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Studying Myostatin as a Diagnostic Indicator in Sera of Iraqi Women with Metabolic Syndrome

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Abstract

Obesity in the abdominal area, insulin resistance, hyperglycemia, dyslipidemia, and hypertension are all included as part of metabolic syndrome. Myocytes manufacture and release the myokine known as myostatin, which acts on muscle cells inhibiting cellular growth. This study aimed to compare Myostatin along with other biochemical parameters (Fasting blood sugar (FBS), cholesterol, high-density lipoprotein (HDL), triglycerides (T.G), low-density lipoprotein (LDL), and very Low-density lipoprotein (VLDL)) in sera of Iraqi with metabolic syndrome patients and healthy subjects. Eighty women were separated into two groups: group I, 40 metabolic disorders patients, and group II, 40 healthy people, which served as the control group. Myostatin levels significantly increased compared to the control group in the group with metabolic syndrome, according to the data $p > 0.001$. A definite cut-off value was visible on the Myostatin ROC curve (5), in the metabolic syndrome. The outcomes suggest myostatin as possible diagnostic tool for metabolic syndrome.

Keywords: Cholesterol, Fasting blood Glucose, Myostatin, Metabolic Syndrome, Low-density lipoprotein.

Introduction

Hyperglycemia, hyperlipidemia, and hypertension are some of the prevalent illnesses referred to as "metabolic syndrome." Additionally, hypertension and cardiovascular disease are common in those with this condition. It is thought that certain persons experience cardiovascular disease, diabetes, dyslipidemia, and hypertension concurrently because these disorders do not randomly cluster together. The onset of numerous metabolic illnesses has long been believed to be significantly influenced by insulin resistance[1,2]. Except for hyperglycemia critical, the direct roles of intra-abdominal visceral fat accumulation in the emergence of various risks and cardiovascular disease have been acknowledged in a consensus on the concept of metabolic syndrome from the International Diabetes Federation. However, insulin resistance cannot be used to interpret these diseases that cause metabolic syndrome. The primary mechanisms in these lifestyle-related diseases may involve the secretion of several adipokines by adipose tissue [3,4]. Myostatin, a secreted protein, inhibits the growth of skeletal muscle. Cells in the myotome and growing skeletal muscle express the hormone myostatin, which controls the number of muscle fibers that are ultimately produced during

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embryogenesis. Adults' skeletal muscles manufacture the myostatin protein, which circulates in the blood and regulates the synthesis of new muscle fibers [5,6]. Myostatin, a potent member of the TGF-beta/bone morphogenetic protein (BMP/TGF) group of secreted proteins, inhibits the growth of skeletal muscle. There is an accumulating proof that myostatin also impacts the growth and metabolic condition of different tissues, such as the liver and adipose tissue. This is in addition to its influence over the growth of skeletal muscle, which has an impact on the overall amount of body mass [7,8] . Obesity is associated with greater myostatin expression, according to a number of studies[9,10]. Wild-type mice fed a high-fat diet for a month and genetically obese, leptin-deficient ob/ob mice both had increased skeletal muscle and fatty tissue myostatin mRNA levels[11,12].

Additionally, compared to non-obese women, secretion of myostatin by myotubes produced from myoblasts isolated from muscle biopsies is higher in obese human participants. Myostatin protein levels in the blood and muscles both rise[12] . On the other hand, as weight goes down, myostatin mRNA levels reduce. Upon two weeks of daily administration of recombinant leptin, myostatin mRNA levels decreased in the muscle and adipose of hyperphagic, obese, hyperinsuline and hyperglycemic mice ob/ob[13,14] . Experimental manipulation of myostatin signaling or expression can also have a major impact on the onset of obesity in rats. For example, muscle-specific increased expression of myostatin resulted in a decrease in muscle mass and an increase in the mass of the epididymal fat pad[15,16]. Instead, multiple studies have shown that myostatin inhibition can delay the development of obesity in rats. When myostatin null rats crossed with intrinsically obese ob/ob or agouti fatal yellow rats, the elevated lipid mass, high blood sugar, high cholesterol, and high insulin levels that are frequently seen in these animals with this condition reduced. By modulating TNF expression, which can block insulin's effects on glucose uptake, myostatin may subtly modify glucose uptake[17,18]. This study aimed to compare Myostatin along with other biochemical parameters between those with and without metabolic syndrome.

