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Evaluation of BMI, Waist Circumference and Blood Pressure in Metabolic Syndrome

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ABSTRACT

Introduction: Metabolic syndrome is a strong predictor of type 2 diabetes, with an increased incidence rate of 5 to 7-fold. The risk of developing CVD is approximately doubled in the metabolic syndrome. Obesity, in particular visceral adiposity, is known to be associated with insulin resistance and a heterogeneous disorder, MS. MS is a cluster of interrelated common clinical disorders, including hypertension, insulin resistance, glucose intolerance and dyslipidemia, in addition to obesity. **Aims and Objectives:** A study of correlation of Adiponectin levels in metabolic syndromes. Correspondence of adiponectin levels and various components of metabolic syndrome such as Blood pressure, BMI (Basal metabolic index) and Waist circumference. **Methods and material:** This observational cross-sectional hospital-based study was performed in Department of Biochemistry, Index Medical College, Hospital and Research Centre Indore MP, India. Study population: 300 patients attending in Department of Biochemistry, Index Medical College, Hospital and Research Centre Indore MP, India with metabolic syndrome during the study period. **Study Design:** Cross-sectional study. Department of Biochemistry, Index Medical College, Hospital and Research Centre Indore MP, India. Result: Systolic and diastolic blood pressure was significantly higher in Metabolic Syndrome group in compare to without metabolic Syndrome; but Pulse rate was significantly insignificantly distributed in both groups. **Conclusion:** Adiponectin level was significantly decreased as the number of metabolic syndrome components increases. The serum adiponectin concentration was significantly negative correlated with the SBP, BMI, Total Cholesterol, and LDL.

Keywords: Adiponectin, metabolic syndrome, type 2 diabetes, BMI, Blood pressure and Waist circumference.

Introduction

Metabolic syndrome is a strong predictor of type 2 diabetes, with an increased incidence rate of 5 to 7-fold. The risk of developing CVD is approximately doubled in the metabolic syndrome.1 Obesity, in particular visceral adiposity, is known to be associated with insulin resistance and a heterogeneous disorder, MS. MS is a cluster of interrelated common clinical disorders, including hypertension, insulin resistance, glucose intolerance and dyslipidemia, in addition to obesity.2 It has been shown that visceral fat deposits are more metabolically active than their subcutaneous homologues, being particularly involved in the development of diseases associated with obesity, such as MS, type 2 diabetes mellitus (T2DM) and coronary artery disease (CAD).3 adiponectin levels in plasma are inversely correlated with visceral adiposity. Lower levels of adiponectin were observed in patients with high blood pressure, hyperglycemia, low HDL-C, and hypertriglyceridemia, also in obese patients with MS.4 Adiponectin increases the sensitivity to insulin through several mechanisms. AdipoR1 and AdipoR2 are transmembrane receptors, whose carboxyl terminal group (C-terminal) is located outside the membrane, and the amino terminal group (N-terminal) inside.5 When adiponectin attaches to its receptor it activates adenosine mono phosphate (AMP) kinase,6 promoting so glucose uptake by muscles via intracellular translocation of the GLUT4 transporters. Simultaneously, it hampers gluconeogenesis by inhibiting the hepatic enzyme phosphoenolpyruvate carboxylase, inhibits the synthesis of fatty acids and stimulates their oxidation.7

Serum adiponectin is inversely related to body fat mass and to the degree of insulin resistance. Its concentration is particularly low in adults with T2DM or CAD. So, it is accepted, that adiponectin ameliorates sensitivity to insulin and contributes to cardiovascular protection.[8] Low circulating levels, particularly of the high molecular weight (HMW) component,[9] are also a strong risk marker for the development of MS. The most prevalent abnormality among subjects with the MetS was obesity, particularly abdominal obesity. Abdominal obesity is considered the predominant underlying cause of MetS and is associated with both insulin resistance and low-grade chronic inflammation.[10] Waist circumference is a widely used index of abdominal obesity, and was found to be a better predictor of MetS compared with BMI, waist-to-hip ratio and waist-to-height ratio.[11]

Material and Method

Population/Patients

This observational cross-sectional hospital-based study was performed in Department of Biochemistry, Index Medical College, Hospital and Research Centre Indore MP, India.

Study population: 300 patients attending in Department of Biochemistry, Index Medical College, Hospital and Research Centre Indore MP, India with metabolic syndrome during the study period.

Study Design: Cross-sectional study

Study Location: Department of Biochemistry, Index Medical College, Hospital and Research Centre Indore MP, India.

