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Association between Visceral Adiposity and Cognitive Function: A Cross-Sectional Analysis

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KEYWORDS

cognitive functions, visceral adiposity, visceral Fat, Bioelectrical impedance analysis

ABSTRACT:

Background: Visceral adiposity has been increasingly recognized as a metabolically active fat depot linked to systemic inflammation and vascular dysfunctionfactors that may contribute to cognitive decline. However, evidence on its direct association with specific cognitive domains remains limited.

Objective: To examine the relationship between visceral adiposity and cognitive performance across key domains, including attention, perception, executive function, and working memory.

Methods: In this cross-sectional study, visceral adiposity was assessed using standardized anthropometric and body composition measures. Cognitive performance was evaluated through a series of validated online tasks assessing attentional, perceptual, executive, and working memory functions. Statistical analysis included calculation of mean values and standard deviations, and group comparisons were performed using an unpaired t-test.

Results: Higher levels of visceral adiposity were associated with significantly lower performance in multiple cognitive domains. Participants with greater visceral fat demonstrated reduced attentional accuracy, slower perceptual processing, and poorer executive functioning and working memory scores ($p < 0.05$).

Conclusion: The findings suggest a significant inverse association between visceral adiposity and cognitive performance. These results highlight the potential impact of excess visceral fat on cognitive health and underscore the importance of early lifestyle interventions aimed at reducing visceral adiposity to preserve cognitive function.

INTRODUCTION:

Obesity is a complex condition that arises when the body consumes more energy than it expends, contributing to both immediate and long-term health concerns and ultimately shortening life expectancy. [1] A variety of influences—such as shifts in social and economic environments, reduced physical activity, and the widespread intake of calorie-dense, high-fat foodshave fuelled the rapid global increase in obesity and its associated complications.This imbalance between energy intake and expenditure gives rise to numerous comorbid conditions, including hypertension, diabetes, and coronary artery disease. In addition to its physical consequences, obesity has been linked to cognitive difficulties, impaired motor function, low self-esteem, depression, social challenges, and a heightened risk of developing Alzheimer's disease. [2]

Central obesity, characterized by an accumulation of fat in the abdominal region, may arise from subcutaneous fat, visceral fat, or a combination of the two. Among these, visceral fat is the primary contributor to the metabolic complications commonly associated with obesity. [3] A rise in visceral fat surrounding the abdomen and internal organs triggers chronic, low-grade inflammation, which can lead to conditions such as dyslipidaemia, decreased insulin sensitivity, elevated insulin levels, high blood glucose, and hypertension.[4]Obesity has additionally been found to contribute to structural alterations in the brain, potentially hastening cognitive decline and raising the likelihood of dementia.[5]

Bioelectrical Impedance Analysis (BIA) is a simple, cost-effective method for estimating body composition, particularly body fat percentage. It operates by

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measuring the resistance of body tissues to a low-level electrical current. This measured resistance is used to estimate total body water (TBW), which forms the basis for calculating fat-free mass. [6]Body fat mass is then derived by subtracting fat-free mass from the individual's total body weight. Studies has shown it to more accurate than BMI.[7]

Cognitive function covers a broad set of skills, including memory, attention, decision-making, and motor abilities, and it involves advanced processes such as planning, regulation, and goal-directed behaviour.[8] Although obesity has been connected to a higher risk of dementia, its impact on cognitive performance in individuals who have not developed dementia remains unclear.[9] This study seeks to investigate how central obesity relates to cognitive health, with the aim of informing early interventionssuch as weight control, lifestyle modification, and increased physical activity—to help lower the future risk of dementia and Alzheimer's disease.

MATERIALS AND METHODS:

Study Design: This is a cross-sectional observational study. conducted at Department of physiology MGM Medical College Ch. Sambhajinagar. After the Institutional Ethical Committee clearance was obtained. **Sample Size:** 200. **Samplepopulation:** healthcare students from campus (Male & Female) aged 18–30 years, divided intonon-obese (BMI < 25 group-A) and obese (BMI > 25 group-B).

