folia medica

Folia Medica 67(5)
DOI: 10.3897/folmed.67.e155285

Research Article

Assessment of cognitive functions in obese and type 2 diabetic patients

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Received: 8 April 2025 ♦ **Accepted:** 10 June 2025 ♦ **Published:** 30 October 2025

Citation: Mahmoud NA, Eldin HAG, Mohamed RS, Tawfeek AA. Assessment of cognitive functions in obese and type 2 diabetic patients. Folia Med (Plovdiv) 2025;67(5):e155285. doi: 10.3897/folmed.67.e155285.

Abstract

Introduction: Obesity and diabetes have been associated with increased risk of cognitive disorders, including dementia and Alzheimer's disease**.**

Aim: Assess the influence of obesity and type 2 DM on cognitive functions.

Materials and methods: This case-control comparative study was conducted at El-Kasr Aini Cairo University Hospital. The sample size of the study comprised 228 subjects, including 57 non-diabetic lean patients (controls), 57 diabetic lean patients, 57 non-diabetic obese patients, and 57 diabetic obese patients, all between the ages of 40 and 60 years. The subjects were initially evaluated using the Mini-Mental State Examination (MMSE) to rule out any potential cognitive impairment. Subsequently, they underwent a structured interview, accompanied by a complete physical examination and BMI measurement. The study also assessed the effects of fasting and postprandial blood glucose and HbA1c levels. P300 amplitude and peak latencies were recorded using the standard auditory oddball paradigm.

Results: The mean latencies of P300 were significantly increased in the diabetic patients (lean and obese groups) and obese patients when compared to the control group ($p<0.001$). The mean amplitudes of P300 were significantly reduced in the diabetic patients (lean and obese groups) when compared to the control group ($p<0.001$).

Conclusions: The present study concluded that there was impairment of cognitive functions in diabetic patients (both lean and obese groups) and obese patients. Moreover, the concomitant presence of obesity and diabetes has been demonstrated to elevate the risk of cognitive impairment.

Keywords

cognitive dysfunction, diabetes, obesity, P300

List of abbreviations

T2DM: type 2 diabetes mellitus;
MMSE: Mini-Mental State Examination;
BMI: body mass index;
ADA: American Diabetes Association;
FBG: fasting blood glucose;
HbA1C: glycosylated hemoglobin;
ERP: event-related potential.

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PENSOFT.

Introduction

The incidence of type 2 diabetes mellitus is increasing worldwide[1] and is associated with mortality and significant morbidity, including neurological disability[2]. In addition, obesity is a significant contemporary health concern that carries wide-ranging implications for society. In particular, the neuropsychological sequelae[3] and data from large human cohorts indicate that obesity is linearly associated with cognitive impairment[4]. Factors such as hypertension, macrovascular diseases, and depression are all more common in diabetes and obesity and may be associated with cognitive decrements.[5,6] The purpose of the present study was to try to remove the effect of these potential 'confounding' factors by examining the cognitive performance of a group of patients with uncomplicated type 2 diabetes mellitus (T2DM) and obesity. Patients with T2DM are more likely to be overweight and obese than those without T2DM.[7] Obesity and type 2 diabetes have been associated with a greater risk of brain damage with poor cognitive health and increased risk of cognitive disorders.[8] Only a few studies have examined the relationship between T2DM and cognitive function in the context of obesity.[9,10] We found few longitudinal studies in which changes in cognitive functioning were evaluated in middle-aged populations[11-14], so the present study was a case-control study and concerned with patients in their middle ages. Because the results of cognitive tests can vary due to differences in the tests themselves, consistency across different assessments is often lacking. Thus, multiple cognitive tests are needed[15], so we used two cognitive methods – Mini-Mental State Examination (MMSE) and P300 – to assess cognitive functions. The present study used the MMSE score to exclude patients with cognitive impairment (score <26) and to move to the next step by assessing the cognitive function of the included patients by P300. MMSE is a 30-question assessment of cognitive function.[16] P300 is a form of event-related potential, and P300 ERP is embedded within the EEG signal recordable from the scalp of the human brain.[17] P300 amplitude mirrors attention and short-term memory, and P300 latency reflects the speed of neuronal functions.[18]

Materials and methods

Patients

In this study, the sample size was 228 subjects, with 57 non-diabetic lean (controls) as a comparison group, 57 diabetic lean patients (BMI 18.5–24.9 kg/m^2), 57 non-diabetic obese patients (BMI>30 kg/m^2), and 57 diabetic obese patients (BMI> 30 kg/m^2).