Sample Size: 300 patients (OPD and IPD Medicine)

Inclusion criteria

- Both the sexes consulting in OPD or IPD Index Medical College, Hospital and Research Centre Indore MP, India.
- Study will be conducted on -150 Metabolic syndrome patients (75 female and 75 male) their ages ranged between (44-60 years) from General medicine department in Index Medical College, Hospital and Research Centre Indore MP, India.
- 150 healthy persons (75 female and 75 male) their ages ranged between (40-60 years).
- Visceral obesity, defined as waist circumference ≥ 102 cm in men and ≥ 88 cm in women.
- Systolic blood pressure (SBP) ≥130 mmhg and/or diastolic blood pressure (DBP) ≥85 mmhg, or patient on antihypertensive treatment.

Exclusion Criteria

- Chronic liver disease
- Chronic renal failure
- Patients on corticosteroid therapy
- Autoimmune disease
- Malignancy
- Pregnancy

Study tool

- Case reporting form
- Consent form

Procedure methodology

After written informed consent form had been obtained, detailed history of the presenting symptoms and their onset was recorded. Detailed histories of all the patients were obtained (like demographic details, age of patient, clinical details), blood pressure, BMI, waist circumference was noted on patient's proforma.

BMI

Body mass index (BMI) is a value derived from the mass (weight) and height of a person. The BMI is defined as the body mass divided by the square of the body height, and is expressed in units of kg/m2, resulting from mass in kilograms (kg) and height in metres (m).

BMI will classify you as 'underweight', 'healthy weight', 'overweight' or 'obese', as defined by the World Health Organization.

If your BMI is:

- under 18.5kg/m2 – you are considered underweight and possibly malnourished
- 18.5 to 24.9kg/m2 – you are within a healthy weight range for young and middle-aged adults
- 25.0 to 29.9kg/m2 – you are considered overweight

- over 30kg/m2 – you are considered obese.

Waist Circumference (WC)

To measure WC, patients should stand with their arms crossed on the contralateral shoulders. The placement of the measuring tape should be snugly around the lateral aspect of each ilium at the mid-axillary line. It is an essential measure of anthropometry in adults and children as it directly measures central adiposity. Increasing central adiposity is associated with an increased risk of morbidity and mortality due to an increased risk of diabetes and heart disease.

Blood Pressure

Measurement of blood pressure was done by mercury sphygmomanometer in right arm in sitting and supine position, the care taken during measurement that the patient to sit quietly for 5 min before measuring blood pressure, the patient should be seated comfortably with the back supported and the upper arm bared without constrictive clothing, the legs should not be crossed, the arm should be supported at heart level, and the bladder of the cuff should encircle at least 80% of the arm circumference, larger or smaller cuffs were used as needed. The mercury column would be deflated at 2 to 3 mm/sec, and the first and last audible sounds would be taken as systolic and diastolic pressure.

Statistical Analysis:

The data were analysed using the Statistical Package for the Social Sciences (SPSS) version 23.0. Categorical variables are reported as frequencies and percentages and continuous variables as the mean ± SD. Categorical variables were compared using Chi Square/Fisher's exact test. Continuous variables were compared using independent samples *t*-test. P values less than 0.05 ($p<0.05$) was considered statistically significant.

Result

Weight, BMI, Waist circumferences and Waist hip circumferences ratio were significantly higher in Metabolic Syndrome group in compare to without metabolic Syndrome; but Height and Hip circumferences was significantly insignificantly distributed in both groups.

Table no. 2: Body mass Index and waist hip ratio examination

	Metabolic Syndrome (n=150)	**Without metabolic Syndrome (n=150)**	**P value**
Height (cm)	159.70±10.48	160.53±9.21	0.745
Weight (kg)	72.23±10.88	63.97±10.48	**0.004**
BMI (kg/m^2)	28.22±2.35	24.87±4.15	**<0.001**
Waist circumferences (cm)	88.53±13.01	82.23±8.07	**0.028**
Hip circumferences (cm)	88.30±8.47	87.70±8.55	0.786
Waist hip circumferences ratio	1.01±0.18	0.94±0.05	**0.044**

Systolic and diastolic blood pressure was significantly higher in Metabolic Syndrome group in compare to without metabolic Syndrome; but Pulse rate was significantly insignificantly distributed in both groups.