Inclusion criteria: Healthy non-obese and obese participants willing to enroll in the study.**Exclusion criteria:** Individuals with anxiety, addiction, medication use, or medical/ neurologicaldisorders.

For anthropometric data, height was measured by wall mounted stadiometer, weight by digitalweighing scale and BMIwas calculatedbydividing weight in kilograms by the square of height in meters (kg/m²) and subjects were divided into non-obese and obese group. Resting blood pressure was recorded by digital sphygmomanometer. Cognitive tests for Attention, perception, executive function, and working memory were evaluated using the online tool cognitivefun.net.

Attentional tasks were evaluated through Go/No-Go - in attention task, response speed and accuracy are evaluated using a Go–No-Go test. Participants click when a solid green dot appears and withhold responses to patterned dots. Reaction times for each stimulus are recorded in milliseconds to assess attentional control and cognitive processing.

Perception tasks were assessed using fast counting (FC)- Participants briefly view a set of dots and must quickly press the number key corresponding to the quantity shown, which ranges from four to seven. The average response time across 12 trials is recorded, providing a measure of subitizing efficiency.

Executive tasks were measured usingStroop Test (ST) for color interference reading, Participants must identify the colour in which a word is printed while ignoring the written word itself. When the colour and word are incongruent, interference occurs, engaging the anterior cingulate cortex, which detects such conflicts. The test records both the response time and the percentage of correct colour identifications, and results are generated after 12 trials.

Working memory task was evaluatedthrough picture 2-back remembering, in this task, participants view a sequence of stimuli and must judge whether the current item matches the one presented *N* steps earlier. The test begins with the 2-back condition, requiring subjects to identify the picture that appeared two positions earlier in the sequence. A total of 30 trials is administered, after which the performance results are displayed.

STATISTICAL ANALYSIS –

Data were entered into Microsoft Excel, and the mean and standard deviation of quantitative variables were calculated. Statistical significance was assessed using an unpaired t-test. For comparison between anthropometricparameters and cognitive functions in groups, the Pearson correlation coefficient test was used.

OBSERVATION & RESULTS

: A total of 200 health care subjects who fulfilled inclusion and exclusion criteria were enrolled in the study. Group A comprised 100 non-obese subjects, while Group B consisted of 100 obese subjects.

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Table: 1: Comparison ofdemographic andanthropometric data between the groups

Parameters	Group-A (Mean±SD)	Group-B (Mean ± SD)	t-value	p- value
Age	20.52±1.95	20.07±1.73	1.715	0.0439(NS)
Gender	M-64; F-36	M-54; 46		
Height (cm)	165.65±7.40	165.81±6.25	0.165	0.43(NS)
Weight (kg)	60.52±5.98	78.62±11.14	14.234	< 0.0001 (S)
BMI (kg/m2)	21.84 ± 0.86	28.51 ± 2.99	20.949	< 0.0001 (S)
SBP (mm Hg)	112.66±4.95	129.38±6.09	21.179	< 0.0001 (S)
DBP(mm Hg)	71.81±6.27	85.31±1.92	20.477	< 0.0001 (S)

Table: 1 Both group subjects were matching age and height wise. Mean of Weight, BMI, SBP, DBP, in Group B was more than group A and it was statistically significant (p< 0.001) Blood pressure (both SBP and DBP) was statistically increased in group B obese subjects.

Table: 2: Comparison of Body fat distribution between the groups

Parameters	Group A (Mean±SD)	Group B (Mean±SD)	t-value	P-value
VF (%)	4.15 ± 1.11	11.41 ± 4.13	16.946	<0.0001(S)
SCF (%)	15.15 ± 4.12	27.68 ± 5.79	17.539	<0.0001(S)
TBF (%)	27.13 ± 3.98	34.45 ± 4.12	12.708	<0.0001(S)

(VF- Visceral Fat, SCF- Subcutaneous Fat, TBF – Total body Fat)

Table:2 shows obese group has statistically significantly higher VF, SCF and TBF than the non-obese group.