Materials and Methods

Study Subjects:

Researchers worked along with the National Diabetes Center at Al-Mustansiriya University, Iraq on this investigation 80. 40 female patients with metabolic syndrome and 40 healthy women, with age-range 35 to 60 years, participated in the study. The International Diabetes Federation reported in 2005 that the global consensus on metabolic syndrome served as the foundation for the diagnosis of metabolic syndrome [19,20]. In addition to a patient's BMI above 30 kg/m^2, to be diagnosed with metabolic syndrome, any two of the following four requirements have to be satisfied:

1) Triglyceride levels are high (150 mg/dL) (or particular treatment for this lipid imbalance).
2) Treatment for this lipid imbalance or low HDL cholesterol (40 mg/dL in females).
3) Elevated blood pressure; at least 130 mm Hg for the systolic pressure or 85 mm Hg for the diastolic pressure, or (having received treatment upon having their hypertension diagnosed)
4) Either a diagnosis of type II diabetes or elevated fasting blood sugar (FBS; 110 mg/dl).

Type 1 diabetics, acromegaly, people with severe liver, and kidney diseases were not included in the study. Each patient provided their consent to take part in the research.

Blood Sample Collection and Analysis:

Each person had ten to twelve hours of fasting before venous blood collected (ten milliliters) and two aliquots were created. After a thirty-minute incubation period, the samples were centrifuged (at 1500 g for fifteen minutes). A portion of the acquired serum was used to measure the lipid profile. Criteria of each person including weight and height were measured in kilos (kg). Then the body mass index was determined (BMI) by dividing the weight (in kg) by the square of the length (in m^2) [21]. The kenza (240TX) (Biolabo) equipment and (Biolabo) kit were used to measure the biochemical parameters (FBS, TG, TC, HDL). The ELISA kit was used to measure the myostatin content (My Biosource, America).

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Readings of Blood Pressure

The subject's seated systolic and diastolic blood pressures were found in millimeters of mercury using a sphygmomanometer. Then, the following formula was used to calculate the mean arterial pressure (MAP): MAP equals DBP + (SBP-DBP) / 3 [22].

Statistical Analysis

The mean, standard deviation, and median 25th and 75th percentiles for normal and irregular distributed numerical variables, respectively, were used to interpret the results. The Shapiro-Wilk test was used to see if the data was distributed properly. The (Mann-Whitney) tests were employed to describe numerical variables that were not regularly distributed. The significance level was set at a P value of 0.05. Through the use of receiver operating characteristic (ROC) curve analysis, the myostatin cut-off value was established.

Results and Discussion

The age and BMI of metabolic syndrome patients and, healthy subject are listed in Table 1, by the median (25th and 75th percentiles).

Table 1. The demographic characteristics of metabolic syndrome and healthy subjects.

variables	Metabolic Syndrome	Healthy subject	p-value
Age (Year)	49.0(39.75 -53.0)	46.50 (38.0 – 52.0)	0.31
BMI (kg/m^2)	31.50 (30.0 – 35.0)	24.0 (23.0 – 25.0)	0.00
WHR	0.94 (0.89 – 1.01)	0.83 (0.80 – 0.84)	0.00

There was a significant difference between the two independent means when the gathered data were examined by median (25th and 75th percentiles) using the Man-Whitney test at the p<0.05level.

Table 1, lists data on the median 25th and 75th percentiles of the age distribution of the metabolic syndrome 49.0(39.75 -53.0) years, and the healthy subjects group 46.50 (38.0 – 52.0). The data of BMI distribution of the metabolic syndrome were 31.50 (30.0 – 35.0) patients, and the healthy subjects group 24.0 (23.0 – 25.0). The data of WHR distribution of the metabolic syndrome were 0.94 (0.89 – 1.01) patients, and the healthy subjects group 0.83 (0.80 – 0.84), as shown respectively in Table 1.

The serum levels of cholesterol, T.G, HDL, VLDL, and, LDL metabolic syndrome and healthy subjects. High significant difference appeared in cholesterol, T.G, HDL, VLDL, and, LDL levels when compared with two groups of patient metabolic syndrome and control group with p>0.001 as shown as in Table 2. The cholesterol levels increased significantly in metabolic syndrome patients group 203.0(187.0-216.25) when compared with control group 148.0(136.80-150.0) p>0.001, as shown in Table 2.

The T.G, LDL and, VLDL levels increased significantly in metabolic syndrome patients group 176.50(134.0-191.50), 125.60(108.60-142.35) , 35.30(26.85-38.30), when compared with control group 98.0(75.0-112.98), 80.40(71.77-88.05), 19.80(15.0-22.60), respectively p>0.001 ,while there was a low significant difference in HDL when we compared metabolic syndrome patients group 40.70 (39.05-43.83) with control 46.25(45.0-51.30) ,p<0.001 as shown as in Table 2.

One of the most significant effects of modern lifestyles, which consistently increase the risk for the onset of numerous diseases, is obesity, as is well-known. Markers of obesity include BMI, waist circumference, and body fat percentage. The production of this adipokine is enhanced by an increase in cell count and adipose tissue since obesity is associated with an increase in body fat and this adipokine is produced by adipose tissues[9,23,24].

