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Research Article

The Impact of Obesity on the Distribution Pattern of Serum Copeptin among Iraqi Patients with Multiple Sclerosis

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Abstract

Background: Multiple sclerosis is an autoimmune illness that can be triggered by both hereditary and environmental factors. There are crucial roles for the neuroendocrine system in initiating autoreactive immunity at this stage. Copeptin is a promising inflammatory marker that has the potential to be employed as a prognostic factor in several disorders, particularly those affecting the central nervous system.

Objectives: In this research, we attempted to evaluate the relation of serum copeptin concentrations with weight increment in Iraqi patients with multiple sclerosis (MS).

Methods: A cohort of sixty Iraqi patients with multiple sclerosis took part in this study and were classified equally into three groups: obese group, overweight group, and normal-weight group. The serum lipid profile was estimated using a Biosystem kit, while serum copeptin was estimated using ELISA kit.

Results: Serum copeptin levels were higher in obese as well as overweight MS patients compared to MS patients with normal weight (170.7±29, 160±27.8 *vs*. 149.3±25.9) pg/ml, respectively. Also, it showed a significant positive correlation with body mass index, serum triglycerides and serum very low-density lipoprotein in overweight MS patients**.**

Conclusion: Iraqi multiple sclerosis patients who have excess body weight have higher levels of serum copeptin when compared to those MS patients with normal weight. The positive significant correlation of serum copeptin with body mass index, serum triglycerides, and serum low-density lipoprotein among overweight MS patients may indicate a vital role of copeptin as a neuroendocrine in the adipose dysfunction and related diseases.

Keywords: Body mass index; Copeptin; Expanded Disability Status Scale; Multiple sclerosis; Obesity**.**

Introduction

Multiple sclerosis is defined as an autoimmune-mediated disorder that affects the CNS in which the myelin sheath is ruptured due to genetic and environmental factors. At this stage, the neuroendocrine system plays an essential role in triggering autoreactive immunity. The neuroendocrine system, which is involved in immunomodulation as well, uses the hypothalamic-pituitary-adrenal (HPA) axis and other connecting pathways to exert these effects by hormones (1). A vital neuroendocrine hormone that regulates blood pressure, sodium homeostasis, kidney function, and osmotic balance is arginine vasopressin (AVP), which is additionally known as antidiuretic hormone (ADH). Despite its usefulness as a diagnostic tool for a variety of fluid illnesses, measuring AVP has never been done in a clinical setting because of the technological challenges and the pre-analytical steps involved (2). Originating at the AVP preprotein's C-terminus, copeptin is a 39-amino-acid glycopeptide. Within the neurohypophysis, copeptin is released in an equimolar quantity in reaction to situations that stimulate the release of AVP (3). When the HPA axis is activated, the posterior pituitary gland secretes copeptin into the

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bloodstream in an equal proportion to AVP hence, copeptin levels are a good predictor of AVP levels. Thus, copeptin levels can be utilized to indicate the activation of the HPA axis (4). It has been reported that endocrine or immunological markers are associated with multiple sclerosis (MS), epilepsy, and dementia, three prevalent neurological illnesses. One of the primary physiological regulators of brain development is hormones. Dysfunction in the endocrine and immunological systems may perform an important function in the onset of diseases such as MS, epilepsy, and dementia (5).

Copeptin is a promising inflammatory marker that has the potential to be employed as a prognostic factor in several disorders, particularly those affecting the central nervous system (6-8). Numerous researches suggest that hyperactivity of the HPA axis may accelerate MS progression. Additionally, postmortem examinations of MS patients revealed larger adrenal glands, which is in line with higher glucocorticoid production (9,10). Thus the study attempted to evaluate the association of serum copeptin concentration with weight increment in Iraqi patients with MS.

Patients and Methods

Study population

This case-control study was conducted in cooperation with Baghdad Teaching Hospital at the Medical City Complex in Baghdad, Iraq. Sixty MS patients were involved in the study who were diagnosed based on McDonald criteria; a group of guidelines which combine Magnetic Resonance Imaging (MRI) scans along with clinical and laboratory tests that can verify a diagnosis of diffuse demyelinating lesions with dissemination of lesions in space and dissemination of lesions in time are displayed on their brain MRIs (11). According to their body mass index (BMI); these patients were subdivided into three groups:

- G1group included 20 MS patients with BMI >30 (Kg/m^2).
- G2 group included 20 MS patients with BMI 25-30 (Kg/m^2).
- and G3 group included 20 MS with BMI < 25 (Kg/m^2).

