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Research Article

Serum levels of food intake hormones and the metabolic syndrome

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Abstract. Objective: The aim of this study is to evaluate on the one hand the relationships between adipokines, food intake hormones and MS components, respectively insulin resistance, and on the other hand, whether the level of anthropometric indices is associated with adipokines, food intake hormones and insulin resistance. Methods: the study includes 60 patients, divided into two groups, with and without MS. The two groups are similar in terms of age and gender. Patients were evaluated for anthropometric evidence, biological samples and serum levels were taken for ELISA measurements of adipokines and food intake hormones. Results: Anthropometric indices are statistically significantly higher in patients with MS compared to those without MS. Also, both adipokines and food intake hormones correlate with MS features: adiponectin correlates statistically significantly with three of the components of MS (diabetes [p=0.04], hypertriglyceridemia [p = 0.02] and low levels of HDL cholesterol [p = 0.05]), and ghrelin correlates with two of the elements of MS (obesity [p = 0.03] and low HDL cholesterol [p = 0.006]) Conclusions: Our study complements the existing studies, managing to strengthen the importance of anthropometric indices in the prediction of MS features, but also the importance of adipokines, respectively food intake hormones in the appearance of MS components and insulin resistance.

Key Words: metabolic syndrome, food intake, ghrelin, leptin

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Introduction

Obesity, an essential element of the metabolic syndrome, has reached epidemic proportions, with over 1.9 billion subjects being overweight and obese. The attention is so great that in the last 2 decades various mechanisms have been researched which could be involved in its development (the role of inflammation, the influence of the immune system, insulin sensitivity, etc.) as well as the links with insulin resistance, diabetes, common elements of the metabolic syndrome (Saltier et al 2017). The Hoorn study has shown that the body mass index (BMI), respectively the ratio between the perimeter of the waist and the hip are clear predictors of diabetes mellitus (DM). Currently, it is considered that anthropometric indices, such as the ratio between the perimeter of the waist and the hip, respectively the percentage of adipose tissue, are the best predictive elements of NAFLD (nonalcoholic fatty liver disease). The studies are in a continuous debate on the best predictors for diabetes, respectively NAFLD, raising the hypothesis on the dominant role of visceral adiposity (Smijder et al 2003; Almeida et al 2018). Ghrelin, a peptide which contains 28 amino acids, is produced by the stomach. Low blood sugar levels, starvation, are responsible for accelerating it and, ultimately, increasing appetite. There are many studies that try to assess the behavior of ghrelin in relation to physical activity: some studies concluding that physical activity inhibits the appetite by suppressing ghrelin production, others consider the existence of other mechanisms not targeting ghrelin, incompletely explained mechanisms (Toshiani et al 2003; Broom et al 2007). Leptin, the anorexigenic adipokine, also plays a role in modulating insulin sensitivity, with some considering that elevated leptin levels in patients with NASH are an indication of its inability to stimulate liver lipid turnover (Grill et al 2002; Aller et al 2012). Adiponectin, identified in 1996, is a protein composed of 144 amino acids, has a protective role on the vascular endothelium, anti-inflammatory, improves insulin sensitivity (Matsuzawa et al 2005; Alberti et al 2006). The aim of this study is to assess the relationships between adipokines, food hormones and MS components, respectively insulin resistance. And on the other hand, we studied the relationship between anthropometric indices and adipokines, food intake hormones and insulin resistance.

Patients and methods

The study was analytical, cross-sectional, retrospective, observational, case-control.

A total of 60 outpatients (20 with MS, study group and 40 without MS, control group) were selected from the The Regional Institute of Gastroenterology and Hepatology "Octavian Fodor", Cluj-Napoca, during March-December 2015. Throughout the study all of the Helsinki rules were observed. Our study protocol was approved by the Ethics committee of "Iuliu Hatieganu" University of Medicine and Pharmacy, Cluj-Napoca and the ethics committee of Prof. Dr. Octavian Fodor's Regional Institute of Gastroenterology and Hepatology. The study procedures were applied to patients after signing the written informed consent. The two groups (study and controls) were similar regarding age and gender distribution.

