

Mild Cognitive Impairment in Obese Elderly

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Abstract

Background: Cognitive impairment and dementia are among the leading causes of disability and dependence in elderly having major economic burden for public health systems. The prevalence of mild cognitive impairment is 6.7% for ages 60–64, 8.4% for 65–69, 10.1% for 70–74, 14.8% for 75–79, and 25.2% for 80–84. The relationship between obesity and cognitive function is conflicting as some researchers found that obesity in mid-life is a predictor of mild cognitive impairment at old age while it is protective at late life, so detecting MCI early can prevent or predict further development to dementia.

Objective: To detect the prevalence of mild cognitive impairment in obese older adults in comparison to non-obese older adults.

Methods: A case control study of community dwellings elderly aged 60 and above, total 140 exceeding the sample size which was 130 divided into two groups: Group A: Obese (70) individual (35 females, 35 males) Group B: Non-obese (70) individual (35 females, 35 males), participants with education less than 12 years dementia, delirium, depression, functionally dependent, severe sensory impairment or communication problems, cerebrovascular insult, and acute medical condition were excluded.

Results: Cognitive tests Mini-Mental Status Examination (MMSE) and Montreal Cognitive Assessment (MOCA) were performed revealing no significant association between obesity and cognitive impairment in elderly individuals who had finished their secondary education.

Conclusion: obesity has no association with mild cognitive impairment in elderly individuals.

Keywords: obesity, elderly, body mass index, mild cognitive impairment.

Background

Mild cognitive impairment (MCI) is an intermediate state between the cognitive changes related to aging and very early dementia (Petersen et al.,2014) .

MCI does not necessarily lead to dementia (Sachdev et al.,2014). Among MCI individuals aged 65 years and older the incidence rates of reversion to normal cognition were up to 16% over 1 year in a clinic-based study (Koepsell & Monsell ,2012) and 28-55% in population-based studies over a 2-12 years (Roberts et al.,2014) .

Detection and recovery from MCI to normal cognition has important role for the prevention of dementia. This could be achieved through reduction of risk factors especially modifiable ones, as depression and social isolation (Kuiper et al., 2015), hypertension, diabetes, and smoking (Luchsinger & Gustafson, 2009). Enhancing the protective risk factors as listening to the radio, reading newspapers, reading magazines or journals, reading books, playing games like crosswords, or other puzzles and going to museums may also have a

role in MCI reversibility (Hughes,2010).

As regard obesity there is a debate about its relation to cognitive decline; whether it is a protective or a risk factor for cognitive decline and dementia in elderly (Gustafson et al.,2012).

It has been supposed that, obesity in mid-life is a risk factor for dementia (Fitzpatrick et al.,2009), but in late life being underweight is the risk factor (Burns et al.,2010).

Aim of the work:

To detect the prevalence of mild cognitive impairment in obese older adults in comparison to non-obese older adults.

Methods

A case control study of community dwellings elderly aged 60 and above , total 140 exceeding the sample size which is 130 divided into two groups: Group A: Obese

(70) individual (35 females ,35 males)Group B: Non-obese (70) individual (35 females ,35 males).

Inclusions criteria:

- Patients age > 60 or older.

Exclusion criteria:

- Less than 12 years education.
- Demented (MMSE <24).
- Depressed (GDS more than or equal 5).
- Functionally dependent.
- Delirium
- Severe sensory impairment or communication problems.
- Cerebrovascular insult or acute decompensating illness

Socio-demographic data were collected including level of education, occupation and marital status.

All participants underwent:

- Comprehensive geriatric assessment including complete medical history, physical examination, comorbidities review: (HTN, DM, Hyperlipidemia, Parkinson's disease, sleeping disorder, coronary heart disease), family history of Alzheimer's disease (AD), smoking were also recorded.
- Cognitive assessment using both the Mini mental status examination (MMSE) Arabic version (**EI-Okf, 2002**) and the Arabic version of Montreal Cognitive Assessment (MOCA) (**Abdel Rahman& El Gaafary , 2007**).
- Anthropometric measurements that include:
 - Body mass index (BMI), obesity was considered if BMI was 30 or more (**Keys et al.,1972**).
 - Mid upper arm circumference (MUAC) (**Lohman et al , 1988**).
 - Waist-hip ratio or waist-to-hip ratio (WHR) is the **dimensionless ratio** of the circumference of the **waist** to that of the **hips**. This is calculated as waist measurement divided by hip measurement ($W \div H$). Obese in females if > 0.8, and in males if > 0.9 (WHO, 2011).
 - Calf circumference: three measurements are taken at maximum horizontal distance around the left calf as the subject stand with weight distributed evenly on both feet (Lohman et al , 1988).
 - Functional assessment using activities of daily living (ADL) assessment (Katz, 1963) and instrumental activities of daily living (IADL) assessment (Lawton,1969).
 - Assessment of depression using geriatric depression scale (GDS) Arabic version (Chaaya et al.,2008)
 - The Mini Nutritional Assessment (MNA) using the Arabic version (Abd-Al-Atty et al.,2012).

