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ORIGINAL ARTICLE

Relationship between Body Mass Index and Epilepsy in Adult Patients with Idiopathic Epilepsy at Zagazig University Hospitals

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ABSTRACT

Background: Overweight and obesity are common comorbidities in patients with epilepsy. The relationship between epilepsy and body weight is complex. This connection is often linked to chronic inflammatory processes. This study aims to explore the role of obesity in individuals with epilepsy and determine whether a correlation exists between the two conditions.

Methods: This case-control study was carried out in the Neurology Clinic and Neurology Department, Faculty of Medicine, Zagazig University Hospitals on 224 persons categorized into two groups: patient group(112 epileptic patients who were further subdivided into two group : 52 treatment responsive group and 60 treatment non- responsive group) and 112 control group. Both patients and control group were subjected to comprehensive evaluations, standard laboratory tests, as well as serum interleukin-6 (IL-6) levels with assessment of body mass index (BMI). Electroencephalograms, Brain MRIs were conducted to all patients and controls.

Results: The mean of BMI was significantly higher among the epilepsy patients than control group (P<0.001). patients with resistant epilepsy had higher BMI (P=0.004) in comparison to those with good response to treatment. The mean BMI was higher among generalized tonic-colonic seizures (P=0.02), patients who had a positive history of status epilepticus and those who received polytherapy (P=0.01) and (P=0.001) respectively. In addition, serum IL-6 levels are significantly elevated in overweight and obese epileptic patients (P<0.001).

Conclusions: Our study concluded that high BMI has been involved in causing systemic inflammation as obesity cause an inflammatory response and elevate the level of neuroinflammatory mediators, including interleukin -6(IL-6). Body mass index (BMI) and IL-6 are a significant independent indicator for drug-resistance among epileptic patients.

Keywords: Epilepsy; Obesity; Resistant epilepsy; Neuroinflammation

INTRODUCTION

Despite not being an inflammatory disorder, there is evidence that inflammation is an important contributor in the regulation of synaptic transmission and hyperexcitability, both of which are implicated in the development of epilepsy. [1].

Being overweight or obese is a major nutritional disorder and a public health issue that is caused by several factors, including eating a high-energy, low-nutrient diet and sedentary life. [2].

Body mass index (BMI; weight/height2) is used in epidemiologic research to identify obesity and can be used to determine health risks associated with obesity. Practically, obesity is defined as a BMI of 30 kg/m2, and it is further divided into three classes: class 1 (30–34.9), class 2 (35–39.9), and class 3 (≥ 40) [3].

It is still debatable if obesity and epilepsy are related [4]. Both adolescents and adults with epilepsy are thought to have overweight or obesity as a prevalent comorbidity [5].

Relationship between epilepsy and body weight is complex. The genetic connection between them is one of the potential contributing factors. Obesity among patients with epilepsy is more common.

Furthermore, children whose mothers are obese are more likely to have epilepsy [6].

This study aims to assess the role of obesity in individuals with epilepsy and determine whether a correlation exists between these two conditions.

METHODS

Two groups of 224 people participated in this case control research. Group I (Epilepsy group) included 112 adult patients with idiopathic epilepsy, their ages ranged between 19 and 63 years with a mean age of 35.9 ± 11.4. They were (58%) males and (42%) females. Control group included 112 individuals who were healthy volunteers. Their ages ranged from 19 to 58 years with mean age of 33.6 ± 10.5. They were (54.5%) males and (45.5%) females who visited our clinics.

Additionally, epilepsy group was subdivided into two groups: Drug responsive group included 52 patients. They ranged in age from 23 to 50. with mean ± SD of 33.9 ± 7.83. Males were (61.5%) and females were (38.5%) and Drug-resistant group included 60 patients; with refractory epilepsy according to the ILAE (2010) [7], Their ages were 19–63 years old. with mean ± SD of 37.6 ± 13.6. (55%) were males and (45%) were females.

The Academic and Ethical Committee at Zagazig University gave their approval to the study (IRB#10661). Written informed consent was provided by each and every participant. Every procedure carried out here adheres to the principles stated in the Declaration of Helsinki, which is a component of the Code of Ethics for Research Involving Humans published by the World Medical Association.