Inclusion criteria

Age 40-60 years with type 2 diabetic patients (lean and obese) (according to American Diabetes Association (ADA) criteria 2022) and obese patients (BMI>30 kg/m^2).

Exclusion criteria

Patients with type 1 diabetes, pre-diabetes, hypertension, cerebrovascular accidents, cardiovascular accidents, peripheral vascular diseases, malignancy, chronic diseases, and auditory or psychological disturbances were excluded from the study.

Methodology in details

1. This study was approved by the institutional Ethics Committee in December 2020.

2. Informed consent was obtained from all study participants or their legal representative after proper explanation. The participants were informed that they were free to withdraw from the study at any stage.

3. The participants were recruited from the outpatient clinics of endocrinology and internal medicine at Kasr Al-Aini Hospital from March 2021 to October 2022.

4. Mini-Mental State Examination (MMSE): The subjects were examined using the Arabic version of the Folstein Mini-Mental State Examination, a brief 30-point neuropsychometric test that assesses cognitive functions. These functions include orientation, memory, attention, obedience to verbal and written orders, and writing and copying skills. The MMSE is divided into two portions. The first takes just oral replies and covers orientation, memory, and attention; the highest score is 21. The second part tests the ability to name, follow verbal and written commands, write a sentence spontaneously, and copy a complex polygon, which covers the language; the maximum score is 9, so the total score is 30. The questions were asked in a face-to-face interview. Participants would get a '0' for giving the wrong answer or for answering "I don't know." Each correct answer was worth one point. Participants with a score of more than 26 (maximum score = 30), which indicates no cognitive impairment, were selected for the study.

5. A thorough medical history was obtained in order to rule out any history that might have indicated malignancy, chronic diseases, or any complications, such as peripheral vascular disease, cerebrovascular disease, or coronary artery disease. Patients who met these exclusion criteria were also excluded through a thorough physical examination, which would have affected the study's parameters.

6. The subject's weight was recorded to the nearest kilogram while standing on the weighing machine without shoes and wearing minimal clothing. The height of the subject was recorded with the subject in an erect posture, without shoes, with their feet together, and with their back and heels against the upright bar of the height scale. The

subject's head was also upright. The body mass index (BMI) was calculated using the following formula: BMI = weight in kilograms / (height in meters)^2. Patients were classified as obese if their BMI was >30 kg/m^2.

7. Fasting bloog sugar (FBS) and 2-hour postprandial (2hrppr) blood glucose levels were measured before eating and 2 hours after a meal by a blood glucose meter. A small drop of blood, obtained by pricking the skin with a lancet, is placed on a disposable test strip that the meter reads and calculates the blood glucose level in units of mg/dl. Normal FBS: 70–99 mg/dL and normal 2hrppr: <140 mg/dL.

8. Hemoglobin (HGB) and HbA1c levels were measured using the High-Performance Liquid Chromatography (HPLC). Normal HbA1c level: <5.7%.

9. Blood samples were collected and analyzed for urea, creatinine, and estimated glomerular filtration rate (eGFR). Urea and creatinine were measured using automated biochemical analyzers. The eGFR was calculated using the MDRD or CKD-EPI equation based on creatinine levels. All results were within the normal reference range, confirming normal kidney function (normal range of urea 15–45 mg/dl, normal range of creatinine 0.7–1.3 mg/dl, normal range of eGFR ≥90 mL/min/1.73 m^2.