Multiple sclerosis patients were 27 men and 33 women, ranging in age from 20-60 years, and with a disease duration between 2-10 years. Patients' ages, gender, anthropometric indices, MS types, and disease durations were among the demographic and clinical data documents. Each patient was assessed thoroughly on the first appointment, and an Expanded disability Status Scale (EDSS) (a method for evaluating an individual's current level of disability) was administered to evaluate the severity of their disabilities (12). A human research ethics committee at the College of Science/ University of Baghdad gave approval to this study (Number CSEC/0224/0018) on February 6, 2024, and all patients were asked to sign an informed consent form that followed the guidelines established by the Declaration of Helsinki.

Sample Handling, Anthropometric and Laboratory Test: Each fasting MS patient with different BMI had five milliliters of venous blood drawn, and they were placed in tubes equipped with a gel separator. The samples were centrifuged at 1500 × g for fifteen minutes after an incubation time of thirty minutes. Lipid profile: serum total cholesterol (TC), serum triglycerides (TG), high-density lipoprotein (HDL), and serum copeptin were estimated using a subset of the collected serum that was kept at -20 °C, using the Biosystem kit and My BioSource ELISA kit, respectively. Each individual's height (in cm) and weight (in kg) were recorded. Weight (in kilograms) divided by height squared (in meters squared) yielded the body mass index (BMI) (Kg/m^2).

Exclusion Criteria: In order to avoid the interferences in copeptin results of MS patients, patients with Rheumatoid arthritis, Lupus, or any other autoimmune disease, other neurological disorders, such as epilepsy or Parkinson's disease, were excluded. Furthermore, obese individuals with diabetes mellitus, hypertension, or cardiovascular complications were not included either.

Statistical Analysis

A statistical analysis was conducted using the IBM SPSS software package (version 27.0). Means ± standard deviations were used to report the variables. Statistical tests such as one-way ANOVA and post hoc Tukey were used to compare the groups. A statistically significant difference was indicated by a *P* value of less than 0.05, which was used to detect the relationships between serum copeptin and other factors by Pearson's correlation analysis.

Results

The demographic and clinical data for all groups are illustrated in Tables 1 and 2, respectively. The G1 group showed a significant increase (P <0.001) in BMI compared to the G2 and G3 groups, while a significant decline in EDSS was noticed in both G1 & G2 in comparison to G3 (P = 0.025). A highly significant difference in TC level was shown in G1 and G2 in comparison with the G3 group (P <0.001), also a highly significant increase in low-density lipoprotein (LDL) was shown among obese MS patients (G1 group) compared to G2 &G3 groups. However, no significant differences were noticed in TG, HDL and very low-density lipoprotein (VLDL) levels among the study groups. In addition, serum copeptin levels were significantly higher in obese as well as overweight MS patients compared to MS patients with normal weight (170.7±29, 160±27.8 *vs*. 149.3±25.9) (Kg/m^2).

Table 1: Demographic data of study participants.

Parameter	G1	G2	G3	*P*- value
N	20	20	20	----------
Age (year)	20-60	25-55	24-58	----------
Gender (m/f)	9/11	9/11	9/11	----------
Disease duration (year)	2-10	2-8	2-10	----------

G1: Obese MS group, G2: Overweight MS group, G3: Normal weight MS group.

Table 2: Clinical data of study participants.

PARAMETER	**G1**	**G2**	**G3**	***P*- VALUE**
BMI (Kg/m^2)	33.7 ±2	27.16 ±1.6	21.4 ±2.42	<0.001**
EDSS	3.22 ±0.9	3.25 ±1.4	5 ±1.2	0.025*
TC (mg/dl)	202 ±55	180±50	142±17	<0.001**
TG (mg/dl)	177±40	173 ±60	140 ±47	0.235
HDL-CHOLESTEROL (mg/dl)	36±13	36±15	34.8 ±13	0.929
LDL -CHOLESTEROL (mg/dl)	132 ±49	108±46	79 ±20	0.002**
VLDL -CHOLESTEROL (mg/dl)	33 ±11	34 ±14	27 ±10	0.235
SERUM COPEPTIN (Pg/ml)	170.7 ±29	161 ±27.8	149.3 ±25.9	0.04*

Results were expressed as mean ± SD, ANOVA test was used for the purpose of comparison between the three groups. * Statically significant, ** Statically highly significant. G1: Obese MS group, G2: Overweight MS group, G3: Normal weight MS group, BMI: Body mass index, EDSS: Expanded Disability Status Scale, TC: Total cholesterol, HDL: High density lipoprotein,TG: Triglycerides, LDL: Low-density lipoprotein, VLDL: very low-density lipoprotein.