Statistical Analysis:

The collected data was revised, coded, tabulated and

introduced to a PC using Statistical package for Social Science (**SPSS 25**). Data was presented and suitable analysis was done according to the type of data obtained for each parameter.

• **Descriptive statistics:**

Mean, Standard deviation (\pm SD) and range for parametric numerical data, while Median and Interquartile range (IQR) for non-parametric numerical data.

Frequency and percentage of non-numerical data.

• **Analytical statistics:**

1. **Student T** Test was used to assess the statistical significance of the difference between two study group means.
2. **Chi-Square test** was used to examine the relationship between two qualitative variables
3. **Fisher's exact test** was used to examine the relationship between two qualitative variables when the expected count is less than 5 in more than 20% of cells
4. **Correlation analysis (using Pearson's method)** to assess the strength of association between two quantitative variables. The correlation coefficient denoted symbolically "r" defines the strength (magnitude) and direction (positive or negative) of the linear relationship between two variables.

P- value: level of significance

-P>0.05: Non significant (NS).

-P< 0.05: Significant (S).

-P<0.01: Highly significant (HS).

Results:

A 140 community dwelling elder subjects aged 60 and above were enrolled in the study and were divided into 2 equal groups according to BMI; group A:cases (Obese) BMI \geq 30 group B: controls (Not Obese) BMI<30. Comparisons of sociodemographic characteristics of the two studied groups are shown in table (1). A comparison was done between obese and non-obese regarding total MMSE score and Total MOCA score together with each cognitive domain in it and it was found to be of no statistical significance , 26 obese individual had MCI with mean score of MOCA (26.36 \pm 3.46) and 26 of non-obese individual had MCI with MOCA score (25.90 \pm 3.30)Table (2)

In Table (3) a correlation was done between total MOCA score and its different cognitive domains and anthropometric measures a significant positive correlation was found between both weight, height and Mid Upper Arm Circumference (MUAC) and total score of MOCA (p=0.044) ,(p=0.001) and (p= 0.035) respectively. As regard cognitive domains, executive functions showed a significant positive correlation with weight (p=0.044) and height (p=0.001), while abstraction showed a significant positive correlation with height (p=0.024).

Naming showed a significant positive correlation with calf circumference (CC) (p=0.042) and MUAC (p=

Table (1) comparison of Sociodemographic characteristics of the two studied groups

Sociodemographic characteristics		BMI RESULT		Monte Carlo Fisher's Exact test	
		OBESE	NON OBESE	P-Value	Sig.
		Mean ± SD N (%)	Mean ± SD N (%)		
Age		68.60 ± 7.83	69.54 ± 7.04	0.455(T)	NS
Gender	Female	35 (50%)	35 (50%)	1.000(C)	NS
	Male	35 (50%)	35 (50%)		
occupation	high class professionals	1 (100%)	0 (0%)	0.238	NS
	middle class professionals	8 (72.73%)	3 (27.27%)		
	skilled workers	12 (60%)	8 (40%)		
	retired	34 (47.89%)	37 (52.11%)		
	not working	15 (40.54%)	22 (59.64%)		
Marital status	Married	54 (52.43%)	49 (47.57%)	0.287	NS
	Single	0 (0%)	0 (0%)		
	Widow/widower	16 (45.71%)	19 (54.29%)		
	Divorced	0 (0%)	2 (100%)		
Level of education	Secondary education	44 (52.38%)	40 (47.62%)	0.490	NS
	Post-secondary education	26 (46.43%)	30 (53.57%)		

