

Association between Body Mass Index and Cognitive Performance

Farzad Ashrafi¹, Mehran Arab Ahmadi¹, Behdad Behnam¹, Lida Shashaani², Afsaneh Zarghi¹

¹ Functional Neurosurgery Research Center, Shohada Tajrish Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran

² Department of Internal Medicine, Shohada Tajrish Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran

ABSTRACT

Background and Purpose: Because of well-established role of obesity in brain lesions, progressing cognitive deficits in obese patients has been recently suggested. In current study and for the first time, we aimed to assess cognition status in Iranian obese people and to compare it with non-obese individuals.

Methods: One hundred and eighteen consecutive patients with the different cardiovascular and metabolic primary complaints were assigned to obese group (n=25, 21.2%) and non-obese group (n=93, 78.8%). Cognitive status was assessed at initial visit using the Montreal Cognitive Assessment (MoCA) questionnaire.

Results: Mean of total cognitive score in obese patients was 20.04 ± 4.57 and in non-obese ones was 20.19 ± 5.32 with no difference ($p=0.886$). In total, 8.0% of obese patients and 20.4% of non-obese patients had normal cognitive function ($p=0.149$). No significant difference was also found in different subdomains of cognitive ability between obese and non-obese groups. None of the cognitive domains had significant association with BMI as the considered indicator for defining obesity. Based on multivariate linear regression modeling, obesity could not predict cognitive deficit ($\beta=0.034$, SE:1.10 $p=0.973$).

Conclusion: Our survey could not demonstrate an association between obesity and cognitive impairment in a sample of Iranian patients.

Keywords: Cognition; Obesity; Body Mass Index

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Correspondence to: Behdad Behnam, MD, Functional Neurosurgery Research Center, Shohada Tajrish Hospital, Shahid Beheshti University of Medical Sciences, Tehran, Iran; Mobile: +98.9196345886; E-mail: behdadbehnam10@yahoo.com

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INTRODUCTION

Obesity is a serious and life-threatening health problem throughout the world with a steadily increasing trend within recent decades¹. The main reason for this increasing trend of obesity include unhealthy dietary behaviors, sedentary lifestyle, low physical exercise, and high rate of some metabolic disturbances especially in children and young adults². Approximately, 18% of the young population whole of the world suffers obesity with higher prevalence in North and Central America and in Australia³. Obesity has been well identified as one of the main underlying fundamentals for damaging multi-organs

leading disturbances in metabolic pathways related to glucose and lipid metabolism as well as to blood pressure deregulation leading increased risk for coronary artery disease, cerebrovascular accidents, diabetes mellitus, heart failure, and even various types of cancers^{4,5}. In this regard, obesity has been produced as a major risk factor for coronary atherosclerosis leading higher rates of cardiac-related mortality and morbidity⁶. In fact, the close association between cognitive deficit and obesity has been recently shown that higher prevalence rate of as well as risk of dementias such as Alzheimer's disease in obese individuals has been well identified^{7,8}. Even,

the relationship between early life obesity and cognitive impairment in older ages has been indicated⁹. This fact seems to be more important in those populations with upward trend of overweight and obesity in young adults and children. Iran is one of the main areas of high prevalence rate of obesity among developing countries with the prevalence of 22% in young adults and 40% in middle-age individuals with higher rate in females than in males^{10,11}. According to this point, progressing trend for cognitive deficits in a notable number of our population can be predictable. In current study and for the first time, we aimed to assess cognition status in obese subjects and to compare it with non-obese individuals.

MATERIAL AND METHODS

Participants for the present study include 118 consecutive patients with the different cardiovascular and metabolic primary complaints that referred to our clinics for further clinical assessment. Those with previous history of head trauma, or cerebrovascular ischemic or hemorrhagic events were excluded from the study. Demographic and weight status information were collected by interviewing and visiting in the clinics. Body weight was measured by balance beam scale, height was measured using a standard stadiometer with the participant wearing no shoes, and body mass index (BMI) was calculated as weight in kilograms divided by the square of the height in meters. In this regard, obesity was defined according to the BMI cutoff point as higher than 30 kg/m². Thus, the study population was categorized as obese group (n=25, 21.2%) and non-obese group (n=93, 78.8%). Participants provided written informed assent and consent in accordance with the Institutional Research and Ethic Committee at the University.

Cognitive status of the participants was assessed at initial visiting by a structured interview using the Montreal Cognitive Assessment (MoCA) questionnaire¹². This questionnaire consists of seven cognitive sub-domains

assessing visuoconstructional ability, naming task, verbal abstraction task, attention, working memory, language and orientation to time. Time to administer the MoCA is about 10 minutes. The total possible score is 30 points; a score of 26 or above is considered normal.

For statistical analysis, quantitative variables were compared by the t-test and categorical variables by the chi-square test. Correlation between quantitative variables was tested by the Pearson's coefficient test. To assess the difference in cognitive ability between obese and non-obese subjects, the multivariate linear regression modeling was employed. For the statistical analysis, (SPSS Inc., Chicago, IL) was used. P values of 0.05 or less were considered statistically significant.