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Table 2. The Cholesterol, T.G, HDL, VLDL, and LDL of metabolic syndrome and healthy subjects.

variables	Metabolic Syndrome	Healthy subject	p-value
Cholesterol(mg/dl)	203.0(187.0-216.25)	148.0(136.80-150.0)	0.00
T.G(mg/dl)	176.50(134.0-191.50)	98.0(75.0-112.98)	0.00
HDL (mg/dl	40.70 (39.05-43.83)	46.25(45.0-51.30)	0.00
LDL (mg/dl)	125.60(108.60-142.35)	80.40(71.77-88.05)	0.00
VLDL (mg/dl)	35.30(26.85-38.30)	19.80(15.0-22.60)	0.00

The collected data was analyzed by median (25thand 75th percentiles) via the man Whitney test at the o.o5 level, there was a significant difference between the two independent means .

Data analysis of the diastolic blood pressure, systolic blood pressure FBS, and myostatin in metabolic syndrome patients and healthy subjects groups are shown in Table 3.

The means of systolic blood pressure and diastolic blood pressure, revealed a significant increase in the metabolic syndrome group (131.03 ±13.05) and 77.34±25.10 compared to that of the healthy subjects group (122.03 ±17.07),(73.4±15.10) $p<0.05$. The result showed high significant increase in myostatin level when we compared metabolic syndrome group 7.80(6.23-8.80) with healthy subjects 4.10(3.30-4.75) $p<0.001$. The FBS levels increased significantly in patients' group metabolic syndrome when compared with healthy subjects' group $p<0.001$, as shown as in Table 3.

Table 3. The Distolic blood pressure, Systolic blood pressure, FBS, and myostatin of metabolic syndrome and healthy subjects' group.

variables	Metabolic Syndrome	Healthy subject	P value
Distolic blood pressure (mmHg)	77.34±25.10	73.4±15.10	0.05
Systolic blood pressure (mmHg)	131.03 ±13.05	122.03 ±17.07	0.05
FBS(mg/dl)	117.50(92.0-118.0)	88.50(85.90-91.75)	0.000
Myostatin(ng/ml)	7.80(6.23-8.80)	4.10(3.30-4.75)	0.000

The collected data was analyzed by the mean ± standard deviation and median (25thand 75th percentiles) via the man Whitney test at the p<0.05 level, there was a significant difference between the two independent means.

The capacity of serum myostatin levels to distinguish patients with metabolic syndrome from healthy individuals was evaluated using the ROC curve analysis (Table 4; Fig.1). High sensitivity (100.0) and specificity (85.0) of the ROC curve over the metabolic syndrome diagnostic test indicated improved validity. The AUC of the ROC curve for the presence of a metabolic syndrome diagnosis revealed that the best level of accurate metabolic syndrome prediction was 0.978 ($p<0.001$).

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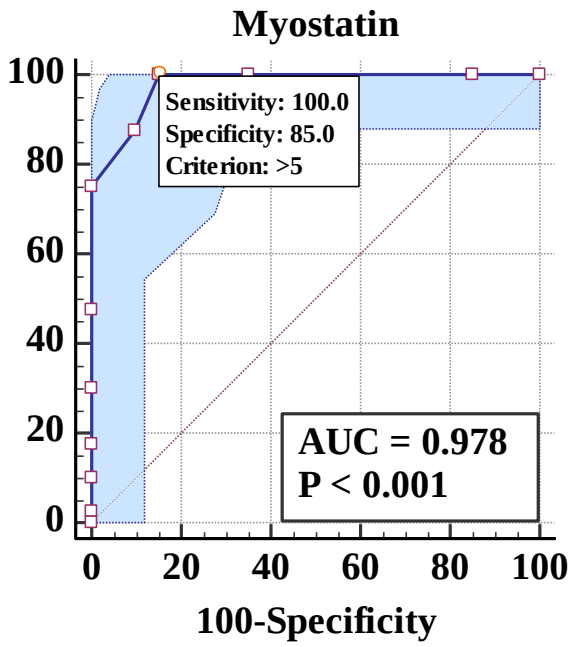

Figure 1. The ROC curve analysis of serum Myostatin concentration in metabolic syndrome patients (n =40) against healthy subjects (n = 40) (AUC is 0.978), p 0.001

Table 4. Myostatin AUC and validity in distinguishing between metabolic syndrome and healthy subjects.

Variable	AUC	P-Value	cutoff value	Sensitivity	Specificity	Accuracy	PPV	NPV
Myostatin	0.978	0.001	5	100.0	85.0	0.8500	87.0	**100.0**

The term AUC refers to the area under the curve. The terms negative predictive value (NPV) and positive predictive value (PPV) are used interchangeably.