Inclusion criteria:

Patients included in the current study were older than 18 years of both genders who were diagnosed with idiopathic epilepsy based on history from patient and witness in accordance with the ILAE criteria [8].

Exclusion criteria:

Patient who were18 years or younger, patients with secondary epilepsy (verified by brain MRI and excluded based on patient and/or family history), individuals who have epileptic syndrome, Those who experience extreme adverse drug reactions, history of drugs or alcohol abuse, or who were taking antiseizure medications why??[who were taking epileptogenic medications], individuals suffering from serious mental or psychiatric illness, as well as patients who have any of the following conditions that may raise IL6 serum levels ,which includes :uncontrolled diabetes mellitus, hypertension, cardiovascular, renal, liver, neoplastic, connective tissue disorders, patients with severe infections or inflammatory diseases, pregnant or lactating women, people with thyroid disease or other secondary causes of obesity, people taking antihypertensive medications, hypolipidemic agents, or steroids, and people adhering to a ketogenic diet.

History and clinical evaluation:

Clinical evaluations were performed on all patients**,** including Comprehensive medical and neurological history and examinations, with a focus on family history, prenatal, natal, and postnatal history, developmental history, age of onset of epilepsy, frequency of monthly attacks, duration of the most recent attack, and duration prior to blood collection. history of status epilepticus, history of febrile seizures, monotherapy or polytherapy drugs, and seizure types classified by ILAE into focal, generalized, and epilepsy syndromes.

Weight, height, and BMI were obtained for all patients and controls. BMI is calculated by dividing weight in kilograms by height in meters squared. Patients were categorized based on their BMI as follows: underweight <18.5 / normal 18.5 – 22.9 / overweight 23 – 24.9 / obese 25 – 29.9 / morbidly obese >30. [9].

Both patients and controls underwent laboratory tests, including complete blood counts, liver and kidney function tests, blood glucose levels, serum electrolyte levels, lipid profiles (total cholesterol, triglycerides, HDL, and LDL), ESR and CRP, and enzyme-linked immunosorbent assays (ELISA) to measure serum levels of IL-6.

Electroencephalography:

Electroencephalography (EEG) was done for all patients in the interictal phase to confirm and classify type of epilepsy and for follow up in cases of drug-resistant epilepsy, using the 10-20 system, with 22 scalp electrodes and 16 channels (alvar-model) EEG equipment, with various provocative methods including photic stimulation, hyperventilation, sleep deprivation, for 60 minutes.

Radiological investigations:

Using a Philips Achevia 1.5T MRI machine, magnetic resonance imaging (MRI) of the brain was performed on all epileptic patients in order to rule out secondary epilepsy.

Statistical analysis

A database application called IBM SPSS (statistical package for social science) 23.0 for Windows SPSS (SPSS Inc., Chicago, IL, USA) was used to collect,

tabulate, and analyze the data. While qualitative data was presented as numbers and percentages, quantitative data was displayed as mean ± standard deviation (SD), median, and inter-quartile range (IQR). The data was analyzed using the Kruskal Wallis test, Fisher exact test, Chi-square test, and Mann-Whitney U test. show noteworthy outcomes, the P value was established at less than 0.05, and for really important outcomes, it was set below 0.001.For correlation between two quantitative variables, we used

Pearson's correlation which is used for parametric normally distributed data and Spearman's rank correlation test which is used for ordinal data or if the assumptions of normality of data not satisfied.

RESULTS

Table (1) Shows that the mean of BMI was significantly higher among the epilepsy patients than control group *(P<0.001)*.

Furthermore, the median value of cholesterol, triglycerides and LDL was higher among the epilepsy group than control group *(P<0.001)*.

As regard acute phase reactants; there was a statistically significant difference between the two groups as ESR and CRP levels are higher among the epilepsy group *(P<0.001)*.

Table (2) shows no significant difference between the drug-responsive group and the drug-resistant group regarding age of onset of illness, duration of disease and type of seizures. Although patients with resistance to treatment have a high statistically significant frequency of epileptic fits *(P<0.001)* and higher BMI *(P=0.004)* in comparison to those with good response to treatment; as (30%) of patients with poor response to treatment where morbidly obese in comparison to (9.6%) of patients with good response to treatment *(P=0.01)*.

