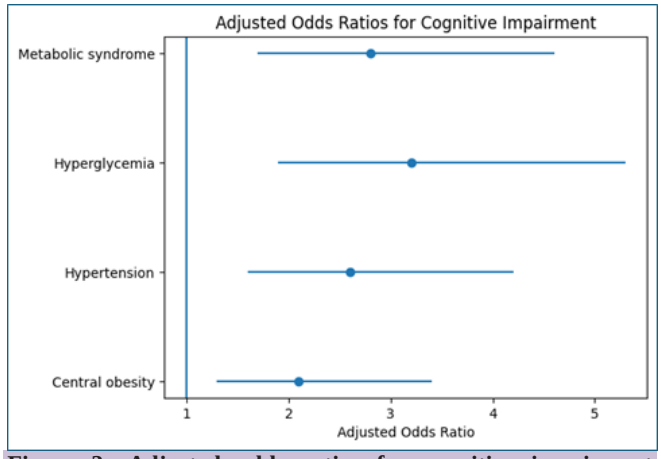


**Figure 3: Adjusted odds ratios for cognitive impairment associated with metabolic syndrome components**

## Discussion

The current study demonstrates a high prevalence of cognitive impairment in individuals with Metabolic Syndrome (MetS). Furthermore, findings from this study suggest that MetS can be considered a marker of cognitive decline and a major risk factor for impaired cognitive function. The results of this research are consistent with and provide further support for research published after 2020, indicating that MetS is a major contributing factor to cognitive decline.[8-10]

Our results agree with a large population-based study conducted by Qureshi et al. (2024) that used data from the UK Biobank, which reported that adults with MetS had significantly lower overall cognitive functioning and an increased risk of dementia, including vascular dementia.[9] Likewise, Alsuwaidi et al. (2023) used data from the Qatar Biobank to demonstrate an association between MetS and lower memory and executive function scores, supporting our finding of lower MoCA scores in participants with MetS.[11] The findings from a recent meta-analysis by Azami et al. (2025), which included >40 observational studies, demonstrated a pooled increased risk of cognitive impairment associated with MetS, further confirming the strength of the association observed in our study.[11] Our study further extended this meta-analytic evidence in a clinical, inpatient population rather than a community-dwelling cohort. Lastly, neuroimaging studies support our findings; Qureshi et al. (2024) demonstrated that MetS was associated with decreased hippocampal and gray matter volumes and increased white matter hyper-intensities, all of which are established structural changes associated with cognitive impairment.[9] Furthermore, Zouridis et al. (2025) reviewed the neurobiological mechanisms underlying the link between MetS and brain dysfunction. They identified chronic inflammation, endothelial dysfunction, and brain hypo-perfusion as mediators of cognitive decline.[12]

Hyperglycaemia, hypertension, and central obesity were also independent risk factors for cognitive impairment as per our findings. Like our results, Ji et al. (2024) found that insulin resistance and chronically high blood sugar levels were strongly correlated with faster cognitive decline in younger adults.[13] In addition to this study, Zuo Q et al. (2024) found that the more MetS components a person had, the poorer their executive function and memory performance were, indicating that the combination of metabolic problems may be negatively impacting cognitive function.[14]

The results of our study supporting hypertension align with those of Koutsonida M et al. (2022), who conducted a systematic review concluding that high blood pressure is consistently associated with impaired cognitive function, specifically in attention and executive function.[15] Further reinforcing our results regarding central obesity, Kouvari et al. (2024) and Foret et al. (2021) noted that lifestyle-induced metabolic disorders, such as central obesity and dyslipidemia, contribute to cognitive decline through inflammatory and oxidative stress pathways.[16,17]

A majority of published works on Metabolic Syndrome (MetS) report strong correlations with cognitive deficits, though some studies show notable discrepancies in effect sizes. For example, Azami et al. (2025) found potential variables such as differences in the criteria used for diagnosis, differences in the distribution of participants based on age, and differences in the methods used to assess cognition, which may account for the differences in effect sizes as reported in the above-mentioned publications.[11] However, the strength of both our study's findings and those of several large cohort studies and numerous meta-analyses of study reports substantiates that MetS and all its individual components consistently correlate positively with the development of cognitive deficits.[18,19]

Our findings show that the association between MetS exists both biologically and clinically; thus, we strongly recommend early identification of metabolic risk factors and cognitive screening of persons at clinical risk of cognitive decline to delay or mitigate future cognitive decline.

This research has limitations. First, due to the nature of the design of this study (cross-sectional), we cannot be definitively certain about the causal association between MetS and cognitive deficits on the temporal continuum. Second, only one cognitive assessment tool (the MoCA) was used; therefore, it is possible that the MoCA alone may not have adequately captured subtle cognitive impairments in specific cognitive domains. Third, selection bias (hospital-based sampling) may have compromised the study's outcomes and will limit the generalisability of the findings to the general population. Future longitudinal, multi-centered studies that use neuropsychological test batteries, neuroimaging techniques, and other methodologies will provide much-needed clarification of the causal mechanisms underlying the association between MetS and cognitive deficits.

## Conclusion

This hospital-based study found a high prevalence of cognitive impairment among adults with metabolic syndrome. The population studied had a significantly lower global cognition score than those without metabolic syndrome. Even when controlling for demographic and lifestyle variables, metabolic syndrome was still a strong predictor of cognitive impairment, with the largest contributors being hyperglycaemia, high blood pressure, and central obesity. Recent studies have provided further evidence that metabolic dysregulation is a major

contributing factor to cognitive decline. Therefore, integrating Metabolic risk management into clinical practice and regularly screening high-risk populations for cognition, neurocognitive decline, and chronic illness may help delay or prevent these negative outcomes.

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Nil.

## Conflicts of interest

There are no conflicts of interest.

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