10. Blood samples are collected and analyzed as part of liver function tests (LFTs) to assess the health and functionality of the liver. The LFTs included: ALT and ALP measured using enzymatic colorimetric assays to assess liver cell integrity, total and direct bilirubin measured using Diazo reaction-based assays to assess liver metabolism and excretion, and albumin quantified using bromocresol green (BCG) dye-binding assay to evaluate liver synthetic function. All results were within the normal reference ranges, confirming normal liver function (normal range of ALT 7–56 U/L, AST 10–40 U/L, total bilirubin 0.1–1.2 mg/dL, direct bilirubin 0.0–0.3 mg/dL, and albumin 3.5–5.0 g/dL).

11. P300 event-related potentials were recorded using oddball paradigm.

Recording of ERP P300

The P300 test was done at the Clinical Neurophysiology Unit, Cairo University Hospitals. It was done using the auditory oddball paradigm.

Procedures

In this study, the P300 ERP was recorded for all the subjects after informing them about the nature of the procedure. Ten hours before to the test and throughout the test, the individuals were prohibited from consuming beverages that included caffeine or tannin. The ERP recording was completed between 9:00 and 11:00 in the morning.

Electrode placement

Three active electrodes were placed at Fz, Cz, and Pz according to the 10-20 international system of electroencephalography electrode placement. The reference electrode was placed over either mastoid (M) process. The ground electrode was placed on the forehead. Thus, there were three recording channels: channel 1: Fz-A, channel 2: Cz-A, and channel 3: Pz-A.

Equipment

Nihon Kohden; Neuropak MEB-9200G/K EP/EMG measuring system (Neuropak M1)—4-channel version 08.1.

Conditions

The gain was initially set at 50 μV per (vertical) division. Monitor time was 0.2 seconds. Bandpass was 0.1–50 Hz (value of low and high cuts, respectively). The common tone frequency was 2 kHz with initial intensity of 75 dB, while the rare tone frequency was 8 kHz with initial intensity of 75 dB, too.

Technique

Disc electrodes were placed over the scalp after properly cleaning the skin with an adhesive gel. The active electrode was applied at Cz of the 10-20 international system of electrode placement. Both the reference electrode and the ground were applied to the ear lobule and FPz, respectively. The electrodes' impedance was kept below 5 kilohms. A total of 200 auditory stimuli (bursts) were presented to the ears through earphones. Tones were chiefly background tones, whereas the rarer ones were target tones. The subjects were instructed to press the button as quickly as possible whenever hearing the infrequent tones. These tones were presented randomly intermixed.

Measurements

The P300 wave was defined as the most positive point of the average waveform to the target tones after 250 msec and before 600 msec. To verify producibility of the response, the procedure was repeated at least once. The responses were displayed on a screen and could be printed out. P300 latency and amplitude were measured for each patient in order to discover any major abnormalities.

Statistical analysis

The data were entered in an Excel sheet. Statistical analysis was done using SPSS version 21. Qualitative data was presented by a number and percentage; quantitative data was presented by mean and standard deviation. Statistical analysis was performed for parametric data with the Student t-test and ANOVA with a Bonferroni post-hoc test and for qualitative data using the chi-square test accordingly to detect neurocognitive functions in diabetics and non-diabetics regarding obesity. A p-value of ≤0.05 was considered statistically significant.

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Results

Demographic features of the participants

Fifty-seven nondiabetic lean patients (controls), 57 diabetic lean patients, 57 nondiabetic obese patients, and 57 diabetic obese patients from Cairo University Hospital outpatient clinics were included in our study for assessment of cognitive functions (29.4% males and 70.6% females) **(Fig. 1)** **(Table 1)**.

Table 2 shows that there was no significant difference caused by age distribution across the groups (p=0.114).

