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# Prevalence of cognitive impairment and metabolic syndrome among older adults in calabar metropolis and the associated risk factors

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## Abstract

**Background** The number of people reaching old age is rising, bringing an increase in age-related diseases like cardiovascular conditions and cognitive dysfunction. Cognitive impairment (CI) impacts various brain functions, affecting daily activities and quality of life. Metabolic syndrome (MetS), a cluster of cardiovascular risk factors, has been implicated in CI. This study examines the prevalence of MetS and CI among older adults in Calabar Metropolis and the associated risk factors.

**Methods** This study was conducted in Calabar Metropolis, Cross River State, Nigeria, with 236 older adults (aged 65 years and above) selected via a multi-stage sampling technique. Informed consent was obtained from all participants. Physical examinations and biomarker assessments included measurements of blood pressure, height, weight, waist and hip circumference, fasting plasma glucose, cholesterol, triglycerides, and high-density lipoprotein (HDL) levels. MetS was defined according to the NCEP Adult Treatment Panel III criteria. CI was assessed using the Mini-Cog™ test, with scores ≤ 3 indicating poor cognitive status. Data analysis utilized SPSS version 26.0, employing chi-square tests and binary logistic regression. Significance was set at $p < 0.05$.

**Results** The prevalence of MetS was 32.2%, and CI was observed in 44% of participants. Females had a slightly higher prevalence (57.9%) of MetS compared to males (42.1%). Significant differences were found between MetS and non-MetS groups in systolic blood pressure, fasting blood sugar, triglycerides, abdominal obesity, and cardiovascular risk. MetS overall was not significantly associated with CI. However, reduced HDL levels were significantly linked to poor cognitive status (OR = 70.528, 95% CI = 3.269-1521.748). Other MetS components did not show significant associations with CI.

**Conclusions** This study highlights the prevalence of MetS and CI among older adults in Calabar Metropolis. The findings suggest that while MetS as a whole is not associated with CI, reduced HDL levels are significantly linked to

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poor cognitive status. The findings emphasize the importance of managing specific metabolic risk factors, particularly HDL, to maintain cognitive health in elderly population.

**Clinical trial registration** Not applicable.

**Keywords** Metabolic syndrome, Cognitive impairment, Older adults

## Background

The world is facing a significant challenge with its elderly population, with the proportion of older adults expected to reach 31% by 2050 [1]. Africa, despite having the lowest percentage of older adults, will experience a significant increase in this demographic, with Nigeria, the continent's most populous country, leading the way [2]. The total number of older Africans is projected to triple between 2020 and 2050, with the population of Nigerians aged 65 and older projected to nearly triple by 2050 [2]. This shift is accompanied by a rise in age-related diseases like cardiovascular disease and cognitive dysfunction, which severely impact quality of life and healthcare systems [3].

Normal aging is typically associated with gradual declines in physical and cognitive functions, but these changes do not necessarily lead to significant impairments. CI, however, represent a deviation from normal aging, characterized by a reduction in cognitive functions such as memory, thinking, orientation, comprehension, calculation, learning capacity, language, judgement, and daily activities [4]. Epidemiological studies have indicated that the prevalence of mild cognitive impairment (MCI) varies from 2.8 to 17.5% in Europe and North America and 5.4–25.0% in different parts of China [5]. However, the prevalence of CI in Nigeria is less studied than in high-income countries, as classified by world bank [6]. This financial constraint impacts the availability and quality of healthcare services, nutritional status, and overall well-being of older adults, potentially influencing the prevalence and progression of CI [7]. For instance, a survey of CI among Yoruba-speaking individuals from Ibadan, Nigeria, revealed that 152 (62%) out of 423 individuals studied were diagnosed with CI no dementia (CIND), while 28 (6.61%) were diagnosed with dementia [6]. In northern Nigeria, a survey of 323 older adults showed a dementia prevalence of 2.79% (CI 1–4.58%), representing 66.67% of all dementia cases in the sample [7]. In southwest Nigeria, a 10.1% prevalence of probable dementia was found using the 10-Word Delay Recall test adapted from the Consortium to Establish a Registry for Alzheimer's Disease (CERAD) [8]. In north central Nigeria, Ochayi and Thacher [9] used the Community Screening Instrument for Dementia (CSID) to show a 6.4% overall prevalence of dementia. In southeast Nigeria, a 23.1% depression prevalence was found in older adults, with 20.7% complaining of forgetfulness [10].

Research has identified MetS, a cluster of cardiovascular risk factors including obesity, hypertension, hyperglycemia, and dyslipidemia as a potential contributor to CI. The underlying mechanisms may involve insulin resistance, inflammation, and cardiovascular changes that affect brain function [11]. MetS is a significant public health burden, affecting approximately 20–30% of adults worldwide, with prevalence varying across regions and countries, including 27.4% of urban Chinese [12], 23% of Americans [13], and 30.52% of South Koreans [14]. Studies have reported higher prevalence in various sub-Saharan African countries, such as Nigeria (23%) [15] and Ghana (41.8%) [16], as well as north African countries, with rate of 39.6% in Tunisia and 48.5% in Morocco [17, 18]. Previous studies that have investigated the association between MetS and CI reported inconsistent results. Some studies showed an association between MetS and CI [19, 20], while other studies showed no association [21, 22]. Moreover, there is paucity of information on the relationship between MetS and cognitive status in south-south Nigeria, specifically in Calabar, highlighting the need for further research in this region. Therefore, this study aims to investigate the prevalence of CI and MetS among older adults in Calabar Metropolis and the associated risk factors, to develop effective prevention and intervention strategies.