In this study, patients with metabolic syndrome had their serum levels of myostatin evaluated. According to the study's findings, serum Myostatin levels were higher in metabolic syndrome patients than in healthy people. According to an earlier study, in individuals with severe obesity, myostatin levels begin to grow during adolescence. A future study is required to clarify the causal relationship between these associations. This idea may be crucial given that myostatin may play an important role in the development of sarcopenic obesity [25]. While these findings contradict those of a prior study. The researchers discovered that myostatin blood levels are markedly lower in patients who have metabolic syndrome and hypothesize that higher serum myostatin is linked to favorable metabolic profiles [26,27]. It is thought that these disorders do not randomly cluster, which accounts for certain people's co-existence of cardiovascular disease and diseases including diabetes, dyslipidemia, and hypertension. Insulin resistance has long been believed to be a significant factor in the onset of numerous metabolic disorders. When compared to non-obese women, obese women had higher levels of muscle, circulating myostatin protein, and myostatin secretion from myotubes made from myoblasts isolated from muscle biopsies [28]. Myostatin expression is found to be elevated in atrophic conditions in humans, such as extended bed rest in young men, chronic disuse atrophy in older patients, age-related muscle wasting (sarcopenia), and hypoxemic patients with severe chronic obstructive pulmonary disease [29,30] . A previous study indicated a significant association between myostatin and impaired glucose metabolism [31]. By controlling blood glucose levels, myostatin is essential for maintaining brain function and, therefore, a high nutritional capacity [32].

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Conclusion

Serum myostatin levels raised with metabolic syndrome in female Iraqi population. The metabolic syndrome can be diagnosed most accurately and sensitively with myostatin estimation. Higher serum myostatin was found to be independently linked with metabolic syndrome, hyperglycemia, poor HDL-C, high triglycerides, and BMI. Therefore, the increase in myostatin levels is associated with metabolic profile.

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Authors' Declaration

- Conflicts of Interest: None.
- We hereby confirm that all the Figures and Tables in the manuscript are ours. Furthermore, any Figures and images, that are not ours, have been included with the necessary permission for re-publication, which is attached to the manuscript.
- Ethical Clearance: The project was approved by the local ethical committee in University of Mustansiriyah.

Authors' Contribution Statement

R.H.A.'s role in this research was collecting samples and, analyzing the results. L.O.F. designed, analyzed, proofread, and presented ideas of the research.

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دراسة الميوستاتين كمؤشر تشخيصي في امصال النساء العراقيات المصابات بمتلازمة التمثيل الغذائي

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الخلاصة

السمنة في منطقة البطن، ومقاومة الأنسولين ، وارتفاع السكر في الدم ، وعسر شحميات الدم ، وارتفاع ضغط الدم كلها جزء من متلازمة التمثيل الغذائي. تقوم الخلايا العضلية بتصنيع وإطلاق الميوكين المعروف باسم myostatin، والذي يعمل على خلايا العضلات لمنعها من النمو. كان الهدف من هذه الدراسة هو مقارنة مستويات الميوستاتين لدى الأشخاص الذين يعانون من متلازمة التمثيل الغذائي والذين لا يعانون منها. صممت الدراسة الحالية لمقارنة بعض المؤشرات الحيوية في امصال مرضى متلازمة الأيض والأشخاص الأصحاء من خلال تقدير الميوستاتين، نسبة السكر في الدم لدى الصائمين (FBS) ، الكوليسترول ، البروتين الدهني عالي الكثافة (HDL) ، الدهون الثلاثية (T.G) ، البروتين الشحمي منخفض الكثافة (LDL) .، البروتين الدهني منخفض الكثافة (VLDL)، في امصال المرضى العراقيين المصابين بمتلازمة التمثيل الغذائي والأشخاص الأصحاء. تم تقسيم ما مجموعة ثمانين امرأة إلى مجموعتين: المجموعة الأولى 40 مريض متلازمة ايضية والمجموعة الثانية 40 شخصًا سليما . أظهرت النتائج زيادة معنوية في مستويات الميوستاتين $p> 0.001$ في مجموعة متلازمة التمثيل الغذائي مقارنة بالمجموعة الضابطة. أظهر منحنى ROCلليموستاتين قيمة فاصلة واضحة (5) عند حسابه في متلازمة التمثيل الغذائي. وخلصت الدراسة إلى أن الميوستاتين هو عامل تشخيصي للميوستاتين.

الكلمات المفتاحية: كوليسترول، كلوكوزالدم الصائم ، ميوستاتين ، متلازمة التمثيل الغذائي, ، بروتين دهني منخفض الكثافة.