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Relationship between Sex Hormone-Binding Globulin (SHBG) and Insulin-Like Growth Factor-I (IGF-I) with Metabolic Syndrome

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GLUCOSE intolerance, insulin resistance, hypertension, visceral obesity, and dyslipidemia are the major components of metabolic syndrome (MS). To evaluate the association between serum SHBG and IGF-1 levels and the risk of MS, Furthermore, to determine the correlations between SHBG and IGF-1 and the main components of MS. A total of 402 subjects with and without MS were enrolled in this study (MS=156, Non-MS=246) aged > 18 years. The age, height, weight, BMI, HC, WC, and incidence of diabetes, hypertension and dyslipidemia of all cases were recorded. The collected serum samples were used to assess lipid profile, glucose and insulin levels. The levels of LDL-cholesterol were calculated using Friedewald's formula. Insulin resistance was measured (as HOMA score). The levels of serum SHBG and IGF-1 were measured using Elisa technique. A positive relationship between SHBG and MS was detected, however no such correlation was observed concerning IGF-1. There were positive correlations between SHBG and main components of MS; with insulin, HOMA-index, TC, TG and HDL. Conversely, IGF-1 showed negative correlations. Finally, SHGB was more sensitive (63.5%), accurate (61.9%) than IGF-1 (51.9%), accuracy (59%). Our study reveals that lower SHBG is more strongly associated with metabolic syndrome and its main components that lower IGF-1. SHBG could be the essential driver of these relations, conceivably reflecting its association with insulin sensitivity; however more studies are required to confirm this relationship.

Keywords: Metabolic syndrome (MS); Sex hormone-binding globulin (SHBG); Insulin like growth factor-1 (IGF-1); Insulin resistance; Visceral obesity.

Introduction

Metabolic syndrome (MS) is an important reason for mortality and morbidity in industrial nations [1]. It is described by the mixture of several disorders including insulin resistance, high blood pressure, obesity, dyslipidemia, and a pro-inflammatory state [2]. The metabolic syndrome is intensely related to a lifestyle characterized by an easy access to unlimited supply of high caloric, little nutrient-dense, foods and physical inactivity [3]. Psychosocial stress has also been proposed to contribute, with most metabolic constituents are more prevalent in socioeconomically deprived populations [4]. The incidence of MS associates with the worldwide prevalence of obesity and is developing at a disturbing rate, influencing over 20% of the global grown-up population [5].

Recently, in the pathogenesis of MS, non-alcoholic fatty liver disease (NAFLD) and type 2 diabetes mellitus (T2DM), further consideration has been paid to the supposed organosilanes, proteins with both endocrine or/and paracrine actions [6]. These contain most identified adipokines (mostly created by adipose tissue), myokines (principally formed by skeletal muscles) and hepatokines (mainly made by the liver) [7]. It was revealed that the liver could influence the glucose and lipid metabolism by hepatokines discharged into the blood and MS appears to be accompanying with altered hepatokines creation. Insulin like growth factor-1 (IGF-1) and sex hormone-binding globulin (SHBG) are considered as the most important hepatokines.

Sex hormone-binding globulin is a serum steroid-transporting protein that is made in the

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liver. Many reports have demonstrated that decreased serum SHBG levels are associated with MS components (insulin resistance and obesity), T2DM and NAFLD [8; 9]. Insulin-like growth factor-I is a polypeptide hormone formed mostly by the liver in response to the endocrine growth hormone stimulus and controls both body composition and metabolism. There is mounting evidence suggesting that IGF-I, besides its mitogenic action, plays an active role in the regulation of protein, carbohydrate and lipid metabolism [10]. Insulin like growth factor-1 has been reported to predict the occurrence of liver steatosis in obese patients [11]. It codes for a membrane glycoprotein involved in insulin sensitivity [12]. Our study was to explore the clinical significance of SHBG and IGF-1 with MS as well as with its major components.

Subjects and Methods

Subjects

The study was performed on consecutive adults (of both sexes) who were recruited from the Medical Center of Excellence - National Research Center. This study was conducted from May 2015 to June 2016. All subjects were of the age more than 18 years old and were asked about their family history, individual health history and current medications (anti-hypertensive, oral hypoglycemic agents and lipid-lowering medicine). Subjects with any malignancy, liver cirrhosis, taking hormones, or antifungal agents, were excluded from the study. Written informed consent was obtained from each individual, and the study protocol was reviewed and approved by the Medical ethics Committee of National Research Center.