## Methods

### Study design

This study was conducted in Calabar metropolis, Cross River State, Nigeria. A total of 236 subjects of both men and women from the age of 65years and above, were enrolled into the study through a multi-stage sampling technique. First, Calabar Metropolis was stratified into its constituent local government areas (LGA). Next, communities within each LGA were randomly selected. Then, households within these communities were chosen by starting at a random point and selecting households at regular intervals. Finally, eligible participants within these households were randomly chosen to participate in the study. The sample size was calculated using the formula for estimating proportions in a population [23]:

$$\mathrm{N} = \frac{z^2 * \mathrm{p}(1-\mathrm{p})}{\mathrm{d}^2}$$

where:

- n is the sample size,
- Z is the Z-value (1.96 for a 95% confidence level),
- p is the estimated proportion of the population with the characteristic of interest, that is, 10.1% prevalence of cognitive impairment among older adults from previous studies [8].
- d is the margin of error (0.05).

Using these values, the minimum required sample size was calculated to be 140 participants. However, to enhance the power of the study and account for potential non-responses, a total of 236 participants were ultimately enrolled. Written informed consent was obtained from all subjects before recruitment into the study. The study was carried out in accordance with the ethical principles for research involving human subjects, as outlined in the Helsinki declaration in 1975 and subsequent revisions. Older adults that were extremely sick and/or having neurological conditions were excluded from this study.

#### Physical examination and assessment of metabolic syndrome

Blood pressure was measured using a standard mercury sphygmomanometer, with systolic and diastolic pressures recorded in triplicate. Height was determined using a stadiometer, and weight was measured with an Omron automatic digital weighing scale. Body mass index (BMI) was calculated as weight (kg) divided by the square of height ($m^2$). Waist and hip circumferences were measured using a measuring tape, and waist-to-hip ratio was calculated accordingly.

#### Biomarker measurement

Overnight fasting blood sample (8 ml) was collected from each subject, 3 ml was dispensed into a fluoride oxalate bottle and gently inverted to ensure that the sample did not clot. Five milliters (5 ml) was also dispensed into a plain bottle and allowed to clot and retract. Both samples were centrifuged at 3000 rpm for 5 min. Plasma obtained was used for fasting blood glucose test immediately and the serum obtained was stored frozen at -80°C until used for Insulin and Lipid profile analyses.

#### Determination of fasting blood glucose

The glucose concentration was measured using the glucose-oxidase enzymatic colorimetric method [24], employing glucose reagent (GLUC-PAP) from Randox Laboratories Limited. In this method, glucose in the plasma was oxidized to gluconic acid and hydrogen peroxide by glucose-oxidase. The hydrogen peroxide is then catalyzed by peroxidase to form water and nascent oxygen. The nascent oxygen reacts with chromogens 4-aminoantipyrine and phenol, resulted in the formation of a pink-colored complex. The intensity of the color is directly proportional to the concentration of glucose in the sample, and the absorbance was read at 505 nm using a spectrophotometer. All measurements were performed in duplicate to ensure accuracy. The glucose concentration was calculated by comparing the absorbance of the test sample to that of a known standard with a concentration of 5.5 mmol/L.

#### Determination of insulin

Serum insulin levels were quantified using Abcam's Human Insulin ELISA kit, an assay based on the simultaneous binding of human insulin by two monoclonal antibodies [25]. One antibody was immobilized on micro-well plates, while the other was conjugated with horseradish peroxidase (HRP). After incubation and washing steps, the enzyme HRP reacted with a substrate to produce a colorimetric change that was measured at 450 nm using an ELISA reader.

#### Lipid profile analysis

For lipid profile analysis, total cholesterol was measured using the cholesterol oxidase method [26]. Cholesterol esters in the serum were hydrolyzed by cholesterol esterase to free cholesterol, which was then oxidized by cholesterol oxidase, producing hydrogen peroxide. The hydrogen peroxide reacted with para-aminophenazone and phenol to form a pink-colored dye, whose absorbance was measured at 515 nm. Triglycerides were determined using the Glycerol-3-Phosphate Oxidase - Phenol Aminophenazone (GPO-PAP) enzymatic colorimetric method [26], where triglycerides were enzymatically split into glycerol and fatty acids. The glycerol then reacted with ATP to form glycerol-3-phosphate, and the absorbance was measured at 510 nm.

HDL cholesterol was quantified after precipitating very low-density lipoprotein (VLDL) and low-density lipoprotein (LDL) using phosphotungstate in the presence of magnesium ions [26]. After centrifugation, the clear supernatant containing HDL cholesterol was measured using the same enzymatic method used for total cholesterol. VLDL was calculated by dividing the triglyceride concentration by 5, and LDL cholesterol was determined using the Friedewald formula:

$$\text{LDL cholesterol} = \text{Total cholesterol} - \text{HDL cholesterol} - \frac{\text{Triglycerides.}}{5}$$

#### Calculation of HOMA-IR and atherogenic index of plasma

The Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) was calculated using the fasting glucose and insulin concentrations according to the formula [27]:

$$\text{HOMA} - \text{IR} = \frac{\text{Fasting Glucose (mmol/L)} \times \text{Fasting Insulin (mU/L)}.}{22.5}$$

The Atherogenic Index of Plasma (AIP) was calculated as the logarithm of the triglyceride-to-HDL cholesterol ratio [28].