Table 1. Descriptive analysis of all groups

	Frequency	Percentages
Controls	57	25.0
Diabetic lean patients	57	25.0
Obese patients	57	25.0
Diabetic obese patients	57	25.0
Total	228	100.0

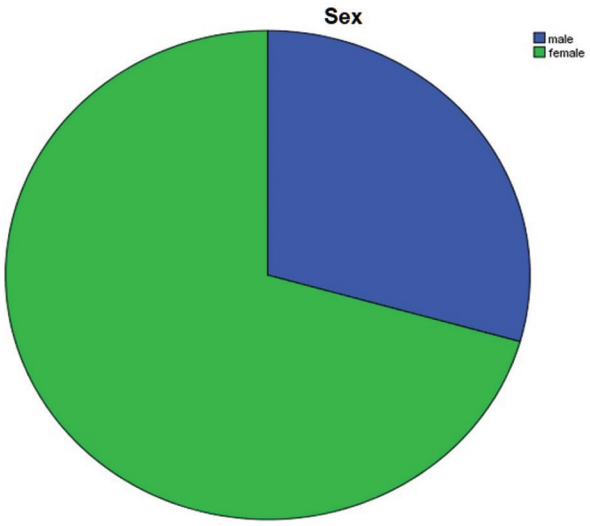

Figure 1. Percentage of males and females.

Table 2. Age distribution across the groups

	Age (year)			
	N	Mean	SD	*P* value
Controls	57	47.1	6.7	0.114
Diabetic lean patients	57	49.8	6.9	
Obese patients	57	49.9	6.9	
Diabetic obese patients	57	48.6	7	

The mean difference is significant when $p \leq 0.05$

Table 3 shows that there was significant difference caused by sex distribution across the groups, females were statistically significantly more than males in all groups (p=0.046).

Comparison of the anthropometric characteristics (height, weight, and BMI) between the groups

Table 4 shows that the mean height of controls was 168.3±6.7, diabetic lean patients 168.6±7, obese patients 167.7±6.8, and diabetic obese patients 168.2±6.8. There was no significant difference in height across the groups (p=0.921).

The mean weight of the control group and diabetic lean patients was 64.7±5.0 kg and 65±4.9 kg, respectively; the mean weight of the obese patients and the diabetic obese patients was 89.3±8.9 kg and 89.7±8.8 kg, respectively. The mean weight was significantly different between the groups (p<0.001).

The mean BMI of controls was 22.2±1.2, of diabetic lean patients – 22.3±1.2, of obese patients – 32±1.4, and of diabetic obese patients – 32.2±1.4. The mean BMI was significantly different between the groups (p<0.001).

Comparison of the biochemical parameters between the groups (FBS, 2hrppr, and HbA1c)

Table 5 revealed that the mean FBS of controls was 84±11 mg/dL, that of diabetic lean patients was 140±36 mg/dL, of obese patients—89±9 mg/dL, and of diabetic obese patients—162±50 mg/dL. The mean FBS was significantly different between the groups (p<0.001).

Table 3. Sex distribution across the groups

Sex	Male n (%)	Female n (%)	Total	*P* value
Controls	14 (24.6%)	43 (75.4%)	57 (100%)	0.046
Diabetic lean patients	24 (42.1%)	35 (57.9%)	57 (100%)	
Obese patients	11 (19.3%)	46 (80.7%)	57 (100%)	
Diabetic and obese patients	18 (31.6%)	39 (68.4%)	57 (100%)	

P value is significant when ≤0.05

Table 4. Comparison of the anthropometric measures between groups

Parameter	Controls	Diabetic lean	Obese	Diabetic obese	*P* value (ANOVA)
	Mean±SD	Mean±SD	Mean±SD	Mean±SD	
Height (cm)	168.3±6.7	168.6±7	167.7±6.8	168.2±6.8	0.921
Weight (kg)	64.7±5	65±4.9	89.3±8.9	89.7±8.8	<0.001
BMI (kg/m^2)	22.2±1.2	22.3±1.2	32±1.4	32.2±1.4	<0.001

Table 5. Comparison of biochemical parameters between groups

Parameter	Controls	Diabetic Lean	Obese	Diabetic obese	*P* value (ANOVA)
	Mean±SD	Mean±SD	Mean±SD	Mean±SD	
FBS (mg/dL)	84±11	140±36	89±9	162±50	<0.001
2hrppr (mg/dL)	128±5	190±69	129±5	225±82	<0.001
HbA1c (%)	4.6±0.5	8.8±1.2	4.8±0.5	9.2±1.5	<0.001

Controls had a mean 2hrppr of 128±5 mg/dL, diabetes lean patients 190±69 mg/dL, obese patients 129±5 mg/dL, and diabetic obese patients 225±82 mg/dL. The mean 2hrppr was significantly different between the groups (p<0.001).