Table no. 3: Hemodynamic examination

	Metabolic Syndrome (n=150)	**Without metabolic Syndrome (n=150)**	**P value**
SBP (mmHg)	137.50±17.80	119.97±14.48	**<0.001**
DBP (mmHg)	85.77±6.13	77.50±8.58	**<0.001**
Pulse rate (beets per min)	67.23±8.76	66.30±9.99	0.702

Discussion

After written informed consent form had been obtained, detailed history of the presenting symptoms and their onset was recorded. Detailed histories of all the patients were obtained (like demographic details, age of patient, clinical details), blood pressure, heart rate, Lipid profile BMI, Blood sugar level (fasting and prandial) was noted on patient's proforma.

The following risk factors and criteria were used: central obesity (waist circumference; men ≥ 90 cm, women ≥ 80 cm) plus any two of the following: (1) raised triglycerides (> 1.7 mmol/L (150 mg/dL) or specific treatment for this lipid abnormality); (2) reduced HDL-cholesterol (men <1.03 mmol/L (40 mg/dL) or women <1.29 mmol/L (50 mg/dL) or specific treatment for this lipid abnormality); (3) raised blood pressure (≥ 130/85 mm Hg or treatment of previously diagnosed hypertension); (4) raised fasting plasma glucose (≥ 5.6 mmol/L (100 mg/dL) or previously diagnosed type 2

diabetes). Peripheral venous blood samples were collected after an overnight fasting. Metabolic syndrome biomarkers such as fasting blood glucose, triglycerides and high-density lipoprotein cholesterol were assessed with commercially available reagents (Randox, UK) by using Vitalab Selectra E chemistry analyzer (Vitalab, Netherlands). Serum adiponectin concentration was determined by using the Human Adiponectin Enzyme-Linked Immunosorbent Assay (ELISA) kit (Chemicon, USA).

Shashank R. Tiwari et al[12]**, Nur Firdaus Isa et al**[13]**, Ming-Chun Chen et al**[14]**,** and **Y. Premchandra singh et al**[15] also used the similar procedures in their perspective study. On average, subjects without Metabolic Syndrome (MetS) were younger, had a smaller body mass index and waist circumference than those with MetS patients. They also had a lower BP, pulse rate, and fasting plasma glucose than the subjects with MetS, as well as significant differences in lipid profile in with MetS patients. The mean serum adiponectin concentration was higher in people without MetS 15.79±2.90 mg/ml than those with MetS 11.02±2.63 mg/ml (P<0.001).

We observed that statistically significant lower adiponectin levels were associated with most features of metabolic syndrome. Adiponectin level was also significantly decreased as the number of metabolic syndrome components increases. According to our multivariate analysis results, the serum adiponectin concentration was significantly negative correlated with the SBP (r= -0.262; p<0.05), BMI (r= -0.288; p<0.05), Total Cholesterol (r= -0.515; P<0.001), and LDL (r= -0.305; p<0.05) respectively. The receiver operating curve (ROC) which shows that adiponectin level was less than 12.45 µg/ml can be used as cut off to predict the occurrence of metabolic syndrome in patients with sensitivity 90.0%, specificity 63.3% and with accuracy was 87.6%.

In present study the mean level of adiponectin with corresponding BMI in the normal subjects of this ethnic population is (11.02±2.63 vs 15.79±2.90 µg/ml) other published reports of adiponectin level in European (9.85 ± 2.33 vs 10.89 ±0.86 µg/ml) or even from the mainland India (9.85 ± 2.33 Vs 16.7 ±7.6 µg/ml).[16] However the mean value of adiponectin and mean BMI is almost similar with those reported from Chinese (9.85 ± 2.33 vs 8.52±0.57 µg/ml) and South Asian population (9.85 ± 2.33 vs 8.26 ±0.45 µg/ml).[17]

The present was found the mean baseline adiponectin level was lower in the diabetic subjects than in the non-diabetic subjects (11.19±3.09 vs. 15.21±3.06 µg/ml; P <0.001). **Snehalatha C et al**[18] also reported the adiponectin level was lower in the diabetic subjects than in the non-diabetic subjects (11.3 ±5.5 vs. 16.7 ±7.6 µg/ml; P = 0.0017). **Y. Premchandra singh et al**[15] reported the adiponectin level was lower in the diabetic subjects than in the non-diabetic subjects (6.07 ± 1.02 vs. 7.48 ± 1.91 µg/ml; P = 0.003). The present study agrees previous findings that type II diabetes and metabolic syndrome were associated with low serum adiponectin concentrations. Low adiponectin level was a strong predictor of future development of diabetes, also showed a positive predictive association. **Nur Firdaus Isa et al**[13] reported the no significant difference of the adiponectin level between hyperglycemic and non-hyperglycemic in their studied subjects. Increasing the sample size and expanding their cross-sectional study to a cohort study with longer follow-up may fill in the gaps.