All subjects (n= 402) in the study were divided into MS (n=156) and Non-MS (n=246) groups; metabolic syndrome was diagnosed according to guidelines from the National Heart, Lung, and Blood Institute (NHLBI) and the American Heart Association (AHA) [13], metabolic syndrome was diagnosed when a patient has at least 3 of the following 5 conditions: 1) Waist circumference ≥102 cm in men or ≥88 cm in women; 2) Blood pressure ≥130/85 mm Hg or receiving drug therapy for hypertension; 3) High-density lipoprotein (HDL) < 40 mg/dL in men or Cholesterol < 50 mg/dL in women or lipid medication use; 4) Fasting glucose ≥100 mg/dL or receiving drug therapy for hyperglycemia; 5) Triglycerides ≥150 mg/dL or receiving drug therapy for hypertriglyceridemia.

Measurements

All subjects underwent the physical examination and fasting blood samples (3 ml) were withdrawn by venipuncture for laboratory evaluation after 14 h of overnight fasting. The body mass index (BMI) was derived from body weight (in kilograms) divided by the square of body height in meters. Waist circumferences (WC) were measured by standard form to the nearest 0.1 cm. Hip circumference (HC) was measured at the maximum 2 protruding part of buttocks at the level of the greater trochanter with the patient wearing minimal clothing and feet together. Subjects were seated with legs uncrossed and were asked to refrain from talking for 10 min. Blood pressure and heart rate measurement were taken three times, with at least a 1-min interval between two consecutive readings using an automatic blood pressure monitor (using a mercury sphygmomanometer).

Biochemical analyses

Fasting plasma glucose levels and serum levels of total cholesterol, triglyceride, and HDL were measured with an enzymatic colorimetric method (Stanbio Laboratory, USA). LDL was calculated using Friedewald's formula [14]. Serum sex-hormone-binding globulin (SHBG) and serum human insulin-like growth factors 1(IGF-1) were assayed by an enzyme-linked immunosorbent assay (SHBG, IBL International GmbH, Germany and EIAab system, respectively).

Statistical analysis

Sample size calculation was done using Stats Direct statistical software version 2.8 for MS Windows, Stats Direct Ltd., Cheshire, UK. Analysis of data was done by IBM computer using SPSS (statistical program for social science version 20) (SPSS Inc., Chicago, IL, USA). Independent sample -t- test was used for comparison between the two groups. Correlations between different variables and metabolic syndrome were analyzed using Spearman correlation test.

Results

Characteristics of subjects with and without metabolic syndrome

The general characteristics of patients with and without MS are illustrated in Table 1. Significant differences were found according to age, BMI, WC, HC, waist/hip ratio, obesity (extreme obesity group only), DBP, SBP, insulin, HOMA-index, lipid profile. However, no significant differences

were found in obesity (for overweight and obese groups).

Frequencies of SHBG and IGF-1 in subjects with and without metabolic syndrome

The frequencies of SHBG and IGF-1 in subjects with and without MS are presented in Table 2. Regarding SHBG, a significant difference occurred with the p value <0.05 but IGF-1 showed a non-significant difference with the p value >0.05.

Correlation between ILGF-1 and metabolic indices and lipid profile

Table 3 shows the spearman's correlation coefficients between IGF-1 and metabolic indices and lipid profile. It was noticed that IGF-1 was negatively correlated with insulin, HOMA-index, TC and LDL and positively correlated with FBG, TG and HDL.

Correlation between SHGB and metabolic indices and lipid profile

Table 4 shows the spearman's correlation coefficients between SHGB and metabolic indices and lipid profile. It was found that SHGB was negatively correlated with FBG and LDL and positively correlated with insulin, HOMA-index, TC, TG and HDL.

Percent sensitivity, specificity, positive and negative predictive values (PPV, NPV) and SHGB and IGF-1 accuracy in MS

Table 5 indicates that SHGB has more sensitivity (sn=63.5%), accuracy (61.9%) with significance value, P=0.020 than IGF-1 with sensitivity (sn=51.9%), accuracy (59%) with P-value= 0.089 (Fig.1&2).

TABLE 1. Characteristics of subjects with and without metabolic syndrome.