Serum copeptin showed a significant positive correlation (P < 0.05) with BMI, TG and VLDL in G2, as shown in Table 3, while no significant correlation between serum copeptin and any other parameter in neither G1 nor G3 groups.

Table 3: correlations between copeptin serum levels and demographic data of patients. Serum copeptin (pg/ml)

Parameter	G1		G2		G3	
	R	*P*	*R*	*P*	*R*	*P*
Age (year)	-0.094	0.695	0.120	0.615	0.086	0.720
BMI (Kg/m^2)	0.060	0.800	0.514	0.02*	-0.024	0.920
EDSS	0.158	0.505	0.312	0.168	0.187	0.429
TC (mg/dl)	0.049	0.837	0.123	0.606	-0.104	0.662
TG (mg/dl)	-0.032	0.895	0.451	0.046*	-.0111	0.642
HDL-CHOLESTEROL (mg/dl)	0.132	0.579	-0.026	0.912	0.393	0.086
LDL -CHOLESTEROL (mg/dl)	0.021	0.930	-0.047	0.844	-0.295	0.207
VLDL -CHOLESTEROL (mg/dl)	-0.032	0.895	0.451	0.046*	-0.111	0.642

r, Pearson coefficient. *Statistically significant at P < 0.05. **highly significant at P < 0.01. G1: Obese MS group, G2: Overweight MS group, G3: Normal weight MS group, BMI: Body mass index, EDSS: Expand Disability Status Scale, TC: Total cholesterol, HDL: High density lipoprotein,TG: Triglycerides, LDL: Low-density lipoprotein, VLDL: very low-density lipoprotein.

Discussion

An altered production of adipokines and systemic polarization of innate and adaptive immune cells are hallmarks of the chronic low-grade inflammatory state induced by obesity (14, 15).

Several immune-mediated diseases, such as rheumatoid arthritis, inflammatory bowel diseases, type 1 diabetes, psoriasis, and multiple sclerosis, have been found to be significantly more common in obese people or to have a worse prognosis overall (13). Secretion of copeptin, a peptide that has been extensively investigated, is directly correlated with the mass of adipose tissue and is elevated in obesity (16- 18). In this study, Serum copeptin concentrations were elevated in MS patients who were overweight and obese compared to those whose weight was normal, according to research by Baranowska-Bik *et al.*MS patients have elevated copeptin levels; they also investigated how BMI affected copeptin levels and found that overweight MS patients had higher amounts (19). Koseoglu *et al*. found that relapse remission multiple sclerosis (RRMS) patients had considerably greater plasma copeptin levels than the control group. They found evidence that HPA activation contributes to MS. Prior to steroid therapy, patients experiencing an acute attack had lower copeptin levels compared to those in remission and those without a recent history of clinical attacks (20). In contrast, İlgezdi, *et al*. showed that patients with clinically silent multiple sclerosis (CSMS) had reduced levels of copeptin, and in a disease state where the HPA axis is not actively working, low copeptin levels may be an indication of this (21). Numerous studies showed that copeptin levels increased in obese patients with other diseases; Al-Fatlawi *et al*. found that plasma copeptin levels were significantly higher in obese patients with fatty liver disease, and this marker is also helpful in identifying metabolic disease risk factors, including diabetes mellitus (22). Atere *et al*. found that when comparing diabetes and non-diabetic populations, those with diabetes had significantly greater levels of copeptin and atherogenic indices. Obese diabetic participants had significantly greater copeptin levels compared to non-obese diabetic subjects, confirming the link between copeptin and dyslipidemia, an indicator for increased risk of atherogenicity (23).

According to our results, serum copeptin showed significant positive correlation with BMI, serum triglycerides, and VLDL among MS patients who are overweight, while no significant correlation was noticed among normal-weight and obese MS patients. This finding was in line with the results of Rothermel *et al*. study; who found that both body mass index (BMI) and triglycerides were strongly correlated with copeptin (24). In addition, El-Masry *et al* found an increment in serum copeptin among obese individuals compared to those having a normal weight. Also, they found that serum copeptin had a significant positive correlation with BMI, and no significant association with TG was found (25). Given the association between elevated blood copeptin levels in MS patients who are overweight or obese and other metabolic risk factors, body mass index (BMI), triglycerides, and VLDL it is reasonable to assume that copeptin may have a pathogenic role in adipose dysfunction and related diseases, such as dyslipidemia.