#### Criteria for MetS

NCEP ADULT TREATMENT PANEL 111 was used in this study. MetS is present if three or more of the following five criteria are met; waist circumference over 40 inches for men or 35 inches for women (waist hip ratio ≥ 0.9 for men or 0.85 for women ), systolic blood pressure ≥ 130 and/or diastolic ≥ 85mmHg, fasting triglyceride level ≥ 150 mg/dl (1.7mmol/l), fasting HDL cholesterol level less < 40 mg/dl (1.0mmol/l) for men or < 50 mg/dl (1.3mmol/l) for women and fasting blood sugar ≥ 100 mg/dl (5.5mmol/l).

#### Mini-Cog™ test for older adults

The Mini-Cog test was selected for cognitive assessment due to its brevity, ease of administration, and high sensitivity for detecting cognitive impairment in older adults [29]. The Mini-Cog combines a three-item recall test with a clock-drawing test, both of which assess different cognitive domains. Although there are other cognitive assessment tools available, the Mini-Cog was chosen because it is less influenced by language and education level, making it particularly suitable for the diverse population in Calabar. Additionally, the Mini-Cog's high sensitivity and specificity for detecting dementia and MCI support its use as an appropriate screening tool in this study [29]. While other assessments, such as the Mini-Mental State Examination (MMSE), could also be used, the Mini-Cog's ability to quickly and effectively screen large populations for cognitive issues made it the most suitable choice for this study.

The Mini-Cog scoring includes a maximum of 3 points for the recall test (1 point for each word correctly recalled without prompting) and a maximum of 2 points for the clock-drawing test [29]. A normal clock must include all numbers (1–12), each only once, in the correct order and direction (clockwise). It must also have two hands. Hand length is not scored in the Mini-Cog algorithm. A score of < 3 is indicative of poor cognitive status [29–32]. Similar studies have employed the Mini-cog test in identifying cognitive impairment [29–32].

#### Statistical analysis

Data analysis was done using the statistical package for social sciences (SPSS) software 26.0, using chi square to compare the baseline characteristics of the older adults by MetS. A binary logistic regression analysis was carried out to ascertain the association between MetS and poor CI. The level of significance was set at $p < 0.05$.

## Results

Figure 1 shows the prevalence of MetS in older adults, out of the 236 subjects, 76 (32.2%) had MetS. Figure 2 shows the cognitive status of the participants, out of the 236 older adults, 104 (44%) had poor CI. Table 1 shows the baseline characteristics of the older adults by MetS. Among the older adults with MetS, 42.1% (32 out of 76) were male, and 57.9% (44 out of 76) were female. There was no statistically significant difference ($p > 0.05$) in the prevalence of MetS in relation to biological sex. A significant difference ($p < 0.05$) was observed in the proportion of subjects with elevated systolic blood pressure between the non-MetS (57.5%) and MetS (73.7%) groups. Additionally, the MetS group had a significantly higher proportion of subjects with elevated fasting blood sugar (FBS) (52.6%, $p < 0.05$), elevated triglyceride (TG) (68.4%, $p < 0.01$), abdominal obesity (84.2%, $p < 0.05$), and high risk for cardiovascular disease (36.8%, $p \leq 0.01$). There was no statistically significant difference in the proportion of subjects with poor cognitive status between the two groups. (Non-MetS: 45.0%, MetS: 42.1%). Table 2 shows the association between MetS and poor cognitive status. After a binary logistic regression, there was no significant association between MetS and poor cognitive status (AOR 1.687, 95% CI 0.096–29.552). However, reduced HDL was significantly associated with poor cognitive status (AOR 70.528, 95% CI 3.269–1521.748) implying that older adults with reduced HDL levels were approximately 70 times more likely to have poor cognitive status. Additionally, MetS components such as elevated systolic blood pressure (SBP) (AOR 1.304, 95% CI 0.067–25.427), elevated diastolic blood pressure (DBP) (AOR 17.914, 95% CI 0.288-1113.834), elevated FBS (AOR 6.118, 95% CI 0.176-212.323), elevated TG (AOR 6.227, 95% CI 0.056-698.022), insulin resistance (AOR 2.243, 95% CI 0.967–5.204), abdominal obesity (AOR 8.240, 95% CI 0.305-222.522), and high risk for cardiovascular disease (CVD) (AOR 0.147, 95% CI 0.001–18.040) showed no significant association with poor cognitive status.

## Discussion

The primary objectives were to determine the prevalence of CI and MetS, identify the associated risk factors for MetS, examine the relationship between MetS and CI, and determine the predictors of CI among older adults in Calabar Metropolis. This study revealed a high prevalence of both poor cognitive status (44%) and MetS (32.2%). Whilst the overall association between MetS and CI was not statistically significant, reduced HDL levels were significantly associated with poor cognitive status.