Characteristics	Total (n = 402)	None Metabolic Syndrome (n = 246)	Metabolic Syndrome (n = 156)	P value	P* value
Male / Female	171 / 231	129 / 117	42 / 114	-	-
Age (Years)	39.53 ± 10.69	37.61 ± 10.66	42.56 ± 10.11	**0.009**	**<0.05**
BMI	32.89 ± 9.55	28.26 ± 8.44	40.18 ± 5.99	**0.000**	**<0.05**
Waist Circumference (WC)	98.57 ± 16.02	91.24 ± 14.88	110.12 ± 9.78	**0.000**	**<0.05**
Hip Circumference (HC)	113.66 ± 13.2	107.83 ± 11.78	122.85 ± 9.65	**0.000**	**<0.05**
Waist/Hip Ratio	0.86 ± 0.07	0.84 ± 0.07	0.9 ± 0.07	**0.000**	**<0.05**
Obesity					
Overweight	27 (6.7%)	21 (77.8%)	6 (22.2%)	0.096	**<0.05**
Obese	135 (33.6%)	66 (48.9%)	69 (51.1%)	0.881	>0.05
Extreme Obesity	102 (25.4%)	21 (20.6%)	71 (79.4%)	**0.001**	**<0.05**
Diastolic BP (mmHg)	121.17 ± 15.17	114.27 ± 11.76	132.06 ± 13.53	**0.000**	**<0.05**
Systolic BP(mmHg)	79.76 ± 12.05	75.3 ± 9.73	86.79 ± 12.08	**0.000**	**<0.05**
FBG (mmol/l)	27.19 ± 43.71	24.6 ± 36.58	31.26 ± 53.2	**0.003**	**<0.05**
Insulin (mIU/ml)	8.87 ± 4.35	7.92 ± 3.48	10.37 ± 5.14	**0.002**	**<0.05**
HOMA-index	2.05 ± 1.18	1.68 ± 0.77	2.63 ± 1.45	**0.000**	**<0.05**
TC (mg/dl)	236.32 ± 64.05	212.91 ± 58.98	273.23 ± 53.88	**0.000**	**<0.05**
TG (mg/dl)	179.27 ± 78.31	144.18 ± 69.93	234.61 ± 55.88	**0.000**	**<0.05**
HDL (mg/dl)	56.58 ± 21.9	65.24 ± 22.61	42.91 ± 11.29	**0.000**	**<0.05**
LDL (mg/dl)	143.89 ± 66.01	118.83 ± 60.42	183.41 ± 54.43	**0.000**	**<0.05**

BMI: Body mass index; TC: Total cholesterol; TG: Triglyceride; HDL: High-density lipoprotein; LDL: Low-density lipoprotein; FPG: Fasting plasma glucose.
Numeric variables are described by mean ± SD, and categorical data are expressed as number (%).
P value for comparison between total subjects, MS and Non-MS groups.
P* value for comparison between the MS and Non-MS groups.
P value <0.05 was considered as statistically significant.

TABLE 2. Frequencies of SHBG and IGF-1 in subjects with and without metabolic syndrome.

Characteristics	Total (n = 402)	None Metabolic Syndrome (n = 246)	Metabolic Syndrome (n = 156)	P value	P* value
ILGF1 (Pg/ml)	6.75 ± 1.59	6.92 ± 1.63	6.49 ± 1.49	0.130	>0.05
SHGB (nmol/L)	3.37 ± 1.35	3.52 ± 1.28	3.13 ± 1.43	**0.020**	**<0.05**

SHBG: Sex-hormone-binding globulin; IGF-1: Human insulin-like growth factors 1.
Data are presented as mean ± SD.
P value for comparison between total subjects, MS and Non-MS groups.
P* value for comparison between the MS and Non-MS groups.
P value <0.05 was considered as statistically significant.

Discussion

Metabolic syndrome is considered as one of the main public health problems of the 21th century. In the current study, we found significant differences between metabolic and non-metabolic syndrome groups according to BMI; WC; HC; SBP; DBP; FBG; HOMA-index; and lipids; which are the main components of the metabolic syndrome. Our finding could be explained as MS is a group of risk factors; containing increased TG levels, decreased HDL, raised central abdominal obesity, increased FBS, hyperinsulinemia, and/or high BP [15].