Also, there was a statistically significant difference between the drug-responsive group and the drug-resistant group regarding the type of treatment, as all the drug-resistant group (100%) received polytherapy, while only (25%) of the drug-responsive group received polytherapy *(P<0.001)*.

Also, there was a statistically significant difference between the drug-responsive group and the drug-resistant group regarding EEG findings as (66.7%) of the drug-resistant group had a generalized epileptiform activity in comparison to (44.2%) of the drug-responsive group *(P=0.04)*.

Table (3) shows a statistically significant difference between drug responsive and drug resistant patients as regard lipid profile; as median of cholesterol, triglycerides and LDL were higher among drug resistant patients *(P<0.05)*.

Also, there was a statistically significant difference between drug responsive and drug resistant patients as regard acute phase reactants; as median of ESR and CRP were higher among drug resistant patients *(P<0.05)*.

Table (4) shows a statistically significant difference between BMI and type of seizures; as the mean BMI was higher among generalized tonic-colonic seizures *(P=0.02)*. Also, there was a statistically significant difference between BMI and history of status epilepticus and type of treatment; as the mean BMI was higher among patients who had a positive history of status epilepticus and patients who received polytherapy *(P=0.01)* and *(P=0.001)* respectively.

Also, there was a statistically significant difference between BMI and EEG findings; as the mean of BMI was higher among patients who had generalized epileptiform activity (GEA).

Table (5) shows a significant positive correlation between BMI and history of status epilepticus *(r=0.332, P<0.001)*, cholesterol *(r=0.735, P<0.001)*, triglycerides *(r=0.437, P<0.001)*, LDL *(r=0.698, P<0.001)*, CRP *(r=0.438, P<0.001)* and ESR *(r=0.502, P<0.001)*. Also, there was a negative correlation with drug response *(r=-0.282, P=0.003)*, while there was no significant correlation with age, age of onset, frequency of seizures, type of treatment and HDL.

Furthermore, there was a significant positive correlation between IL-6 and frequency *(r=0.574, P<0.001)*, cholesterol *(r=0.454, P<0.001)*, triglycerides *(r=0.405, P<0.001)*, LDL *(r=0.493, P<0.001)*, CRP *(r=0.586, P<0.001)* and ESR*(r=0.514, P<0.001)*. Also, there was a negative correlation with drug response *(r=-0.300, P=0.001)*, while there was no significant correlation with age, age of onset, history of status epilepticus,type of treatment and HDL.

Table (6) shows a statistically significant positive correlation between IL-6 and BMI *(r=0.489, P<0.001)*.

Table (1): Body Mass Index (BMI) and laboratory data among the studied groups

		Epilepsy group (n=112)	Control group (n=112)	P Value
BMI (kg/m^2)	Mean ± SD	26.7 ± 3.8	22.68±1.63	**<0.001**[1]
	Range	(19 – 35)	(18.92–24.48)	
BMI classification (N. %)	Underweight	0 (0%)	0 (0%)	**<0.001**[2]
	Normal	21 (18.8%)	46 (41.1%)	
	Overweight	22 (19.6%)	66 (58.9%)	
	Obese	46 (41.1%)	0 (0%)	
	Morbid obesity	23 (20.5%)	0 (0%)	
Cholesterol (mg/dl)	Median (IQR)	195.5 (60)	149 (20)	**<0.001**[3]
	Range	(82 – 281)	(110 – 191)	
Triglycerides (mg/dl)	Median (IQR)	96 (88)	69 (20)	**<0.001**[1]
	Range	(53 – 200)	(45 – 140)	
HDL (mg/dl)	Median (IQR)	37 (10.3)	38 (6)	0.38[1]
	Range	(12 – 52)	(35 – 50)	
LDL (mg/dl)	Median (IQR)	111 (54.5)	42.5 (12.25)	**<0.001**[1]
	Range	(42 – 199)	(30 – 57)	
ESR (mm/h)	Median (IQR)	26 (31)	11 (6)	**<0.001**[1]
	Range	(5 – 102)	(1.3 – 15)	
CRP (mg/dl)	Median (IQR)	10.09 (14.74)	0.7 (0.4)	**<0.001**[1]
	Range	(0.64–66.4)	(0.3 – 1)	