Table (2): comparison between obese and non-obese as regards Performance in different cognitive tests and each cognitive domain

	BMI RESULT		Student t-test of sig.	
	OBESE	NON OBESE	P-Value	Sig.
	Mean ± SD N (%)	Mean ± SD N (%)		
Mini mental status examination (MMSE)	27.73±2.13	27.47±1.99	0.462	NS
Montreal Cognitive Assessment (MOCA)	26.36±3.46	25.90±3.30	0.426	NS
Visuospatial and Executive Functioning	4.23±1.14	4.07±1.12	0.413	NS
Naming	2.83±0.38	2.73±0.54	0.205	NS
Attention	4.79±1.02	4.76±1.01	0.868	NS
Language	2.96±0.20	2.97±0.17	0.652	NS
Abstraction	1.67±0.47	1.56±0.56	0.192	NS
Delayed Recall (Short-term Memory):	3.97±0.93	3.90±0.93	0.651	NS
Orientation	5.79±0.48	5.89±0.40	0.182	NS

0.038)

Attention and language has no significant correlation with any of the anthropometric measures

Delayed Recall showed a significant positive correlation with height (p=0.038).Orientation showed a significant positive correlation with height (p=0.013)

To exclude the effect of comorbidities and habits special on cognitive functions in obese individuals, the relationship between them was studied in table 4. There was no significant association between comorbidities in obese subjects and cognition. Finally table (5) shows that among non-obese diabetic patients are cognitively impaired compared to non-diabetic (p=0.001).

Discussion

Cognitive impairment and dementia are among the leading causes of disability and dependence in elderly having major economic burden for public health systems. [Gustavsson et al., \(2011\)](#). Mild cognitive impairment is an intermediate stage between the normal aging and dementia ([Petersen et al.,2014](#)). Identifying individuals with MCI helps in detection of patients at high risk of developing dementia, thus helps in developing a potential therapeutic window and increases the significance of controlling the modifiable risk factors ([Pandya et al .,2017](#)).

Table (3) correlation between obesity parameters (BMI, anthropometric measures) and different cognitive domains

		Montreal Cognitive Assessment (MOCA)	Visuospatial and Executive Functioning	Naming	Attention	Language	Abstraction	Delayed Recall (Short-term Memory):	Orientation
Weight WT	r	0.170	0.171	0.125	0.123	0.017	0.166	0.105	-0.065
	P-Value	0.044	0.044	0.142	0.149	0.840	0.050	0.218	0.447
	Sig.	S	S	NS	NS	NS	NS	NS	NS
Height HT	r	0.268	0.275	0.097	0.163	0.044	0.191	0.176	0.210
	P-Value	0.001	0.001	0.256	0.055	0.604	0.024	0.038	0.013
	Sig.	S	S	NS	NS	NS	S	S	S
Body mass index (BMI)	r	0.082	0.100	0.069	0.048	-0.029	0.090	0.046	-0.076
	P-Value	0.338	0.238	0.418	0.574	0.733	0.289	0.592	0.373
	Sig.	NS	NS	NS	NS	NS	NS	NS	NS
Waist	r	0.100	0.069	0.078	0.060	0.045	0.155	0.069	-0.019
	P-Value	0.242	0.417	0.362	0.482	0.600	0.067	0.420	0.828
	Sig.	NS	NS	NS	NS	NS	NS	NS	NS
Hip	r	0.096	0.061	0.096	0.085	-0.085	0.127	0.090	-0.038
	P-Value	0.262	0.472	0.261	0.316	0.317	0.134	0.292	0.657
	Sig.	NS	NS	NS	NS	NS	NS	NS	NS
waist Hip ratio WHR	r	0.100	0.066	-0.144	0.099	0.039	0.069	0.095	.037
	P-Value	0.242	0.438	0.089	0.247	0.644	0.418	0.265	.668
	Sig.	NS	NS	NS	NS	NS	NS	NS	NS
Mid upper arm circumference MUAC	r	0.178	0.141	0.175	0.124	0.057	0.147	0.160	-0.059
	P-Value	0.035	0.096	0.038	0.143	0.506	0.083	0.060	0.492
	Sig.	S	NS	S	NS	NS	NS	NS	NS
Calf circumference CC	r	0.154	0.111	0.172	0.118	0.020	0.113	0.132	-0.071
	P-Value	0.069	0.192	0.042	0.165	0.814	0.186	0.120	0.404
	Sig.	NS	NS	S	NS	NS	NS	NS	NS