Growth hormone (GH) is the main regulator of postnatal growth and also controls both body composition and metabolism. The growth promoting the action of GH is mainly mediated by IGF-I, a component of the insulin-like growth factor system [16].

The mechanisms underlying the association between IGF-I levels and MS are still largely unknown. The insulin-like activity of IGF-I may account for a positive effect on insulin resistance which is closely associated with metabolic syndrome [17]. This may be due to resemblances between insulin and IGF-I indicate the probability of IGF-I involvement in the phenotypic expression of this disorder [18]. The increased insulin levels can induce a down-regulation of IGF-I secretion by the liver and other tissues, as a compensatory homeostatic mechanism, caused most likely through a differential variation of IGF-I production. This could be responsible for the increment levels of IGF-I indicated in accordance with states of IR, as the MS [19].

On the contrary, the present study suggested that serum IGF-I level was not significantly associated with MS. This could be explained by the greater incidence of IR and MS in adult population compared with younger individuals

TABLE 3. Correlation between ILGF-1 and metabolic indices and lipid profile.

Variables	Correlation Coefficient	P value
FBG	0.411	0.002
Insulin	-0.018	0.920
HOMA-index	-0.008	0.965
TC	-0.189	0.286
TG	0.008	0.966
HDL	0.083	0. 64
LDL	-0.228	0.194

TABLE 4. Correlation between SHGB and metabolic indices and lipid profile.

Variables	Correlation Coefficient	P value
FBG	-0.116	0.513
Insulin	0.220	0.21
HOMA-index	0.277	**0.045**
TC	0.084	0.636
TG	0.255	0.146
HDL	0.068	0.704
LDL	-0.009	0.961

TABLE 5. Percent sensitivity, Specificity, PPV, NPV and accuracy of SHGB and IGF-1 in Metabolic Syndrome.

Parameter	Area under the curve	Cutoff value	Sensitivity %	Specificity %	PPV	NPV	Test Accuracy	95% CI	P value
SHGB	0.620	2.936	63.5 %	61.0 %	50.8 %	72.5 %	61.9 %	0.518 to 0.721	**0.020**
ILGF1	0.587	6.150	51.9 %	63.4 %	47.4 %	67.5 %	59.0 %	0.488 to 0.687	0.089

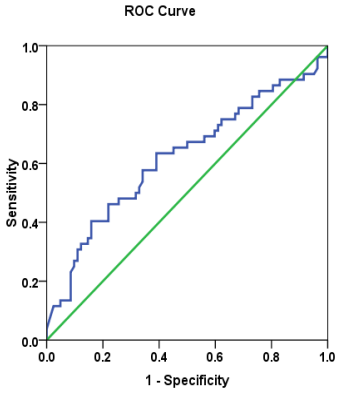

Fig.1. ROC Curve for SHGB in Metabolic Syndrome .

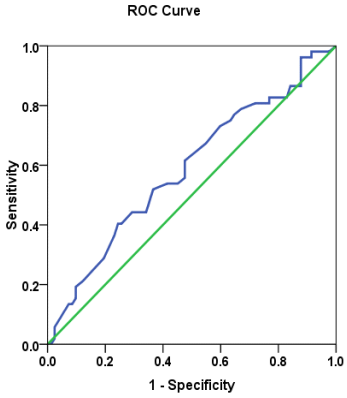

Fig. 2. ROC Curve for ILGF-1 in Metabolic Syndrome

might also be attributable, nevertheless partially, to the decay concentrations of serum and tissue IGF-I with progressing age [20]. Reduced IGF-I levels are independently associated with glucose intolerance, diabetes, abdominal obesity [21; 22] and atherogenic dyslipidemia [23]. There are interesting discrepancies for understanding the physiological relevance of the reduced IGF-I axis in aging. Several studies have suggested that reduced IGF-I activity promotes longevity [24], and a significant amount of evidence has been accumulated indicating that IGF-I might play a role in several pathological conditions commonly seen during aging. These pathologies are associated with oxidative tissular damage. This effect can be an additional mechanism to explain the antioxidant activity displayed by this hormone in conditions of "IGF-I deficiency" [25]. The mechanisms responsible for the effects of IGF-I are not fully understood that require further investigation [26].