Limitations

The challenges in collecting MS samples and the high cost of the investigations resulted in a relatively small sample size in this study, which could restrict the findings' generalizability.

Conclusion

Iraqi multiple sclerosis patients who have excess body weight have higher levels of serum copeptin when compared to those MS patients with normal weight. The positive significant correlation of serum copeptin with BMI, serum TG, and serum LDL among overweight MS patients might indicate a vital role of copeptin as a neuroendocrine in adipose dysfunction and related diseases.

Authors' declaration

We confirm that all the Figures and Tables in the manuscript belong to the current study. Authors sign on ethical consideration's Approval-Ethical Clearance: The project was approved by the local ethical committee in the College of Science Institutional Ethics Committee at the University of Baghdad, according to the code number (CSEC/0224/0018) on (6/ 2/ 2024).

Conflict of Interest: None

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Data availability: Upon reasonable request, the corresponding author will make the data sets generated and/or analyzed during the current work available.

Authors' contributions

Study conception & design: (Suzan A. Hamza, Namir I. A. Haddad and Gheyath AL Gawwam). Literature search: (Suzan A. Hamza). Data acquisition: (Suzan A. Hamza). Data analysis & interpretation: (Suzan A. Hamza, Namir I. A. Haddad and Gheyath AL Gawwam). Manuscript preparation: (Suzan A. Hamza). Manuscript editing & review: (Namir I. A. Haddad and Gheyath AL Gawwam).

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تأثير السمنة على نمط توزيع الكوبيبتين في المصل لدى المرضى العراقيين المصابين بالتصلب المتعدد

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الخلاصة
الخلفية: التصلب المتعدد هو مرض مناعي ذاتي يمكن أن يحدث نتيجة عوامل وراثية وبيئية. هناك أدوار حاسمة للجهاز العصبي الصماوي في بدء المناعة الذاتية في هذه المرحلة. كوبيتين هو علامة التهابية واعدة لديها القدرة على استخدامها كعامل تنبؤ في العديد من الاضطرابات، وخاصة تلك التي تؤثر على الجهاز العصبي المركزي.
الهدف: نحاول في هذا البحث تقييم العلاقة بين تركيزات كوبيتين في المصل وزيادة الوزن لدى المرضى العراقيين المصابين بالتصلب المتعدد.
الطريقة: شاركت مجموعة من ستين مريضا عراقيا مصابا بالتصلب المتعدد في هذه الدراسة وتم تصنيفهم بالتساوي إلى ثلاث مجموعات؛ السمنة وزيادة الوزن والوزن الطبيعي على التوالي. تم تقدير ملف الدهون في المصل باستخدام مجموعةBiosystem ، تم تقدير كوبيتين في المصل باستخدام مجموعة .ELISA
النتائج: كانت مستويات الكوبيتين في مصل الدم أعلى لدى مرضى التصلب المتعدد الذين يعانون من السمنة وزيادة الوزن، مقارنة بمرضى التصلب المتعدد ذوي الوزن الطبيعي (170.7±29، 160±27.8 مقابل 149.3±25.9)pg/ml. كما أظهرت ارتباطا إيجابيا ذا دلالة إحصائية مع مؤشر كتلة الجسم، ودهون الثلاثي، ودهون البروتين الدهني منخفض الكثافة جدا لدى مرضى التصلب المتعدد الذين يعانون من زيادة الوزن.
الاستنتاج: يظهر مرضى التصلب المتعدد العراقيون الذين يعانون من زيادة الوزن مستويات أعلى من الكوبيتين في مصل الدم مقارنة بمرضى التصلب المتعدد ذوي الوزن الطبيعي. قد يشير الارتباط الإيجابي الدال بين الكوبيتين في مصل الدم ومؤشر كتلة الجسم، ودهون الثلاثي، ودهون البروتين الدهني منخفض الكثافة في مصل الدم لدى مرضى التصلب المتعدد الذين يعانون من زيادة الوزن إلى الدور الممرض للكوبيتين كعامل عصبي للغدة الصماء في اختلال وظائف الأنسجة الدهنية والأمراض المرتبطة بها.
الكلمات المفتاحية: التصلب المتعدد، السمنة، كوبيتين، مؤشر كتلة الجسم.