The mean HbA1c of controls was 4.6±0.5%, diabetic lean patients – 8.8±1.2%, obese patients – 4.8±0.5%, and diabetic obese patients – 9.2±1.5%. The mean HbA1c was significantly different between the groups (p<0.001).

Comparison of the cognitive parameters between all groups (MMSE score, amplitude, and latency)

The mean MMSE score for the control group was 28.6±1, for the diabetic lean patients was 27.8±0.9, for the obese patients was 28.1±1, and for the diabetic obese patients was 27.8±0.9. The average MMSE score was significantly different between the groups (p<0.001) **(Table 6)**.

The mean amplitude of controls was 13±4, diabetic lean patients 10.2±5, obese patients 12±5, and diabetic obese patients 10±5. The mean amplitude was significantly different between the groups (p<0.001).

The mean latency of controls was 256±55, diabetic lean patients 309±65, obese patients 321±45, and diabetic obese patients 322±55, with p<0.001. The mean latency was significantly different between the groups (p<0.001).

As illustrated in **Figs 2**, **3**, **4**, **and 5**, there are differences in amplitudes and latencies among the four groups. The amplitude in diabetic patients (both lean and obese) was lower than in the control group, and the latency in diabetic patients (both lean and obese) was higher than in the control group. Additionally, obese patients exhibited higher latency compared to the control group. Furthermore, obese and diabetic obese patients exhibited higher latency compared to diabetic lean patients. The ERP latency was represented on the horizontal axis, while the amplitude was depicted on the vertical axis.

Discussion

It is evident that type 2 diabetes is linked to cognitive dysfunction, as older patients with the disease were found to have impairments in a variety of cognitive domains.[19] Furthermore, data from sizable human cohorts shows a linear relationship between obesity and cognitive deterioration.[4]

The current study compared four groups of individuals: the first group was controls, the second was diabetic lean patients, the third was obese patients, and the fourth was diabetic obese patients. Also, the present study tried to correlate the different parameters with the amplitude and latency of P300. We discussed the results we found in the comparison between the groups generally and in comparison to the results found in previous studies.

Expectedly, there was a significant increase in the mean BMI in the obese groups (obese and diabetic obese) versus the controls and the diabetic lean group (p<0.001). Also, there was a significant increase in the mean FBS, 2hrppr,

Table 6. Comparison of cognitive parameters across all groups

Parameter	Controls	Diabetic Lean	Obese	Diabetic Obese	*P* value (ANOVA)
	Mean±SD	Mean±SD	Mean±SD	Mean±SD	
MMSE score	28.6±1	27.8±0.9	28.1±1	27.8±0.9	<0.001
Amplitude (μV)	13±4	10.2±5	12±5	10 ±5	<0.001
Latency (ms)	256±55	309±65	321±45	322±55	<0.001

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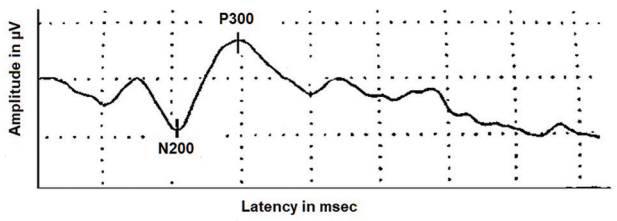

Figure 2. Event related potential P300 obtained from the controls.

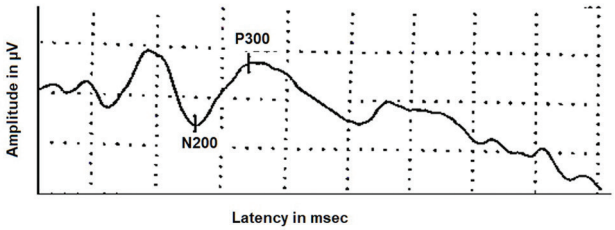

Figure 4. Event related potential P300 obtained from the obese patients.