*[1]Mann-Whitney U test,[2]Fisher exact test, [3]Student's T test
Non-significant: P >0.05, Significant: P ≤0.05

Table (2): characteristics of seizures, BMI and EEG among drug-responsive & drug-resistant patients

Variables		Drug responsive (n=52)	Drug-resistant (n=60)	P Value
Duration of epilepsy (years)	Median (IQR)	20 (16)	13 (11)	0.17[1]
	Range	(1 – 39)	(4 – 34)	
Type of seizures (N.%)	GTCS	44 (84.6%)	46(76.7%)	0.12[3]
	Focal seizures	6 (11.5%)	5 (8.3%)	
	Focal to bilateral tonic clonic	2 (3.8%)	9 (15%)	
History of status epilepticus (N.%)	Absent	46 (88.5%)	39 (65%)	**0.003**[2]
	Present	6 (11.5%)	21 (35%)	
Frequency of seizures (/m)	Median (IQR)	1 (2.83)	4 (3.75)	**<0.001**[1]
	Range	(1 – 12)	(1 – 90)	
BMI (kg/m^2)	Median (IQR) Range	25.17±3.21 (20 – 31)	27.45±4.05 (19 – 35)	**0.004**[1]
BMI classification (N. %)	Normal	15 (28.8%)	6 (10%)	**0.01**[2]
	Overweight	10 (19.2%)	12 (20%)	
	Obese	22 (42.3%)	24 (40%)	
	Morbid obesity	5 (9.6%)	18 (30%)	
Type of treatment (N. %)	No therapy	0 (0%)	0 (0%)	**<0.001**[3]
	Monotherapy	39 (75%)	0 (0%)	
	Polytherapy	13 (25%)	60 (100%)	
EEG findings (N. %)	Normal	23 (44.2%)	18 (30%)	**0.04**[3]
	Focal	6 (11.5%)	2 (3.3%)	
	GEA	23 (44.2%)	40 (66.7%)	

*[1]Mann-Whitney U test, [2]Chi-square test, [3]Fisher exact test.
Non-significant: P >0.05, Significant: P ≤0.05

Table (3): Lipid profile and acute phase reactants among drug-responsive & drug-resistant patients

Variables		**Drug responsive (n=52)**	**Drug-resistant (n=60)**	**P Value**
Cholesterol (mg/dl)	Median (IQR)	176 (54)	208 (54)	**0.006[1]**
	Range	(82 – 242)	(112 – 281)	
Triglycerides (mg/dl)	Median (IQR)	95 (88)	111 (94)	**0.02[1]**
	Range	(53 – 185)	(66 – 200)	
HDL (mg/dl)	Median (IQR)	37 (11)	37 (11)	0.21[1]
	Range	(12 – 50)	(12 – 52)	
LDL (mg/dl)	Median (IQR)	104.5 (55)	117 (63)	**<0.001[1]**
	Range	(52 – 155)	(42 – 199)	
ESR (mm/h)	Median (IQR)	25 (11)	48 (25.8)	**<0.001[1]**
	Range	(5 – 63)	(14.4 – 102)	
CRP (mg/dl)	Median (IQR)	6 (10.7)	15.3 (20.1)	**0.008[1]**
	Range	(0.64 – 64.3)	(1.26 – 66.4)	

*[1]Mann-Whitney U test.
Non-significant: P >0.05, Significant: P ≤0.05

Table (4): Association of BMI with demographic and clinical data among Epileptic group

Variables		**BMI** Mean ± SD	**P Value**
Sex	Male	26.7 ± 2.78	0.14[1]
	Female	26 ± 4.96	
Family history of epilepsy	Absent	25.9 ± 3.62	0.1[1]
	Present	25.1 ± 4.11	
Type of seizures	GTCS	27.1 ± 2.55	**0.02[2]**
	Focal seizures	23.4 ± 2.69	
	Focal to bilateral tonic clonic	26.7 ± 3.95	
History of status epilepticus	Absent	25.8 ± 3.54	**0.01[1]**
	Present	27.8 ± 4.15	
Type of treatment	Monotherapy	24.5 ± 2.26	**0.001[2]**
	Polytherapy	27.2 ± 4.1	
EEG findings	Normal	24.9 ± 3.14	**0.04[2]**
	Focal	26 ± 4.75	
	GEA	26.8 ± 3.85	