According to the International Federation of Diabetology criteria, subjects were considered with MS if they had abdominal circumference over 94 cm for men and 80 cm for women, to which at least two of the following criteria were added: (i) blood pressure (BP) > 135/85 mmHg (HBP) or antihypertensive treatment, (ii) the serum blood level of triglyceride >150 mg/dL or the presence of hypertriglyceridemia treatment, (iii) HDL cholesterol <40 mg/dL in men and <50 mg/dL in women, (iv) basal blood glucose >100 mg/dL or the presence of T2DM. (Alberti et al 2006). Study cases were represented by adult patients who were diagnosed with MS (meeting at least 3 of the 5 criteria), while the control group were represented by patients without MS. The cases and controls were similar in age and gender distribution.

Exclusions criteria: viral, autoimmune liver diseases (excluded either by their charts at the family doctor, or by blood viral markers in our department), and alcoholic liver disease (by applying the rapid test of alcohol use disorders identification (AUDIT)). Iatrogenic hepatitis, right heart diseases (CPHD-chronic pulmonary heart disease, grade 3 tricuspid regurgitation, constrictive pericarditis), obesity due to hypercorticism, hypothyroidism, drugs (tricyclic antidepressants, corticosteroids, hormonal therapies, anticonvulsants), acute inflammatory diseases, oncologic active pathologies.

The collection of data: history, physical examination and anthropometric indices were determined by trained staff. The study protocol also included the electronic evaluation of medical history, treatments followed, as well as their subsequent monitoring. Chronic diseases, findings from clinical evaluation, anthropometric indices, laboratory tests, the level of hormones that control food intake and abdominal ultrasound results were recorded in a database.

Abdominal circumference was measured at half distance between the costal rebord and the iliac crest. The perimeter of the hip was determined immediately below the gluteal muscles (Snijder et al 2003). Weight was taken in orthostatism using an electronic scale and the height was evaluated with a vertical centimeter. The last two parameters helped later to the measurement of BMI, representing the weight-to-square height ratio. BP was evaluated at 15 minutes from the arrival to the general practice's office with an electronic device. The percentage of fat tissue was calculated with a body fat analyzer, ("OMRON bf 306").

Biological samples were drawn by trained staff and the blood count was harvested in a special vacutainer which was processed in the hospital's laboratory, as well as biochemical tests, for the determination of the level of high-density lipoproteins (HDL) and those with low density (LDL). We mention that the serum insulin level and glycated hemoglobin were processed at a private laboratory (Bioclinica). The glycated hemoglobin level was determined by high–performance liquid chromatography, having as a Bio-Rad producer, and serum insulin level was determined by Bioclinica lab from Targu Mureş, by the chemiluminescence method, produced by Roche.

Blood samples were harvested in the morning, after 12 hours fasting, and kept in appropriate conditions after centrifugation. Plasma ghrelin levels were measured by ELISA technique, using the FineTest kit, leptin, also measured by the ELISA technique, with Biovendor products. We mention that these kits were obtained through a research funding project (PCD 2015, University of Medicine and Pharmacy Cluj Napoca) and they were processed at the Nanomedicine laboratory of the Institute of Gastroenterology "O. Fodor" – Cluj-Napoca, Romania.

Statistical analysis was performed using MedCalc Statistical Software version 19.4.1 (MedCalc Software Ltd, Ostend, Belgium; https://www.medcalc.org; 2020). Descriptive indices of centrality and dispersion (mean ±standard deviation or median with interquartile range defined based on the first quartile and third quartile (Q1-Q3)) were used to describe the data. The choice of appropriate descriptive statistics was made testing the presence of Gauss distribution for quantitative characteristics. Regarding the distribution of qualitative variables we used absolute frequencies (number of patients) and relative ones (%). For testing the differences between cases and controls regarding to quantitative characteristics we used parametric or non-parametric tests like Student – t test for independent samples of Mann Whitney, while Chi square test and Fisher's exact test were used for checking the bivariate associations between qualitative characteristics measured in the sample. For all the bilateral statistical tests we considered an alpha level of 0.05 for statistical significance.