The prevalence of MetS and CI among older adults in Calabar Metropolis aligns with global trends where older populations show increased metabolic and cognitive health issues [33–35]. The gender distribution

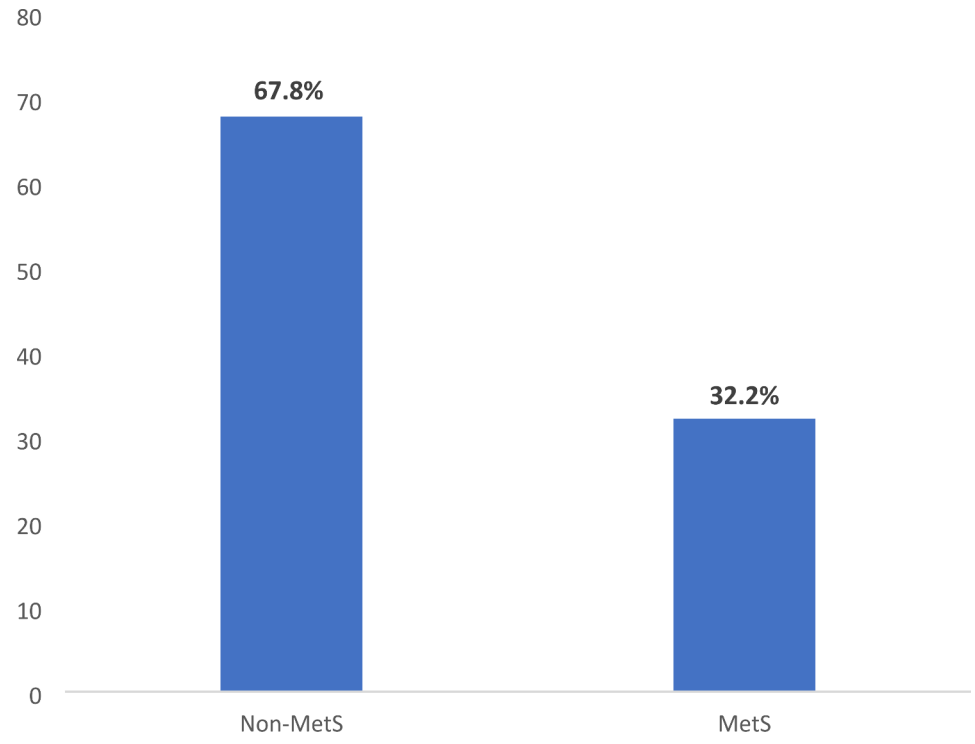


**Fig. 1** Prevalence of MetS among older adults in calabar metropolis

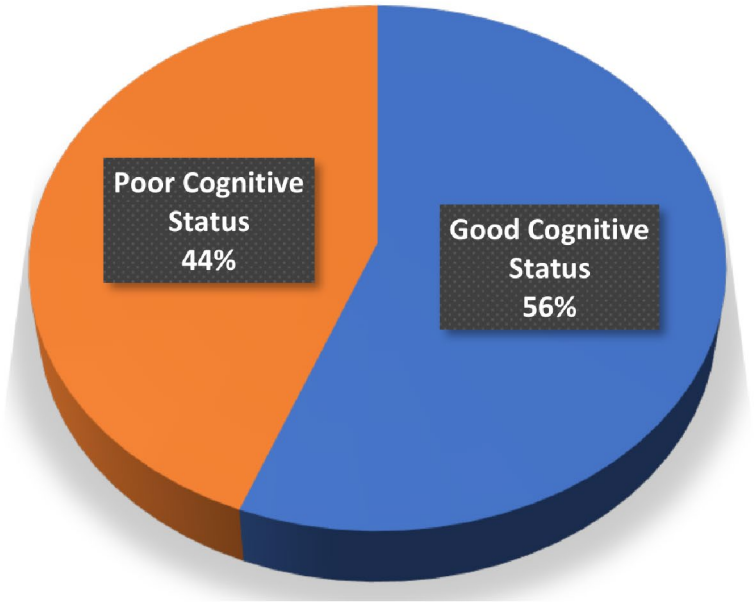


**Fig. 2** Cognitive status of the participants

of MetS, with a slightly higher prevalence in females, is consistent with previous studies indicating that post-menopausal hormonal changes might contribute to higher metabolic risks in women [36, 37]. Oestrogen is known to have protective effects on the cardiovascular system, and its decline during menopause is associated with increased risks of MetS components such as central obesity, dyslipidemia, and hypertension [36, 37]. These changes could potentially influence cognitive functions. Previous studies have shown that the loss of oestrogen's

**Table 1** Baseline characteristics of the older adults by MetS

| Variables | Non-MetS *n* = 160 | MetS *n* = 76 | *p*-value |
|---|---|---|---|
| **Gender** | | | |
| Male | 48(30.0) | 32(42.1) | 0.359 |
| Female | 112(70.0) | 44(57.9) | |
| **Components of MetS** | | | |
| Elevated SBP | 92(57.5) | 56(73.7) | 0.030* |
| Elevated DBP | 60(37.5) | 40(52.6) | 0.272 |
| Elevated FBS | 20(12.5) | 40(52.6) | 0.003* |
| Reduced HDL | 32(20.0) | 28(36.8) | 0.173 |
| Elevated TG | 20(12.5) | 52(68.4) | < 0.001* |
| Insulin resistance | 84(52.5) | 60(78.9) | 0.046 |
| Abdominal obesity | 88(55.0) | 64(84.2) | 0.029* |
| High risk for CVD | 4(2.5) | 28(36.8) | 0.001* |
| **Metal Status** | | | |
| Poor | 72(45.0) | 32(42.1) | 0.834 |
| Good | 88(55.0) | 44(57.9) | |