RESULTS

Among 118 study subjects, 25 were obese and 93 were non-obese that 68.0% and 64.5% were female, respectively (p=0.745). The mean age in obese group was 59.08±11.81 years and 61.62±10.02 years in non-obese group with no difference (p=0.331). Except for mean blood glucose that was significantly higher in obese group than in non-obese individuals (140.04±48.39 mg/dl versus 117.43±46.05 mg/dl, p=0.043). Other baseline parameters including lipid profile, systolic blood pressure, and serum HbA1c were similar between the two groups (Table 1).

Mean of total cognitive score in obese patients was 20.04±4.57 and in non-obese ones was 20.19±5.32 with no difference (p=0.886). In this regard, 8.0% of obese patients and 20.4% of non-obese patients had normal cognitive function (p=0.149). As shown in table two, no significant difference in different subdomains of cognitive ability between obese and non-obese groups. According to the Pearson's correlation test, none of the cognitive domains had significant association with BMI as the considered indicator for defining obesity. Based on multivariate linear regression modeling (table 3), obesity

Table 1. Baseline characteristics between obese and non-obese groups

Characteristics	Obese group (n = 25)	Non-obese group (n = 93)	P-value
Age, year	59.08±11.81	61.62±10.02	0.331
BMI, kg/m ²	32.94±2.76	25.43±2.38	< 0.001
LDL, mg/dl	105.33±29.57	109.55±38.86	0.558
Triglyceride, mg/dl	147.76±41.36	153.63±52.89	0.557
HDL, mg/dl	47.68±12.37	44.95±10.76	0.321
Cholesterol, mg/dl	182.56±28.64	185.23±43.18	0.715
HbA1c, %	6.80±1.14	6.46±1.41	0.278
Blood sugar, mg/dl	140.04±48.39	117.43± 6.05	0.043
Systolic BP, mmHg	132.60±16.59	127.78±18.54	0.216

Table 2. Mean scores for cognition domains in obese and non-obese groups

Characteristics	Obese group (n = 25)	Non-obese group (n = 93)	P-value
MoCA Score	20.04±4.57	20.19±5.32	0.886
Visuoconstructional	2.44±1.47	2.58±1.64	0.681
Naming	2.56±0.58	2.48±0.60	0.568
Memory	1.84±1.55	1.83±1.32	0.972
Attention	4.52±1.61	4.57±1.56	0.891
Language	1.36±0.76	1.57±1.16	0.282
Abstraction	0.92±0.81	0.89±0.79	0.880
Orientation	5.56±0.65	5.44±0.99	0.572

Table 3. Association of MoCA score and obesity adjusted for baseline variables

	Unstandardized Coefficients		t	P-value	95% Confidence Interval for B	
	B	Std. Error			Lower Bound	Upper Bound
Constant	47.326	5.081	9.315	0	37.256	57.396
Obesity	0.034	1.015	0.034	0.973	-1.977	2.045
Sex	-1.999	0.907	-2.204	0.030	-3.798	-0.201
Age	-0.168	0.041	-4.090	0	-.250	-0.087
LDL	-.079	0.039	-2.019	0.046	-.156	-0.001
Triglyceride	-0.011	0.010	-1.075	0.285	-0.032	0.009
Total Cholesterol	0.051	0.039	1.321	0.189	-0.026	0.128
Blood sugar	-0.014	0.009	-1.609	0.110	-0.032	0.003
Systolic blood pressure	-0.086	0.023	-3.710	0	-0.132	-0.040

could not predict level of cognitive deficit (beta=0.034, SE=1.015, p=0.973).

DISCUSSION

Various studies could indicate negative association between BMI measure and cognitive impairment emphasizing progressive cognitive deficit in obese individuals especially in advanced ages¹³⁻¹⁷. Most studies focused middle-aged people to assess this relationship that could show a direct association between obesity in Middle Ages even in young adolescence and mild to moderate cognitive deficit in older ages¹⁸⁻²⁰. In this regard, almost all domains of cognition can be affected by obesity. Different basic pathological reasons have been described to explain relation between obesity and increased risk for cognitive deficit. It has been well demonstrated that the accumulation of adipose tissue in neural tissues can potentially reduce volume of some brain areas leading generalized brain atrophy^{21,22}. Some studies evidenced lowering gray matter density in the post-central gyrus, putamen, middle frontal gyrus, and frontal lobe following adiposity accumulation²³. In other studies, lower gray matter density in frontal, occipital, and temporal lobes as well as in anterior lobe of cerebellum in those with higher BMI values could be revealed in imaging studies^{24,25}. Moreover, hippocampal formation as a main area responsible for learning and memory

can be adversely affected by increased level of adipose tissue²⁶.

No association between obesity and cognitive deficit in the present study could be affected by some reasons. First, our included subjects were randomly selected from a heterogeneous patients population who refer to clinics because of their metabolic and cardiovascular complaints and thus this high heterogeneity may be affect both obesity and cognition arms. Second, our limited sample size might affect the study power that despite a notable numerical difference in the frequency of normal cognitive function between obese and non-obese ones (8.0% versus 20.4%), this difference remained non-significant. In addition, the correct definition of obesity is now based on waist circumference measurement that because of unavailability of this marker in all subjects, we had to assess BMI and its definitive cutoff value to define obesity. According to the pointed reasons, further studies by employing larger sample size and including homogenous population subgroups are recommended.

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