Results

The study includes 60 patients, of whom 20 are with metabolic syndrome, 40 are patients without metabolic syndrome, similar in terms of age and gender.

Regarding weight, there are statistically significant differences ($p < 0.001$), patients with MS presenting higher weight compared to those without MS (Mean & SD 93.70; 17.39 vs 74.58; 13.13). The same statistically significant differences ($p = 0.001$) are found in terms of BMI (25.95 [24.31; 28.56] vs 21.85 [19.77; 24.09]), waist circumference ($p < 0.001$), [MEAN & SD (111.85 ; 14.86 vs 90.65; 9.93], hip circumference ($p < 0.001$) [Mean & SD 120.60; 14.84 vs 103.6; 9.40], waist and hip ratio ($p = 0.007$) [Mean & SD 0.93; 0.05 vs 0.88; 0.07], all being statistically significantly higher in patients with MS compared to those without MS, following the application of the student t-test for unpaired (independent) samples. The percentage of adipose tissue, another defining element, is significantly ($p = 0.001$) higher in patients with MS compared to those without MS [Mean & SD 30.80; 7.83].

From the biological parameters point of view, in the lipid profile we find a tendency to statistical significance ($p = 0.06$) of

triglycerides showing increased values in patients with MS compared to those without MS (135.00 [104.50; 200.75] vs 114.00 [88.25; 153.00]), on the other hand, HDL cholesterol levels are statistically significantly (p = 0.007) lower in patients with MS compared to those without MS [43.00 (36.75; 51.50) vs. 52.50 (43.50; 64.50)].

For the glycemic profile, our study showed statistically significantly (p = 0.000) higher results of insulinemia [21.45 (12.95; 28.75)] in patients with MS compared to those without MS [11.90 (7.10; 14.50)], of glycated hemoglobin (p <0.001) [Mean & SD] in patients with MS [6.25 (5.67; 8.00)] compared to those without MS [5.60 (5.40; 5.87)] and of the HOMA index (p <0.001) in patients with MS compared to those without MS [5.25 (3.54; 9.37) vs. 2.68 (1.61; 3.43)].

From the point of view of the hormones that control food intake, the serum value of ghrelin is higher in patients with MS compared to those without MS (411.905 [79.43; 1148.76] vs 377.56 [54.56; 1889.72]), lower levels of leptin in those with MS compared to those without MS (3.71 [3.30; 59.50] vs 3.75 [2.43; 4.40]), but without statistical significance (p = 0.259).

Compared to the MS components, we can find that ghrelin is significantly (p = 0.038) higher in obese patients compared to nonobese (1336.91 [274.04; 2032.16] vs 571.59 [64.94; 1435.10]) and significantly lower (p = 0.006) in patients with low HDL cholesterol levels compared to those with normal levels (94.20 [43.09; 823.35] vs 671.60 [208.36; 2085.20]).

Compared to triglyceride levels, ghrelin is higher in patients with MS compared to those without MS [561.88 (124.44; 1093.70) vs 241.80 (54.56; 1889.72)] and in those with diabetes compared to those without diabetes [492.19 (124.44; 1002.24).) vs 329.38 (56.07; 1766.57)], but without statistical significance (p = 0.67).

Regarding leptin, we can find that it is statistically significantly (p = 0.04) higher in female patients [4.11 (3.41; 4.76) vs 3.52 (2.38; 4.16)], and with a tendency to statistical significance (p = 0.1) higher in diabetic patients [4.18 (3.36; 169.00); 3.70 (2.63; 4.40)].