Where: MetS = Metabolic syndrome, SPB = Systolic blood pressure, DBP = Diastolic blood pressure, FBS = Fasting blood sugar, HDL = High density lipoprotein, TG = Triglyceride, CVD = Cardiovascular disease, Insulin resistance = Homeostatic model assessment for insulin resistance (HOMA-IR) ≥ 3, Abdominal obesity = Waist hip ratio (WHR) ≥ 0.9 for men/≥ 0.85 for women, High risk for CVD = Atherogenic index of plasma (AIP) ≥ 0.24

**Table 2** Association between MetS and poor cognitive status – binary logistic regression

| Variables | AOR | 95% CI | S.E. |
|---|---|---|---|
| MetS | 1.687 | 0.096–29.552 | 1.461 |
| Elevated SBP | 1.304 | 0.067–25.427 | 1.516 |
| Elevated DBP | 17.914 | 0.288–1113.834 | 2.107 |
| Elevated FBS | 6.118 | 0.176–212.323 | 1.810 |
| Reduced HDL | 70.528 | 3.269–1521.748* | 1.567 |
| Elevated TG | 6.227 | 0.056–698.022 | 2.408 |
| Insulin resistance | 2.243 | 0.967–2.564 | 1.112 |
| Abdominal obesity | 8.240 | 0.305–222.522 | 1.682 |
| High risk for CVD | 0.147 | 0.001–18.040 | 2.454 |

Where: MetS = Metabolic syndrome, SPB = Systolic blood pressure, DBP = Diastolic blood pressure, FBS = Fasting blood sugar, HDL = High density lipoprotein, TG = Triglyceride, CVD = Cardiovascular disease, Insulin resistance = Homeostatic model assessment for insulin resistance (HOMA-IR) ≥ 3, Abdominal obesity = Waist hip ratio (WHR) ≥ 0.9 for men/≥ 0.85 for women, High risk for CVD = Atherogenic index of plasma (AIP) ≥ 0.24, AOR = Adjusted odd ratio (adjusted for all the MetS components), * = significant at $p < 0.05$

neuroprotective effects may exacerbate cognitive decline, particularly in postmenopausal women, due to its impact on glucose metabolism and cerebral blood flow [38, 39]. The significant differences in elevated SBP, FBS, TG, abdominal obesity, and cardiovascular risk between the MetS and non-MetS groups reflect the established understanding of MetS components [40]. These components are known risk factors for various chronic conditions, including cardiovascular diseases and diabetes, which can indirectly affect cognitive functions through vascular and inflammatory pathways [41, 42]. Despite the established link between MetS components and cognitive decline in other studies [43–45], our study did not find a significant association between MetS as a whole and poor cognitive status. This may be due to the complexity and multifactorial nature of CI, which involves genetic, lifestyle, and environmental factors beyond metabolic health alone [46]. One of the most notable findings was the significant association between reduced HDL levels and poor cognitive status; this is in tandem with previous studies [33, 47]. Older adults with low HDL were approximately 70 times more likely to have poor cognitive status. HDL is known for its role in reverse cholesterol transport and anti-inflammatory properties, which are crucial in maintaining vascular health and preventing atherosclerosis [48, 49]. Reduced HDL can lead to increased plaque formation in cerebral arteries, contributing to vascular dementia and cognitive decline [50, 51]. However, factors such as poor diet, physical inactivity, and genetic predispositions can contribute to low HDL levels. While reduced HDL was a significant predictor of poor cognitive status, other components of MetS, such as elevated blood pressure, fasting blood sugar, and triglycerides, did not show significant associations. This finding is intriguing and suggests that the relationship between MetS components and CI may not be straightforward. Elevated blood pressure and blood sugar are well-documented risk factors for cognitive decline due to their effects on cerebral blood flow and glucose metabolism [33, 52]. However, the lack of significant associations in this study might be due to several reasons, including sample size limitations, measurement variability, or the presence of other unmeasured confounding factors such as diet, physical activity, and genetic predispositions [53, 54].

## Conclusion

This study highlights the high prevalence of MetS (32.2%) and CI (44%) among older adults in Calabar Metropolis, with a notable association between reduced HDL levels and poor cognitive status. Whilst MetS was not significantly associated with CI, the findings emphasize the importance of managing specific metabolic risk factors, particularly HDL, to maintain cognitive health in aging populations. Further research should explore the complex interactions between various metabolic syndrome components and cognitive functions, employing larger sample sizes and longitudinal designs to better understand causality and the underlying mechanisms.

### Recommendations

To develop effective prevention and intervention strategies, several approaches can be recommended based on our findings:

i. Interventions focused on improving HDL levels should include promoting physical activity and a

diet rich in healthy fats. Public health campaigns targeting older adults could emphasize these lifestyle changes.

ii. Routine cognitive assessments, particularly for older adults with low HDL levels, could help in the early detection and management of CI. This could be integrated into regular health check-ups in primary care settings.

iii. For postmenopausal women, further research into the benefits of hormone replacement therapy (HRT) on cognitive function may be warranted, although the risks and benefits must be carefully weighed on an individual basis.