Our data reported that serum levels of SHBG were decreased in MS group as compared to non- MS. Our finding was in agreement with Li et al. [19] and Liao et al. [20]; who found that the serum concentration of SHBG was associated with MS. Our data could be explained on the basis that the crucial abnormality detected in MS seems to be IR in peripheral tissues. Since insulin is a powerful suppressor of SHBG generation in the liver, it is conceivable that reduced levels of SHBG might be an initial indicator for MS. Likewise, Heald et al. [27] in an investigation examining Afro-Caribbean, European and Pakistani populaces and Chubb et al. [28] in a population-based study, recommended that SHBG is a potential marker for MS. Recently, Caldas et al., [29] described that a rise of one unit in insulin concentrations lead to a drop of 0.25 units in SHBG concentrations, in a non-interventional study examining 80 subjects with MS.

A powerful relationship was observed between lipids and SHBG, making SHBG to be expected as a valuable predictor for the metabolic syndrome distinct by the National Cholesterol Education Program Adult Treatment Panel [30]. This description excludes insulin resistance as a risk factor for this disorder, and along these lines, it has a tendency to be more weighted toward lipid constituents and abdominal obesity as compared to the WHO explanation of the metabolic syndrome. Since IGF-1 does not have a relationship with either insulin or insulin resistance, and its relation with SHBG is stronger than for lipids, IGF-1 is most likely less significant in expecting the MS well-defined by the Program of National

Cholesterol Education.

Conclusion

In conclusion, serum SHBG level inversely correlates with the prevalence of metabolic syndrome, but not serum level of IGF-1. Metabolic Syndrome is increasing in emerging countries, making this disease a public health problem. Although, the exact mechanisms is linking MS disease remain only partly known. We recommended that further research is warranted for the better understanding of the pathophysiology of MS and for better identifying potential therapeutic targets in this ever growing disease.

Declaration of interest

The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of the research reported.

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العلاقة بين الهرمون الجنسي المرتبط بالجلوبيولين (SHBG) وعامل النمو الأنسولينى- ١ (IGF-١) ومتلازمة التمثيل الغذائي

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تعتبر كلاً من الحساسية المفرطة تجاه الجلوكوز، ومقاومة الأنسولين، وارتفاع ضغط الدم، والسمنة إلى جانب ارتفاع نسبة الدهون من المكونات الرئيسية لمتلازمة التمثيل الغذائي. لتقييم العلاقة بين مستويات SHBG ، IGF-١ في مصل الدم وخطورة حدوث متلازمة التمثيل الغذائي ، وعلاوة على ذلك، تحديد الارتباطات بين SHBG ، IGF-١ والمكونات الرئيسية لمتلازمة التمثيل الغذائي.

تمت الدراسة على ٤٠٢ حالة من الأشخاص الذين يعانون والذين لايعانون من متلازمة التمثيل الغذائي فوق ١٨ عام. وقد تم تسجيل بيانات الحالات كاملة من حيث السن والطول، والأرتفاع، ومؤشر كتلة الجسم الكر-والكوليسترول ونسبة وجود السكرو الضغط وارتفاع معدل الدهون . تم تقدير نسبة الدهون والجلوكوز في البلازما والانسولين واحتساب مقاومة النسولين، كما تم قياس الأمصال SHBG، IGF-١، عن طريق تقنية الاليزا. وجود علاقة إيجابية بين SHBG مع متلازمة التمثيل الغذائي ولكن لم تتضح تلك العلاقة في وجود IGF-١كما كتن هناك ارتباط ايجابي بين SHBG والكونات الرئيسية لمتلازمة التمثيل الغذائي مثل الإنسولين و HOMA والكوليسترول والدهون الثلاثية والكوليسترول النافع. على النقيض كان هناك ارتباط عكسي في حالة وجود .IGF-١ وأخيراً أظهرت النتائج ان SHBG الأكثر حساسية ودقة بالمقارنة بـ IGF-١.

أظهرت هذه الدراسة عن الارتباط الوثيق بين مصل SHBG و متلازمة التمثيل الغذائي ومكوناتها الأساسية أكثر من IGF-١. وهذا الارتباط يعكس أهمية SHBG كمؤشر اساسي لهذه العلاقات، مما يعكس علاقته مع حساسية الأنسولين ولكن هناك حاجة لمزيد من الدراسات لتأكيد هذه العلاقة.