Regarding adiponectin, it is statistically significantly (p = 0.04) lower in patients with diabetes compared to those without diabetes [15.88 (11.21; 22.10) vs. 21.40 (17.31; 23.94)]. Also, adiponectin is statistically significantly (p = 0.02) lower in patients with hypertriglyceridemia, compared to those with normal triglyceride values [17.31 (15.42; 21.66] vs. 22.35 [17.44; 24.33]) and statistically significantly (p = 0.05) higher in patients with low serum HDL cholesterol levels [22.13 (17.28; 24.16) vs 18.87 (13.31; 22.36)]

In all patients studied we can see a positive correlation with a tendency to statistical significance (p = 0.01) between ghrelin and HDL, with a correlation coefficient (r) of 0.323, a negative correlation between leptin and cholesterol (r = -0.270 ; p = 0.03) but also between leptin and age (r = -0.416, p = 0.001).

We also found a negative conection, statistically significant, between adiponectin and markers of insulin resistance (insulinemia [r = -0.255; p = 0.04], HOMA index [r = -0.275; p = 0.03]).

In terms of anthropometric indices, we can see that there is a negative correlation between BMI and adiponectin levels in all patients, with a tendency to statistical significance (p = 0.01, r = -0.327), which is also maintained in the MS group, but being intensely statistically significant (p = 0.009, r = -0.57).

We find the same negative correlation between the waist to hip ratio and the level of adiponectin in all patients studied (p = 0.008, r = -0.338), which is maintained in patients without MS (p = 0.008, r = -0.41) . There is a negative correlation between age and leptin, in patients with MS (p = 0.001; r = -0.68) and without MS (p = 0.04, r = -0.31). We find the same negative correlation between age and adiponectin level in patients with MS (p = 0.009, r = 0.56), a correlation that is no longer observed in patients without MS (p = 0.7, r = 0.06).

Regarding the lipid profile, we found a negative correlation between cholesterol and leptin levels in all patients (p = 0.001, r = -0.27), which is maintained also in those without MS (p = 0.05; r = -0.31). Regarding HDL cholesterol, we find a positive correlation between it and the level of adiponectin in all patients studied (p = 0.03; r = -0.27), which is maintained in those without MS (p = 0.003; r = -0.31), but we find the same positive correlation between HDL cholesterol and ghrelin in patients without MS (p = 0.07; r = 0.28), which is no longer observed in those with MS (p = 0.38, r = 0.20).

Regarding the insulin resistance markers (insulinemia, HOMA index) we found a negative correlation with the level of adiponectin in all patients studied (p = 0.04, r = -0.255; p = 0.03, r = - 0.275).

Discussions

The review state that certain clinical parameters are predictive diagnosis elements both for MS and for some altered biological samples (Han et al. 2002; Goldani et al. 2015; Knowles et al. 2011). From a clinical point of view, our study showed that there are statistically significant differences in abdominal circumference, waist circumference, hip, waist to hip ratio, and the percentage of fat tissue among MS patients compared to those without MS. Finding these differences between the clinical indices from the 2 groups, we also assessed the biological parameters.

From the lipid profile point of view, a basic element in the development of MS, we find an increase with a tendency to statistical significance of the triglyceride level, being higher in patients with MS compared to those without MS, but also of the level of the HDL cholesterol, which is lower in patients with MS compared to those without MS. Regarding the glycemic profile, we find that the levels of insulinemia, glycosylated hemoglobin and HOMA index are higher in patients with MS than in those without MS.

Our study confirms the literature data regarding these links, some authors consider that the ratio between waist circumference and hip is a better predictor of MS events, others consider that the abdominal circumference is the one that best reflects the level of adiposity and the prediction of the events within the MS (Ho et al 2001). Another study on a Chinese population aged over 40, establishes that there are also differences depending on the gender of the prediction of cardiovascular events, the BMI being considered a good predictor in men, the ratio between the perimeter of the waist and the hip - predictor in women. Thereby, we can consider that anthropometric indices are valuable tools in the diagnosis and prediction of metabolic syndrome complications (He et al 2012).