#### Abbreviations

| | |
|---|---|
| MetS | Metabolic syndrome |
| SPB | Systolic blood pressure |
| DBP | Diastolic blood pressure, FBS: Fasting blood sugar |
| HDL | High density lipoprotein |
| TG | Triglyceride: CVD: Cardiovascular disease |
| HOMA-IR | Homeostatic model assessment for insulin resistance |
| WHR | Waist hip ratio |
| AIP | Atherogenic index of plasma |

#### Author contributions

IKI, KJE and IEB designed the study. MEJ, UPO, IUK, and GOA carried out the participant recruitment and field surveys. KJE and UPO conducted the data collection and statistical analysis. IKI, KJE and IEB wrote the manuscript. All authors read and approved the final manuscript.

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This study did not receive any funding.

#### Data availability

The dataset generated and/or analyzed during this study are not publicly available due to threats to participant privacy but are available from the corresponding author on reasonable request.

## Declarations

#### Ethics approval and consent to participate

Ethical approval for this study was obtained from the Health Research Ethics Committee (HREC) with reference number (Rec No. CRSMOH/REC/2022/235) of the Ministry of Health, Calabar, Cross River State. All respondents gave written informed consent and information supplied by the respondents were kept highly confidential including their test results.

#### Consent for publication

Not applicable.

#### Competing interests

The authors declare no competing interests.

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#### References

1. Akintayo-Usman NO, Usman SO. Comparative analysis of ageing in Nigeria and United Kingdom using life course approach: the implication for the nursing profession in Nigeria. Pan Afr Med J. 2021;38:411.
2. Mbam KC, Halvorsen CJ, Okoye UO. Aging in Nigeria: a growing Population of older adults requires the implementation of National Aging policies. Gerontologist. 2022;62(9):1243–50. https://doi.org/10.1093/geront/gnac121.
3. Noto S. Perspectives on aging and quality of life. Healthc (Basel). 2023;11(15):2131. https://doi.org/10.3390/healthcare11152131.
4. Petersen RC. Clinical practice. Mild cognitive impairment. N Engl J Med. 2011;364(23):2227–34.
5. Cheng Y, Xiao S. Recent research about mild cognitive impairment in China. Shanghai Arch Psychiatry. 2014;26(1):4–14.
6. Baiyewu O, Unverzagt FW, Ogunniyi A, Hall KS, Gureje O, Gao S, et al. Cognitive impairment in community-dwelling older nigerians: clinical correlates and stability of diagnosis. Eur J Neurol. 2002;9(6):573–80.
7. Yusuf AJ, Baiyewu O, Sheikh TL, Shehu AU. Prevalence of dementia and dementia subtypes among community-dwelling elderly people in northern Nigeria. Int Psychogeriatr. 2010;23(3):379–86.
8. Gureje O, Ogunniyi A, Kola L. The profile and impact of probable dementia in a sub-saharan African community: results from the Ibadan Study of Aging. J Psychosom Res. 2006;61(3):327–33.
9. Ochayi B, Thacher TD. Risk factors for dementia in central Nigeria. Aging Ment Health. 2006;10(6):616–20.
10. Uwakwe R. The pattern of psychiatric disorders among the aged in a selected community in Nigeria. Int J Geriatr Psychiatry. 2000;15(9):873–80.
11. Bahchevanov KM, Dzhambov AM, Chompalov KA, Massaldjieva RI, Atanassova PA, Mitkov MD. Contribution of components of metabolic syndrome to cognitive performance in middle-aged adults. Arch Clin Neuropsychol. 2021;36(4):498–506. https://doi.org/10.1093/arclin/acaa081.
12. Song QB, Zhao Y, Liu YQ, et al. Sex difference in the prevalence of metabolic syndrome and cardiovascular-related risk factors in urban adults from 33 communities of China: the CHPSNE study. Diabetes Vasc Dis Res. 2015;12(3):189–98. https://doi.org/10.1177/1479164115576860.
13. Beltrán-Sánchez H, Harhay MO, Harhay MM, et al. Prevalence and trends of metabolic syndrome in the adult U.S. population, 1999–2010. J Am Coll Cardiol. 2013;62(8):697–703.
14. Lee SE, Han K, Kang YM, et al. Trends in the prevalence of metabolic syndrome and its components in South Korea: findings from the Korean National Health Insurance Service database. PLoS ONE. 2018;13(3):e0194490. https://doi.org/10.1371/journal.pone.0194490.
15. Adejumo EN, Adejumo AO, Azenabor A, Ekun AO, Enitan SS, Adebola OK, et al. Anthropometric parameter that best predict metabolic syndrome in South West Nigeria. Diabetes Metab Syndr. 2019;13(1):48–54. PMID: 30641748.
16. Annani-Akollor ME, Laing EF, Addai-Mensah O, Fondjo LA, Adu EA, Owiredu EW. Population-derived cut-off for HbA1c could enhance the identification of metabolic syndrome among non-diabetic population. Heliyon. 2020;6(8):e04583. PMID: 32775749.
17. Abourazzak F, Talbi S, Lazrak F, Azzouzi H, Aradoini N, Keita S et al. Does metabolic syndrome or its individual components affect pain and function in knee osteoarthritis women? Curr Rheumatol Rev. 2015. Advance online publication. PMID: 26002459.
18. Aradoini N, Keita S. Menopause and metabolic syndrome in Tunisian women. Biomed Res Int. 2014;457131.
19. Razay G, Vreugdenhil A, Wilcock G. The metabolic syndrome and Alzheimer disease. Arch Neurol. 2007;64(1):93–6. https://doi.org/10.1001/archneur.64.1.93.
20. Vanhanen M, Koivisto K, Moilanen L, Helkala EL, Hanninen T, Soininen H, et al. Association of metabolic syndrome with Alzheimer disease: a population-based study. Neurology. 2006;67(5):843–7. https://doi.org/10.1212/01.wnl.0000234037.91185.99.
21. Kalmijn S, Foley D, White L, Burchfiel CM, Curb JD, Petrovitch H, et al. Metabolic cardiovascular syndrome and risk of dementia in Japanese-American elderly men: the Honolulu-Asia Aging Study. Arterioscler Thromb Vasc Biol. 2000;20(10):2255–60. https://doi.org/10.1161/01.atv.20.10.2255.
22. Solfrizzi V, Scafato E, Capurso C, D'Introno A, Colacicco AM, Frisardi V, et al. Metabolic syndrome and the risk of vascular dementia: the Italian longitudinal study on aging. J Neurol Neurosurg Psychiatry. 2010;81(4):433–40. https://doi.org/10.1136/jnnp.2009.181743.
23. Dell R, Holleran S, Ramakrishnan R. Sample size determination. J Inst Lab Anim Res. 2002;43:207–13.