*[1]Mann-Whitney U test, [2]Kruscal-Wallis test, Non-significant: P >0.05
Significant: P ≤0.05

Table (5): Correlation of BMI and IL-6 with the clinical and laboratory data among studied patients

Variable	BMI		IL-6	
	R	***P***	***r***	***P***
Age	0.211	0.25[1]	0.037	0.71[2]
Age of onset of seizures	0.105	0.19[1]	0.05	0.95[2]
Frequency of seizures	0.134	0.158[2]	0.574	**<0.001[2]**
History of status	0.332	**<0.001[2]**	-0.018	0.85[2]
Type of treatment	0.181	0.06[2]	-0.009	0.92[2]
Drug response	-0.282	**0.003[2]**	-0.300	**0.001[2]**
Cholesterol	0.735	**<0.001[1]**	0.454	**<0.001[1]**
Triglycerides	0.437	**<0.001[2]**	0.405	**<0.001[2]**
HDL	0.070	0.15[2]	0.079	0.21[2]
LDL	0.698	**<0.001[2]**	0.493	**<0.001[2]**
CRP	0.438	**<0.001[2]**	0.586	**<0.001[2]**
ESR	0.502	**<0.001[2]**	0.514	**<0.001[2]**

*[1]Pearson correlation, [2]Spearman rank correlation test
Non-significant: P >0.05, Significant: P ≤0.05

Table (6): Correlation of Interlukin-6 with BMI among studied patients

Variable	IL-6	
	R	***P***
BMI	0.489	**<0.001**

*Spearman rank correlation test.
Non-significant: P >0.05, Significant: P ≤0.05

DISCUSSION

Frequent spontaneous epileptic seizures are a common symptom of epilepsy, which is a common brain disorder [10]. One of the hallmarks of metabolic syndrome is obesity. Inflammation plays an important role in the connection between epilepsy and obesity [11]. The relationship between overweight or obesity and epilepsy remains controversial. Obesity and epilepsy are causally connected, according to Mendelian randomization research by Zhou et al. [12].

According to our results, our patients had body mass index (BMI) ranged from 19-35 ,41.1% of them were obese ,20.5% were with morbid obesity, 19.6% were overweight and only 18.8% of them were with normal BMI. We observed that patients with epilepsy had a significantly high BMI relative to the control group (P<0.001).

This agrees with many other studies done by, Arya et al. [6], and Khuda et al. [13] who found that obesity was more common in epileptic patients compared with healthy controls.

Additionally, studies have shown that ghrelin, leptin, and adiponectin levels might be disturbed by epileptic seizures. Changes in these hormones can ultimately result in weight gain and obesity since they are linked to hunger, satiety, and metabolism [14].

On the other hand, Janousek et al. [5] did not discover that adult epileptic patients were more obese than the general population. Notably, historical controls were used in that study in place of direct comparison with the control population. Additionally, Moses et al.[15] discovered that the BMI of those with epilepsy was lower than that of those without it.

Very few studies have evaluated the relationship between obesity and epileptic patients' risk of developing drug-resistant epilepsy (DRE). One hundred and sixty of our patients [sixty of our patients]were drug resistant and all of them (100%)were on polytherapy . Compared to drug-responsive patients, their BMI was substantially higher since only 10% of drug-resistant patients were normal, 20% were overweight, 40% were obese,30% were morbidly obese.

Consistent with our findings, two studies showed that patients receiving polytherapy for epilepsy had

greater rates of overweight and obesity than patients receiving a single medication. Patients having a history of status epilepticus also had higher BMIs. Additionally, they found that adult epileptic patients who were obese had a higher prevalence of drug-resistant epilepsy (DRE) than those who were not obese. Sex, age of onset, duration of epilepsy, developmental delay, history of status epilepticus, and other comorbidities did not affect the relationships. [5, 16]

A link between being overweight and hippocampus volume atrophy was found in a large prospective study conducted over an eight-year period. Hippocampal atrophy, on the other hand, contributes to the pathophysiology of drug-resistant epilepsy [15].