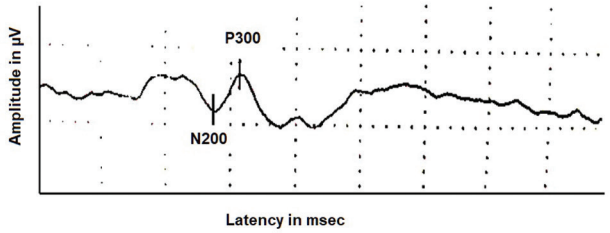

Figure 3. Event related potential P300 obtained from the diabetic lean patients.

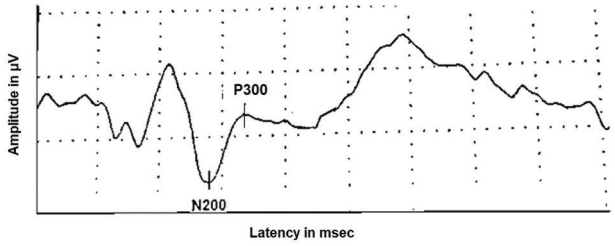

Figure 5. Event related potential P300 obtained from the diabetic obese patients.

and HbA1c in the diabetic groups (lean and obese) versus controls (p<0.001). In this study, HbA1c was utilized not only as a diagnostic indicator for diabetes but also as a critical tool to classify participants into specific metabolic subgroups (non-diabetic, diabetic lean, and diabetic obese). This classification was essential for the comparison of cognitive functions across groups with different metabolic profiles. In order to ascertain glycemic control and its relationship to cerebrovascular diseases, Ajilore et al. analyzed the HbA1c levels of type 2 diabetic patients with healthy control volunteers. They discovered that patients with type 2 diabetes had noticeably higher HbA1c levels.[20] Van der Heide et al. have proposed that HbA1c is a strong indicator of long-term consequences from diabetes and represents long-term glycemic management in diabetic patients.[21]

The present study used the MMSE score, which is a brief cognitive evaluation test. There was a significantly lower MMSE score in T2DM patients (lean and obese groups) in comparison to controls (p<0.001). This result was in agreement with Gaber et al., who examined 20 T2DM patients versus 17 non-diabetic controls using MMSE and documented significant differences in MMSE between the two groups.[22] Additionally, this study showed that the MMSE score was significantly lower in obese patients than in controls (p<0.001). This is consistent with the findings of Katuri et al., who used the MMSE to compare 66 obese patients to 66 non-obese controls and found that the MMSE score was significantly lower in obese patients than in controls.[23] According to these findings, the MMSE, which is a common tool for cognitive testing, shows significant findings among obese patients with or without T2DM as well. The present study also examined the auditory P300 component of ERPs elicited during the oddball paradigm in T2DM patients (lean and obese) and also in obese patients.

Diabetic patients, both lean and obese, had significantly longer P300 latencies and significantly lower P300 amplitudes compared to control subjects (p<0.001), which agrees with a study by Bhanu et al. that found similar results in 30 T2DM patients compared to 30 controls—significantly prolonged latency in the diabetics.[24] Also, Van der Westhuizen and colleagues examined 32 individuals with diabetes and compared them with 32 individuals with diabetes and found a statistically significant difference between the two groups.[25] These results align with the findings of most other studies, which demonstrated that diabetics exhibited significantly prolonged auditory P300 latencies compared to the control group.[26-30] The data are sparse with regard to the P300 amplitude. Measurements of amplitude were not reported by many researchers.[31,32] A number of studies have found no statistically significant differences between diabetic patients and control groups.[27] These results are inconsistent with those of the present study, which may be explained by the relatively small sample size of the participants (16 diabetic subjects versus 17 non-diabetic individuals). However, other studies have found lower amplitudes in diabetic patients than in control groups[22,25,26,33], which are consistent with the results of the present study. Chronic persistent hyperglycemia, which has been demonstrated to inflict harm upon brain neurons and to inhibit the development of neural tissue within the hippocampus, may be the likely causative agent of cognitive dysfunction in patients suffering from diabetes mellitus.[24] Given this established relationship, it is widely accepted that the frontal lobe and the hippocampus/temporal-parietal processes cooperate to produce the P300.[34] It is possible that these neuropatho-

logical and functional changes are reflected in the P300 abnormalities in T2DM we described in this investigation.