24. Lott JA, Turner K. Evaluation of Trinder's glucose oxidase method for measuring glucose in serum and urine. Clin Chem. 1975;21(12):1754–60.
25. Cieniewicz AM, Kirchner T, Hinke SA, Nanjunda R, D'Aquino K, Boayke K, Cooper PR, Perkinson R, Chiu ML, Jarantow S, Johnson DL. Novel monoclonal antibody is an allosteric insulin receptor antagonist that induces insulin resistance. Diabetes. 2017;66(1):206–17.
26. Jan NA, Mahmood R, Wasi N, Jan NA, Anjum N, Salahuddin H. Evaluation and Comparison of Lipid Profile in Pregnancy and Preeclampsia. Annals Of Abbasi Shaheed Hospital And Karachi Medical & Dental College. 2019;24(4):231-7.
27. Vogeser M, König D, Frey I, Predel HG, Parhofer KG, Berg A. Fasting serum insulin and the homeostasis model of insulin resistance (HOMA-IR) in the monitoring of lifestyle interventions in obese persons. Clin Biochem. 2007;40(13–14):964–8.
28. Kim JJ, Yoon J, Lee YJ, Park B, Jung DH. Predictive value of the atherogenic index of plasma (AIP) for the risk of incident ischemic heart disease among non-diabetic koreans. Nutrients. 2021;13(9):3231.
29. Borson S, Scanlan J, Brush M, Vitaliano P, Dokmak A. The mini-cog: a cognitive 'vital signs' measure for dementia screening in multi-lingual elderly. Int J Geriatr Psychiatry. 2000;15(11):1021–7. https://doi.org/10.1002/1099-1166(20001)15:11<1021::aid-gps234>3.0.co;2-6.
30. Abayomi SN, Sritharan P, Yan E, Saripella A, Alhamdah Y, Englesakis M, et al. The diagnostic accuracy of the Mini-cog screening tool for the detection of cognitive impairment—A systematic review and meta-analysis. PLoS ONE. 2024;19(3). https://doi.org/10.1371/journal.pone.0298686.
31. Isong IK, Emmanuel KJ, Abam GO, Bassey IE, Jackson ME, Obadare UP, KokoAbasi IU. Assessment of subnutritional indices and associated risk factors of malnutrition among older adults. Gerontol Geriatr Med. 2024;10:23337214241279636. https://doi.org/10.1177/23337214241279636.
32. Borson S, Scanlan JM, Watanabe J, Tu SP, Lessig M. Simplifying detection of cognitive impairment: comparison of the Mini-cog and Mini-mental State examination in a multiethnic sample. J Am Geriatr Soc. 2005;53(5):871–4. https://doi.org/10.1111/j.1532-5415.2005.53269.x.
33. Wang X, Ji L, Tang Z, et al. The association of metabolic syndrome and cognitive impairment in Jidong of China: a cross-sectional study. BMC Endocr Disord. 2021;21:40. https://doi.org/10.1186/s12902-021-00705-w.
34. Liu CY, Zhou HD, Xu ZQ, Zhang WW, Li XY, Zhao J. Metabolic syndrome and cognitive impairment amongst elderly people in Chinese population: a cross-sectional study. Eur J Neurol. 2009;16(9):1022–7. https://doi.org/10.1111/j.1468-1331.2009.02640.x. Epub 2009 Apr 16. PMID: 19473351.
35. Prince M, Bryce R, Albanese E, Wimo A, Ribeiro W, Ferri CP. The global prevalence of dementia: a systematic review and meta-analysis. Alzheimers Dement. 2013;9(1):63–75.e2. doi: 10.1016/j.jalz.2012.11.007. PMID: 23305823.
36. Lovre D, Lindsey SH, Mauvais-Jarvis F. Effect of menopausal hormone therapy on components of the metabolic syndrome. Ther Adv Cardiovasc Dis. 2016;11(1):33–43. doi: 10.1177/1753944716649358. Epub ahead of print. PMID: 27234158; PMCID: PMC5933555.
37. Carr MC, Brunzell JD. Abdominal obesity and dyslipidemia in the metabolic syndrome: importance of type 2 diabetes and familial combined hyperlipidemia in coronary artery disease risk. J Clin Endocrinol Metab. 2004;89(6):2601–7. https://doi.org/10.1210/jc.2004-0432.
38. Sochocka M, Karska J, Pszczołowska M, Ochnik M, Fułek M, Fułek K, Kurpas D, Chojdak-Łukasiewicz J, Rosner-Tenerowicz A, Leszek J. Cognitive decline in early and premature menopause. Int J Mol Sci. 2023;24:6566. https://doi.org/10.3390/ijms24076566.