The adiponectin concentration found in our study patients with hypertension was (12.12±3.57 µg/ml) lower than those without hypertension was (14.09±3.55 µg/ml) and association was statically significant. **Masato Furuhashi et al**[19] reported the adiponectin concentrations were reduced in insulin-resistant essential hypertensives but not normotensives or non–insulin-resistant hypertensives, suggesting that hypoadiponectinemia in essential hypertensives is associated with insulin resistance. **Renaldi O et al**[20] stated the hypoadiponectinemia and insulin resistance represent independent risk factors for metabolic syndrome development.

In this study, the relationship of adiponectin with waist circumference was in male (<90 cm-13.74±3.34 µg/ml vs ≥90cm-11.18±2.38 µg/ml; P=0.034) and association was significant but in female (<80 cm-12.96±3.73 µg/ml vs ≥80cm-15.37±3.83 µg/ml; P=0.094) and association was insignificant. While **Y. Premchandra singh et al**[15] reported the relationship of adiponectin with waist circumference appeared to be stronger than other obesity indices or BMI, indicating that central fat distribution (visceral obesity) is a better determinant of circulating adiponectin than total fat mass. Waist circumference in female was (>80 cm- 5.98±1.18 µg/ml vs 9.9±2.7 µg/ml; P<0.001) and Waist circumference in male was (> 90 cm 5.81±4.10 µg/ml vs 7.90±0.05 µg/ml; P<0.001).

In our study the serum adiponectin concentration was significantly negative correlated with the SBP (r= -0.262; p<0.05), BMI (r= -0.288; p<0.05), Total Cholesterol (r= -0.515; P<0.001), and LDL (r= -0.305; p<0.05) respectively. **Blaslov K et al**[21] reported the patients with higher adiponectin level (n = 39) had significantly lower waist circumference (P< 0.002), fasting venous glucose levels (P< 0.001), higher HDL3-cholesterol (P = 0.011), and eGDR (P = 0.003) in comparison to the group with lower adiponectin who showed higher prevalence of MS (P = 0.045). eGDR increased for 1.09mg/kg−1 min−1 by each increase of 1 μg/mL total fasting plasma adiponectin (P = 0.003). In the logistic regression model, adiponectin was inversely associated with the presence of MS (P = 0.014). **Taniguchi A et al**[22] reported the serum adiponectin level was negatively correlated to BMI (r = −0.308, P = .002), diastolic blood pressure (r = −0.269, P = .012), and triglycerides (r = −0.338, P< .001), and positively correlated to high-density lipoprotein cholesterol (r = 0.300, P = .003) in their patients. **Chen MC et al**[14] reported the serum Adiponectin was inversely associated with Metabolic Syndrome

Conclusion

- Subjects without Metabolic Syndrome were younger, had a smaller body mass index and waist circumference than those with Metabolic Syndrome patients.
- A lower BP, pulse rate, and fasting plasma glucose than the subjects with Metabolic Syndrome, as well as significant differences in lipid profile in with Metabolic Syndrome patients.

- Statistically significant lower adiponectin levels were associated with most features of Metabolic Syndrome.
- Adiponectin level was significantly decreased as the number of metabolic syndrome components increases.
- The serum adiponectin concentration was significantly negative correlated with the SBP, BMI, Total Cholesterol, and LDL.