It has been suggested that stabilizing BMI or weight loss is linked to improved seizure control, and this relationship is believed to be the basis of a ketogenic diet for seizure management [17].

According to our research, individuals with epilepsy had significantly higher CRP and ESR than controls, and patients who were drug resistant had much higher CRP and ESR. In a comparable way, Alapirtti et al. [18] reported that whereas partial seizures did not raise CRP, secondary generalized tonic-clonic seizures did.

Our findings are consistent with those of Sohn et al. [19], who discovered that patients with refractory epilepsy had both acute and chronic CRP elevations. Additionally, the findings of Soliman et al. [20], and Tao et al. [21] confirm the link between epilepsy and inflammatory markers.

Our investigation revealed a substantial relationship between BMI, ESR, and CRP, with an increase in BMI being correlated with an increase in ESR and CRP. Cohen et al. [22] supported our findings as they found that ESR and CRP levels tend to rise with BMI.

Our findings showed that the lipid profile indices [total cholesterol, triglycerides (TG), and low-density lipoprotein (LDL)] were also greater in drug-resistant patients than in drug-responsive patients, and that they were significantly higher in epileptic patients than in controls. The study conducted by Soliman et al. [20] similarly revealed a significant rise in TG and LDL in their epileptic patients compared to the control group.[These investigations were done for patients only]

Similarly, Mintzer et al. [23] discovered in a community-based study that male adults with epilepsy had a greater frequency of hyperlipidemia than the general population.

Additionally, Khuda et al. [13] found that younger epileptic patients had a significantly greater percentage of abnormally raised LDL than did controls in the same age group who were not epileptic.

Furthermore, in our research, a significant positive correlation was seen between BMI and LDL ($r=0.698$, $P<0.001$), triglycerides ($r=0.437$, $P<0.001$), and cholesterol ($r=0.735$, $P<0.001$), but not with high density lipoprotein (HDL).

One study found that obese epileptic patients had higher TG, very low density lipoprotein (VLDL), and non-HDL-cholesterol levels, among other lipid abnormalities [24]. Another study found that Apo A-I and HDL-C levels are generally low [25]. It is believed that these small, dense LDL particles are more pro-atherogenic than big LDL particles [26].

According to many reports, the majority of antiepileptic medications (AEDs), particularly those from the older generation like phenytoin, carbamazepine, and phenobarbital, are enzyme inducers and as such have negative effects on the body's multiple metabolic and biochemical functions. When these medications are taken for an extended period of time, they can stimulate the hepatic synthesis of cholesterol, which can lead to an increase in total cholesterol, LDL cholesterol, and even HDL cholesterol [27].

Although the exact cause of the strong correlation between obesity and an increased risk of many diseases is unknown, it has been hypothesized that obesity contributes to systemic inflammation by directly accessing and secreting inflammatory cytokines and free fatty acids into the portal circulation [28].

Obesity raises the levels of neuroinflammatory mediators such as TNF, IL-6, and IL-8 and triggers an inflammatory response. As weight increases, adipocytes undergo changes that ultimately result in the release of pro-inflammatory adipokines. Moreover, seizures cause inflammation. Consequently, obesity and epilepsy together produce a persistent inflammatory state that may lead to an aberrant microenvironment and a decreased responsiveness to medication [29].

We found a strong positive association between IL-6 and BMI after examining the relationship between the two variables. Our findings are consistent with those of Baikpour et al. [2], who found a substantial positive connection between BMI and blood levels of IL6, TNF-α, IL-8, IL-12, and IL-18.

Elia and Mustafa [30] discovered that a higher BMI was associated with a significantly higher mean of IL-6. A similar outcome was obtained in a study conducted by van Vliet et al. [29]. Furthermore, circulating levels of IL6 and BMI were reported to be statistically significantly positively correlated by El-Mikkawy et al. [31].

CONCLUSIONS:

Our study concluded that high BMI has been involved in causing systemic inflammation as obesity cause an inflammatory response and elevate the level of neuroinflammatory mediators, including interleukin -6(IL-6). Body mass index (BMI) and IL-6 are a significant independent indicator for drug-resistance among epileptic patients.

More research on various forms of epilepsy and age groups is required, with a bigger sample size to evaluate the association between seizure severity and BMI as a measure of obesity.

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