39. Rettberg JR, Yao J, Brinton RD. Estrogen: a master regulator of bioenergetic systems in the brain and body. Front Neuroendocrinol. 2014;35(1):8–30. https://doi.org/10.1016/j.yfrne.2013.08.001.
40. Alberti KG, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JI, Donato KA, the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association. Harmonizing the metabolic syndrome: a joint interim statement of. Circulation. 2009;120(16):1640–5. https://doi.org/10.1161/CIRCULATIONAHA.109.192644. World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity.
41. Varghese RP, Vijaya Kumar PA, Chand S. Assessment of metabolic syndrome and its components in patients with acute coronary syndrome. Clin Epidemiol Glob Health. 2023;22:101341. https://doi.org/10.1016/j.cegh.2023.101341.
42. Craft S. The role of metabolic disorders in Alzheimer disease and vascular dementia: two roads converged. Arch Neurol. 2009;66(3):300–5. https://doi.org/10.1001/archneurol.2009.27.
43. Chang YM, Lee CL, Wang JS. Sex disparity in the association of metabolic syndrome with cognitive impairment. J Clin Med. 2024;13(9):2571. https://doi.org/10.3390/jcm13092571.
44. Alsuwaidi HN, Ahmed AI, Alkorbi HA, Ali SM, Altarawneh LN, Uddin SI, Roueentan SR, Alhitmi AA, Djouhri L, Chivese T. Association between metabolic syndrome and decline in cognitive function: a cross-sectional study. Diabetes Metab Syndr Obes. 2023;16:849–59. https://doi.org/10.2147/DMSO.S393282.
45. McEvoy CT, Laughlin GA, Barrett-Connor E, Bergstrom J, Kritz-Silverstein D. Metabolic syndrome and 16-year cognitive decline in community-dwelling older adults. Ann Epidemiol. 2012;22(5):310–7. https://doi.org/10.1016/j.annepidem.2011.12.003.
46. Deckers K, van Boxtel MP, Schiepers OJ, de Vugt M, Muñoz Sánchez JL, Anstey KJ, et al. Target risk factors for dementia prevention: a systematic review and Delphi consensus study on the evidence from observational studies. Int J Geriatr Psychiatry. 2015;30(3):234–46. https://doi.org/10.1002/gps.4245.
47. Feinkohl I, Janke J, Hadzidiakos D, et al. Associations of the metabolic syndrome and its components with cognitive impairment in older adults. BMC Geriatr. 2019;19:77. https://doi.org/10.1186/s12877-019-1073-7.
48. Gordon DJ, Rifkind BM. High-density lipoprotein—the clinical implications of recent studies. N Engl J Med. 1989;321(19):1311–6. https://doi.org/10.1056/NEJM198911093211907.
49. Barter P, Gotto AM, LaRosa JC, Maroni J, Szarek M, Grundy SM, et al. HDL cholesterol, very low levels of LDL cholesterol, and cardiovascular events. N Engl J Med. 2007;357(13):1301–10. https://doi.org/10.1056/NEJMoa064278.
50. Singh-Manoux A, Gimeno D, Kivimaki M, Brunner E, Marmot MG. Low HDL cholesterol is a risk factor for deficit and decline in memory in midlife: the Whitehall II study. Arterioscler Thromb Vasc Biol. 2008;28(8):1556–62. https://doi.org/10.1161/ATVBAHA.108.163998.
51. van Vliet P. Cholesterol and late-life cognitive decline. J Alzheimers Dis. 2012;30(S2):S147–62. https://doi.org/10.3233/JAD-2011-111028.
52. Tzourio C, Laurent S, Debette S. Is hypertension associated with an accelerated aging of the brain? Hypertension. 2014;63(5):894–903. https://doi.org/10.1161/HYPERTENSIONAHA.113.00147.
53. Nguyen JC, Killcross AS, Jenkins TA. Obesity and cognitive decline: role of inflammation and vascular changes. Front Neurosci. 2014;5:75. https://doi.org/10.3389/fnins.2014.00375.
54. de Almeida Faria AC, Dall'Agnol JF, Gouveia AM, et al. Risk factors for cognitive decline in type 2 diabetes mellitus patients in Brazil: a prospective observational study. Diabetol Metab Syndr. 2022;14:105. https://doi.org/10.1186/s13098-022-00872-3.


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