Regarding food intake hormones, the literature states that ghrelin levels are lower in patients with MS compared to those without

MS, probably due to the pathophysiological mechanisms underlying the metabolic syndrome (role of dysfunctional adipose tissue, insulin resistance, etc.). Our study did not find statistically significant differences. In depth, studies have tried to determine which of the MS elements would be associated with low ghrelin levels. Ghrelin has been shown to negatively correlation with abdominal circumference, triglyceride levels, blood sugar, BMI, and hypertension and positively correlation with HDL levels (Ukkola et al 2006; Chendraui et al 2014).

Our study confirms the literature data, establishing a positive correlation of ghrelin with HDL levels in all patients studied, which is maintained in those without MS. Regarding insulin resistance markers, our study found a negative correlation between them and ghrelin levels in all patients studied, but especially in those without MS, but the results were without statistical significance. We also found that ghrelin is statistically significantly lower in patients with low HDL levels and statistically significantly higher in obese patients.

Regarding leptin, as in the literature, in our study we find that it does not differ statistically significantly between the 2 groups, being slightly lower in patients with MS. Also, compared to the MS components, we find that leptin is higher in diabetic, hypertensive, obese and hypertriglyceridemia patients, the results being without statistical significance, results in accordance with the literature. Lee et al. on a study of menopausal women with and without MS, demonstrates statistically significantly lower levels of leptin in women with MS compared to those without MS, more likely due to hormonal changes that will lead to metabolic changes. Our study revealed an intense negative relation between leptin levels and age in all patients studied, which is also preserved in those with MS, probably due to hormonal changes that will lead to lipid changes (Lee et al 2012). Regarding the lipid profile, leptin links negatively with all the elements of MS, but especially with cholesterol levels, being statistically significant in all patients, especially those without MS.

Compared to markers of insulin resistance (IR), leptin correlates negatively with all elements of IR in both patients with and without MS, but the results are without statistical significance. The literature states that leptin is strongly correlated with insulin resistance, probably due to its role in modulating insulin sensitivity, but also the fact that some authors believe that liver changes in metabolic syndrome are also due to the inability of leptin to stimulate liver lipids turnover (Grill et al 2002; Aller et al 2012).

From the literature data also state that there is a positive, highly statistically significant correlation between adiponectin levels and age, raising several hypotheses. Some believe that the decrease in renal function with age would have an effect on serum adiponectin, others believe that increased visceral fat with age is the element that contributes to changes in adiponectin values (Koh et al. 2008). Our study reveals a positive, highly statistically significant correlation between adiponectin levels and age. We mention that these patients did not show changes in renal function, the average age being 60 years.

Compared to the lipid profile, a study in monkeys showed that plasma levels of adiponectin decrease with increasing obesity, increased insulinemia and insulin resistance (Goodarzi et al 2007; Huang et al 2004). Our study confirms the literature data, finding an intensely positive correlation between adiponectin and HDL and total cholesterol levels in MS patients but also a negative correlation, highly statistically significant between insulin resistance markers (HOMA index, insulinemia) and serum adiponectin levels. The mechanism by which adiponectin regulates insulin sensitivity is not fully elucidated, the suspicion of a gene polymorphism present in adiponectin is raised. (Goodarzi et al 2007). Compared to the elements of the metabolic syndrome, our study shows that adiponectin is higher in diabetic, hypertriglyceridemia and low HDL cholesterol patients. The limited number of patients with MS could explain the lack of significance of the correlation between the MS parameters and the anthropometric characteristics of the subjects. Consequently, the small number of patients has restricted the use of a different multivariate method for the association between MS and anthropometric factors and laboratory criteria.

Conclusions

Our study showed the importance of anthropometric indices in diagnosis of metabolic syndrome. We have also shown that ghrelin, the orexigenic hormone, is significantly higher in MS patients with obesity and low HDL, two of the 5 features of metabolic syndrome, and adiponectin (known as adipokine with a protective role regarding MS traits) is statistically significantly lower in patients with diabetes mellitus, in those with low levels of HDL cholesterol and in those with high levels of serum triglycerides. Thereby, our study strengthens the literature data according to which adipokines, respectively food intake hormones could be valuable tools in diagnosis or predicting the components of the metabolic syndrome.

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