Table 3: Comparison of cognitive functions between the groups

Parameters	Group A Mean ± SD	Group B Mean ± SD	t-value	p-value
Attention	498.09 ± 71.09	672.05± 58.99	18.735	< 0.0001 (S)
Perception	(85%)901.05 ± 77.08	(65%)1050.90±101.18	11.721	< 0.0001 (S)
Execution	(94%)812.75 ± 98.35	(87%)990.44 ± 64.96	15.018	< 0.0001 (S)
Working memory	(60%)975.84 ±86.60	(53%)1074.34 ± 95.69	7.593	< 0.0001 (S)

Table 3: shows statistically significant increase in duration of cognitive functions (attention, perception, execution and working memory) and decreased correct responses (%) between the groups

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Table 4: Correlations between Visceral fat and duration of cognitive functions in obese group

Visceral Fat	r-value	p-value
Attention	0.288	0.0001(S)
Perception	0.191	0.013(S)
Execution	0.176	0.007(S)
Working memory	0.153	0.03(S)

Table 4: In obese group, VF shows significant positive correlation with duration of cognitive tests. Indicating impaired cognitive function with higher VF.

Discussion: This study set out to evaluate the degree of cognitive impairment and examine how it relates to visceral fat (VF) in young healthcare workers. The results showed clear differences in cognitive performance between obese and non-obese participants, suggesting the presence of cognitive dysfunction. VF was also found to be positively associated with cognitive function. Moreover, both systolic and diastolic blood pressure were significantly elevated in the obese group. This rise in blood pressure, driven by increased sympathetic activity, can cause thickening of vascular walls, which in turn narrows blood vessels and decreases cerebral blood flow. Such reduced perfusion to the brain may be a key factor in the cerebrovascular changes that contribute to cognitive decline.

The global prevalence of abnormal or excessive fat accumulationand the health complications associated with itcontinues to rise steadily.[1] Regardless of its underlying cause, obesity is linked to a wide range of medical problems. Increasingly sedentary lifestyles, along with the high consumption of fat-rich fast foods, have significantly contributed to the growing rates of obesity and its related disorders. Yet, some individuals appear more vulnerable to these changes and are more likely to become overweight. Difficulty controlling overeating or binge-eating behaviours may stem from reduced cognitive control efficiency.[2] Research has also demonstrated a connection between Western high-fat diets and impaired cognitive performance.[10]

Neuroimaging studies examining cognitive dysfunction in older adults with obesity reveal structural brain changes, including atrophy in the prefrontal cortex, cingulate gyrus, hippocampus, and thalamus.[11] Metabolic disturbances associated with obesity can also lead to vascular abnormalities, increasing the risk of dementia.[12] Elevated midlife BMI has been linked to lower cognitive function,[13,14]and when combined with metabolic abnormalities, it is associated with an even greater risk of dementia in later life.[15-17] Experimental research in juvenile and adult mice further supports these findings, showing that early exposure to high-fat diets reduces neurogenesis and negatively affects learning, memory, and overall cognition.[18]

Several additional mechanisms have been proposed to explain how obesity influences cognition, including structural alterations in the brain, leptin[19] and insulin[20]dysregulation, oxidative stress and inflammation,[21] impaired cerebrovascular function, disrupted leptin transport across the blood–brain barrier, and enhanced inflammatory signalling within the brain.[22]

While previous research on middle-aged and older adults has shown that higher BMI correlates with cognitive decline, these studies primarily relied on questionnaire-based assessments. In contrast, our study is the first to explore whether overweight and obese individuals in a younger age group with increase visceral fat exhibit cognitive deficits using an online cognitive testing approach.

Conclusions

The findings of this study highlight that obesity can be reliably identified using anthropometric measurements. Increased VF is significantly associated with a higher risk of cognitive decline. Based on our results, VF may serve as a convenient and effective tool for predicting obesity-related reductions in cognitive function.These findings emphasize the importance of recognizing the risk of cognitive decline associated with obesity at a younger age. Encouragingly, such decline may be

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reversible through early intervention strategies—including yoga, lifestyle modification, and weight reduction—which can help prevent long-term brain changes and reduce the future risk of dementia and Alzheimer's disease.

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