Furthermore, the present study showed that the obese patients have significantly prolonged P300 latencies only as compared to the controls ($p<0.001$), similar to the results of the study done by Katuri et al., who documented significantly prolonged latency in the obese group versus controls.[23] However, the current study could not detect significant changes regarding the amplitude. Results from one study did not support our conclusions. This study concluded that there was no statistically significant difference in the P300 latency between obese and non-obese individuals.[35] Nevertheless, this study was conducted on young participants (18–21 years old), and late middle age is when cognitive impairment is most pronounced. Obesity seems to have a compounding effect on cognition over the adult life period.[36] Obesity can be regarded as a risk factor for decreased neuronal integrity and structural and morphological changes in the obese brain, which is most likely the reason for the delayed latency in obese groups.[37]

Obesity may cause insulin resistance, and it is common in adults with T2DM.[38] It was demonstrated that, in comparison to T2DM participants of normal weight, obese individuals with the disease exhibited more severe and progressive impairments in their brain structures and cognition.[39] To clarify the relationship between T2DM and cognitive decline in obese patients, we demonstrated a correlation between T2DM and cognitive impairment (decreased amplitude and longer latency) in light of prior results. Because the mean latencies of obese and diabetic obese patients (321 msec and 322 msec, respectively) were higher than the mean latency of diabetic lean patients (309 msec), we provide additional evidence of the detrimental effects of obesity on latency. Additionally, we have shown that the co-occurrence of obesity and diabetes increases the risk of cognitive impairment, primarily in the areas of information processing and executive functions.

Conclusions

The present study confirmed that P300 could reflect impairment in attention, memory, and the speed of information processing and executive functions. This finding is indicative of early cognitive impairment in diabetic patients and impairment of the speed of information processing and executive functions only in obese people. Furthermore, the coexistence of obesity with diabetes further increases the risk of cognitive impairment, mainly in the speed of information processing and executive functions. Therefore, P300 is a sensitive tool that can be used to uncover early cognitive impairment in type 2 diabetes mellitus and obesity.

Limitations

The limitations of the present investigation are enumerated below. First, the presence of subclinical difficulties, which may be undetected, may have an impact on mental capacity. Secondly, while the assessment did not include an evaluation of instructional level, there is a possibility that a correlation exists between P300 attributes and an individual's instructional level.

Recommendations

Future studies using advanced neuroimaging as a mind structure evaluation tool (practical X-ray) might be beneficial in determining what heftiness means for cerebrum design capacity. Furthermore, an upcoming investigation will determine if increased glycemic control in diabetic patients is a likely beneficial advantage in terms of improving mental capabilities and the long-term effects of maintaining optimum glucose control on mental degeneration in diabetics. Future research is also planned to follow up on mental aspects in individuals following weight loss and their relationship to improved mental outcomes.

Ethics approval and consent to participate

The study was approved by the Kasr Alainy (Cairo University) Ethics Committee in March 2021 (MD-368-2020). Informed consent was obtained from all study participants or their legal representatives after proper explanation, and they were informed that they were free to withdraw from the study at any stage.

Availability of data and materials

Original data and any secondary data are available upon request.

Competing interests

No competing interests.

Funding

No source for funding.

Author contributions

Prof. Hala Ali Gamal Eldin and Dr. Rasha Sobh Mohamed formulated the main idea. Dr. Noha Ali Mahmoud was responsible for participant recruitment and data collection. Dr. Aliaa Ali Tafweek performed the P300 test and interpreted the results. Dr. Rasha Sobh Mohamed analyzed the data statistically. Dr. Noha Ali

Mahmoud wrote the main manuscript, and all the authors reviewed the manuscript.

Acknowledgments

We acknowledge all patients included in the research.

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