References

1. Grundy SM: Metabolic syndrome pandemic. ArteriosclerThrombVasc Biol. 2008;28:629-36.
2. Moller DM, KAufman KD. Metabolic syndrome: a clinical and molecular perspective. Annual Review of Medicine. 2005;56:45– 62.
3. Hamdy O, Porramatikul S, Al-Ozairi E. Metabolic obesity: the paradox between visceral and subcutaneous fat. Curr Diabetes Rev. 2006;2:367–73.
4. Matsuzawa Y, Funahashi T, Kihara S, Shimomura I. Adiponectin and metabolic syndrome. ArteriosclerThrombVasc Biol. 2004;24:29–33.
5. Lima Marcos M, Rosa Francisco J, Marín A, Romero-Vecchione E. Adiponectina y susefectospleiotrópicos en el sistema cardiovascular. Rev VenezEndocrinolMetab 2009;7:3–9.
6. Yamauchi T, Kadowaki T. Physiological and pathophysiological roles of adiponectin and adiponectin receptors in the integrated regulation of metabolic and cardiovascular diseases. Int J Obes (Lond). 2008;32(Suppl 7):S13–S18.
7. Silva TE, Colombo G, Schiavon LL: Adiponectin: A multitasking player in the field of liver diseases. Diabetes Metab. 2014;40:95–107.
8. Kadowaki T, Yamauchi T, Kubota N, Hara K, Ueki K, Tobe K. Adiponectin and adiponectin receptors in insulin resistance, diabetes, and the metabolic syndrome. J Clin Invest. 2006;116:1784–92.
9. Kaser S, Tatarczyk T, Stadlmayr A, Ciardi C, Ress C, Tschoner A, et al. Effect of obesity and insulin sensitivity on adiponectin isoform distribution. Eur J Clin Invest. 2008;38:827–34.
10. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, Gordon DJ, Krauss RM, Savage PJ, Smith SC, et al. Association/National Heart, Lung, and Blood Institute Scientific Statement Diagnosis and Management of the Metabolic Syndrome: An American Heart Association/ National Heart, Lung, and Blood Institute Scientific Statement. Circulation.2005;112:2735–52.
11. Bener A, Yousafzai MT, Darwish S, Al-Hamaq A, Nasralla EA, Abdul-Ghani M. Obesity index that better predict metabolic syndrome: body mass index, waist circumference, waist hip ratio, or waist height ratio. J Obes.2013;269038:9.
12. Shashank R Tiwari, Lokesh S, Arunprasath P, Arun Kumar R. A comparative study of serum adiponectin levels in patients of acute coronary syndrome with and without central obesity. Int J Adv Med. 2019 Feb;6(1):xxx-xxx
13. Nur Firdaus Isa, Laila Ruwaida Mohd Zainuddin, Wan Manan Wan Muda, Hamid Jan B. Jan Mohamed. Association of serum adiponectin levels with metabolic syndrome risk factors in Malay adults. World Nutrition Journal July 2017; vol.1 No. 1:17-22.
14. Ming-Chun Chen, Chung-Jen Lee, Chiu-Fen Yang, Yu-Chih Chen, Ji-Hung Wang and Bang-Gee Hsu. Low serum adiponectin level is associated with metabolic syndrome and is an independent marker of peripheral arterial stiffness in hypertensive patients. Diabetol Metab Syndr 2017; 9:49
15. Premchandra singh Y, Tilakeswari Devi L, Joshita L, Romola Devi Th, Ranabir S, Amuba M, et al. Plasma Adiponectin Is An Independent Predictor Of Metabolic Syndrome. IOSR Journal of Dental and Medical Sciences (JDMS). Nov- Dec. 2012; Volume 2, Issue 4: PP 34-43.
16. Pischon T, Girman CJ, Hotamisligil GS, Rifai N, Hu FB & Rimm EB. Plasma adiponectin levels and risk of myocardial infarction in men. JAMA 2004;291: 1730–37.
17. Andrew M, Fahad R, Beng, Stefan SB. Vuksan V, Darlene D , Ruby M, Koon T, Hertzel G, Arya M, Salim Y,Sonia .Ethnic
18. Variation in Adiponectin and Leptin Levels and Their Association with Adiposity and Insulin Resistance. Diabetes Care 2010; 33:1629–34.
19. Snehalatha C, Mukesh B, Simon M, Viswanathan V, Haffner SM, Ramachandran A. Plasma adiponectin is an independent predictor of type 2 diabetes in Asian indians. Diabetes Care 2003 Dec; 26(12):3226-9.
20. Masato Furuhashi, Nobuyuki Ura, Katsuhiro Higashiura, Hideyuki Murakami, Marenao Tanaka, Norihito Moniwa, Daisuke Yoshida, Kazuaki Shimamoto. Blockade of the Renin-Angiotensin System Increases Adiponectin Concentrations in Patients with Essential Hypertension. Hypertension. 2003; 42:76-81.
21. Renaldi O, Pramono B, Sinorita H, Purnomo LB, Asdie RH, Ahmad H Asdie; Hypoadiponectinemia: A Risk Factor for Metabolic Syndrome; 2009; Vol.41.
22. Blaslov K, Tomislav Bulum, Karin Zibar, and Lea Duvnjak; Relationship between Adiponectin Level, Insulin Sensitivity, and Metabolic Syndrome in Type 1 Diabetic Patients; Received 31 May 2013; Accepted 1 July 2013.

23. Taniguchi,A Fukushima M, Ohya M, Nakai Y, Yoshii S, Nagasaka S. Interleukin, adiponectin, leptin, and insulin resistance in nonobese Japanese type 2 diabetic patients. Metabolism clinical and experimental.2006; 55(2):258-62.