Table (4) Relationship between comorbidities, special habits and cognition in obese elderly

Obese	MOCA RESULT		Monte Carlo Fisher's Exact test	
	NORMAL	cognitive impairment	P-Value	Sig.
Smoking	N (%)	N (%)		
	5(83.33%)	1(16.67%)	0.278	NS
Ex-smoker	2(40.00%)	3(60.00%)	0.272	NS
Shisha SMOKING	5(100.00%)	0(0.00%)	0.074	NS
Alcohol USE	1(50.00%)	1(50.00%)	0.703	NS
Substance abuse	0(0.00%)	1(100.00%)	0.190	NS
Family history of Dementia	0(0.00%)	1(100.00%)	0.190	NS
Diabetes mellitus	13(72.22%)	5(27.78%)	0.340	NS
Hypertension	9(50.00%)	9(50.00%)	0.190	NS
Ischemic heart disease	6(50.00%)	6(50.00%)	0.311	NS
Heart failure	6(54.55%)	5(45.45%)	0.534	NS
Parkinson's disease	0(0.00%)	0(0.00%)		
Sleeping disorder	3(100.00%)	0(0.00%)	0.174	NS
COPD	11(78.57%)	3(21.43%)	0.174	NS
Renal diseases	5(71.43%)	2(28.57%)	0.621	NS
Liver diseases	5(62.50%)	3(37.50%)	0.982	NS
Visual or hearing impairment	6(85.71%)	1(14.29%)	0.187	NS

Table (5) Relationship between comorbidities, special habits and cognition in non- obese elderly

Non Obese	MOCA RESULT		Monte Carlo Fisher's Exact test	
	NORMAL N (%)	cognitive impairment N (%)	P-Value	Sig.
Smoking	7(77.78%)	2(22.22%)	0.321	NS
Ex-smoker	3(50.00%)	3(50.00%)	0.495	NS
Shisha smoker	4(80.00%)	1(20.00%)	0.410	NS
Alcohol use	0(0.00%)	0(0.00%)		
Substance abuse	0(0.00%)	0(0.00%)		
Family history of Dementia	1(100.00%)	0(0.00%)	0.439	NS
Diabetes mellitus	5(29.41%)	12(70.59%)	0.001	S
Hypertension	15(65.22%)	8(34.78%)	0.775	NS
Ischemic heart disease	5(62.50%)	3(37.50%)	0.982	NS
Heart failure	2(66.67%)	1(33.33%)	0.889	NS
Parkinson's disease	0(0.00%)	0(0.00%)		
Sleeping disorder	0(0.00%)	0(0.00%)		
COPD	12(66.67%)	6(33.33%)	0.698	NS
Renal diseases	5(71.43%)	2(28.57%)	0.621	NS
Liver diseases	11(55.00%)	9(45.00%)	0.390	NS
Visual or hearing impairment	1(50.00%)	1(50.00%)	0.703	NS

COPD = chronic obstructive pulmonary disease

The association between BMI and cognitive function is weak and complex in elderly(**Dahl et al .,2014**) , due to inaccurate adiposity measurement in the older adults due to age related body composition (**Smith et al .,2011**).

This study aimed to detect the prevalence of mild cognitive impairment in obese older adults in comparison to non-obese older adults. In the present study there was not a significant relation between obesity and mild cognitive impairment in elderly individuals who had finished their secondary education (illiterate elders were excluded from the study). The findings of this study are at odds with those of many studies carried out that have shown obesity to be associated with lower risk of cognitive impairment like the Chinese observational study that has been performed on elderly with age 60 years and older conducted by (**Hou et al .,2019**). Another study conducted in Yogyakarta, Indonesia showed that obesity in elderly individuals aged 65 and more was less likely associated with mild cognitive impairment (**Vidyanti et al .,2020**). Our small sample size may relatively led to the inability to observe this minimal association.

Also in our study, abdominal obesity, represented by waist hip ratio, was not associated significantly with cognitive impairment, which is not consistent with

(**Hou et al .2019**) for elderly individuals (≥ 65 years) where increased WHR was associated with increased risk of cognitive impairment and this may be due to the negative correlation between waist-to-hip ratio and hippocampal volume and the positive correlation between waist to-hip ratio and white matter hyper intensities (**Jagust et al .,2005**). We did not perform MRI for our participants to compare between hippocampal volume or white matter hyper intensities in obese and non-obese and correlate them to MCI.

Several studies like a cohort study conducted in the USA by(**Suemoto et al.,2015**) to cognitively normal individuals in their late 50s reported that obesity in later life was associated with a lower risk of cognitive impairment, while obesity in midlife was associated with a higher risk of cognitive impairment and this is called obesity paradox.

Obesity in late-life was associated with functional brain connectivity which acts as a neuroprotection for cognition (**Hsu et al.,2015**). The possible mechanisms that may explain the beneficial role of overweight on cognition in late life are the hormone leptin, that act as a cognitive enhancer by regulating hippocampal synaptic plasticity and β -amyloid processing(**Oomura et al.,2006**).

Midlife obesity fasten cognitive aging, but this is

weaker in older adulthood (**Dahl & Hassing, 2013**). A genome wide association study (GWAS) found that the fat mass and obesity associated gene (FTO) located on chromosome 16 was associated with increased body mass index (BMI) in both childhood and midlife (**Frayling et al., 2007**), the FTO gene was found to be associated with decreased brain volume in cognitively healthy elderly subjects (**Biesselset al., 2006**). Also midlife obesity is related to brain pathology like hypoperfusion, neuronal injury and death (**Gorelick et al., 2011**) and increased levels of β -amyloid precursor protein (**Lee et al., 2009**).

Some studies have reported associations of lower late-life BMI and faster decline in weight or BMI in late life with increased risk of dementia and cognitive decline and this may be due to the deposition of AD pathology in areas of the brain that regulate body composition (**Fitzpatrick et al., 2009**).

Regarding other anthropometric measures:

In our study height showed a significant positive correlation with the total score of MOCA and with most cognitive domains like abstraction, delayed recall, orientation and executive functions, this means that superior height values are associated with better cognitive performance. This is may be because both cognition and adult body height are strongly affected by early-life events. socially higher height is associated with better education and social success, that may affect health by providing better social status and socio-economic environment so a better cognition in adult life (**Huang et al., 2015**). The association between height and cognition may be explained by genetic factors (**Joshi et al., 2015**).

In this context, some studies revealed that height itself is a predictor of cognition. Adults with short stature display worst cognitive performance and at higher risk of early onset dementia and dementia-related death (**Russ et al., 2014**).

Mid upper arm circumference (MUAC) showed a significant positive correlation with the total score of MOCA and naming and this may suggest that higher muscle mass is associated with better global cognition. As age-related decrease in muscle mass is associated with impaired cognitive function as small upper arm circumference is associated with weight loss, nutritional deficiency, and decreased physical activity (**Yoon et al., 2012**).

The results of our study showed no relationship between comorbidities like hypertension, CVD and special habits and mild cognitive impairment in obese and non-obese. This relation was studied to find the impact of comorbidities and special habits besides obesity. Our results was contradictory to those of the previous studies like the study conducted by (**Zou et al. 2014**) in southwest China where a total of 597 participants (60 years) from hospital and community

population were enrolled in the cross-sectional study, which showed that those with vascular risk factors were associated with a higher risk of cognitive impairment, probably this is due to the selection criteria to have patients with controlled comorbidities without complications or end organ damage.

In this study non obese participants who have DM showed a significant association with mild cognitive impairments and this agrees with the study conducted in Japan by (**Yamazaki et al. 2011**).

We believe that prevention is the first line of treatment so detecting individuals with MCI may be a life changing step to avoid the functional, social and psychological burdens of dementia

We had some limitations in our study, the measure of current BMI may not have detected lifetime or midlife BMI and the changes in BMI with aging. May be a high BMI in midlife, is more likely to be detrimental for cognitive function in later life, rather than current BMI. Future longitudinal studies will be needed to address other measures of body composition, and changes in body weight over time on heterogeneous groups. This will help to target health promotion recommendations for older adults.

Conclusion

Neither obesity based on BMI categories nor abdominal obesity defined by WHR cutoffs is associated with a higher risk of cognitive impairment among elderly. However, further multicentered, prospective cohort studies with a long-term follow-up are needed.

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