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ORIGINAL RESEARCH OPEN ACCESS

Correlation between serum leptin levels and metabolic syndrome parameters

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Abstract

Background: Metabolic Syndrome (MetS) is a complex clinical condition characterized by central obesity, dyslipidemia, hypertension, and insulin resistance (IR). Adipose tissue functions as an active endocrine organ secreting adipocytokines, including leptin, which play a crucial role in metabolic, endocrine and immune regulation. Altered leptin signalling contribute to the pathogenesis of obesity-related complications. Lower circulating leptin levels are associated with improved insulin sensitivity, lipid metabolism, reduced adiposity and decreased inflammation. The present study aimed to estimate serum leptin levels in individuals with and without MetS and to evaluate its correlation with various metabolic syndrome parameters.

Materials and methods: A cross-sectional comparative study was conducted in the Department of Biochemistry in collaboration with the Department of Medicine, Regional Institute of Medical Sciences (RIMS), Imphal, Manipur, India, from January 2021 to October 2022. Fifty patients diagnosed with MetS and fifty age-matched controls without MetS were included. Serum leptin levels and components of MetS were evaluated. Statistical analysis was performed using the Independent Sample t-test and Pearson's correlation coefficient.

Results: The mean age of cases and controls was 61.10 ± 12.52 years and 57.08 ± 10.67 years, respectively. Serum leptin levels were significantly elevated in MetS cases (26.58 ± 14.6 ng/mL) compared to controls (12.99 ± 8.59 ng/mL). Leptin showed a positive correlation with triglycerides, body mass index (BMI), fasting blood glucose and blood pressure, whereas a negative correlation was observed with high-density lipoproteins (HDL).

Conclusion: Elevated serum leptin levels are significantly associated with an increased risk of metabolic syndrome. Leptin may serve as a promising biomarker for early identification and risk stratification of MetS.

Keywords: metabolic syndrome; leptin; adipocytokines; triglycerides; blood pressure; high-density lipoproteins; dyslipidemia; insulin resistance; inflammation

Introduction

Metabolic syndrome represents a constellation of disorders that heighten the risk of atherosclerotic cardiovascular disease, including myocardial infarction, cerebrovascular accidents, peripheral vascular disease, insulin resistance and type II diabetes mellitus. It is defined by the presence of central obesity, insulin resistance, hypertension and an atherogenic dyslipidemia profile [1]. Insulin resistance, adipose tissue dysfunction, chronic low-grade inflammation, circadian rhythm disruption, oxidative stress, and genetic predisposition are important contributing factors in the development of MetS [2]. According to the International Diabetes Federation (IDF), MetS is diagnosed in the presence of central obesity defined by waist circumference (WC) ≥ 90 cm for men and ≥ 80 cm for women, or ethnicity-specific values assumed if body

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mass index (BMI) is > 30 kg/m^2, in addition to any two of the following criteria: (1) raised triglycerides (TG) ≥150 mg/dL or treatment for this lipid abnormality, (2) reduced high-density lipoprotein (HDL) cholesterol < 40 mg/dL in males and < 50 mg/dL in females or specific treatment, (3) blood pressure (BP) ≥ 130/85 mmHg or treatment of known hypertension, and (4) fasting plasma glucose ≥ 100 mg/dL or previously diagnosed type 2 diabetes mellitus (T2DM) [3]. Hyperuricemia is currently being evaluated for inclusion within the MetS diagnostic criteria, as its incorporation may improve early identification of individuals at metabolic risk and help prevent long-term adverse outcomes [4]. The prevalence of metabolic syndrome globally is estimated to be around 25% with variations due to genetic, environmental and lifestyle factors [5]. The prevalence of metabolic syndrome in India is estimated to be 30% and is significantly higher among women (35%) than men (26%) [6]. In the northeastern states of India, the prevalence of metabolic syndrome is reportedly lower, at around 1%-2% [7].

Leptin is a 16-kDa monomeric non-glycosylated protein predominantly secreted by adipocytes, and its circulating concentration is directly proportional to total body fat mass. Leptin levels increase with excessive caloric intake and obesity, and decrease during fasting or weight reduction. Initially identified as a satiety hormone regulating body weight through appetite suppression and enhanced energy expenditure via hypothalamic pathways, leptin is now known to exert major roles in glucose and lipid metabolism, immune modulation, and neuroendocrine regulation [8]. Dysregulation of leptin contributes to the development of obesity, insulin resistance, type 2 diabetes mellitus, and cardiovascular disorders [9]. Considering the growing prevalence of non-communicable lifestyle diseases and their increasing healthcare burden, this study was undertaken to investigate the association between serum leptin levels and components of metabolic syndrome.

The objectives of the study were to estimate serum leptin levels in individuals with and without metabolic syndrome and to evaluate the correlation between serum leptin levels and various metabolic syndrome parameters.

Materials and methods

A cross-sectional study was conducted in the Department of Biochemistry in collaboration with the Department of Medicine, Regional Institute of Medical Sciences (RIMS), Imphal, Manipur, India, from January 2021 to October 2022. The study received approval from the Research Ethics Board, RIMS, Imphal (Ref. No. A/206/REB-Comm(SP)/RIMS/2015/680/22/2020). Informed written consent was obtained from all study participants prior to enrollment.

The study included 50 diagnosed cases of metabolic syndrome attending the Emergency Department, Medicine Outpatient Department (OPD), and inpatient wards of RIMS, Imphal, along with 50 apparently healthy individuals without MetS who served as controls. Anthropometric measurements, including height, weight, skinfold thickness (SFT), waist circumference (WC), and body mass index (BMI) were recorded for all participants. Individuals aged 30 years and above, irrespective of sex, caste or ethnicity, and those with a confirmed diagnosis of metabolic syndrome who voluntarily provided written consent were included. Apparently healthy individuals without features of MetS were recruited as controls. Participants with type 1 diabetes mellitus (T1DM), chronic kidney disease, cardiovascular diseases such as rheumatic heart disease, liver disorders including primary dyslipidemia, hepatitis B and C, alcoholic liver disease or carcinoma, immunodeficiency conditions, malignancies, renal failure, connective tissue disorders, liver cirrhosis, congestive heart failure, pregnancy, or those undergoing steroid therapy or antiretroviral therapy (ART) were excluded.

Eligible participants were identified based on the International Diabetes Federation criteria for MetS. Approximately 5 mL of venous blood was collected after an overnight fast of 8–12 hours using aseptic precautions. About 2 mL of blood was transferred into a fluoride vial for estimation of fasting blood glucose, while the remaining sample was collected in a plain vial. The plain sample was centrifuged at 3000 rpm for 10 minutes to obtain serum, which was used to estimate leptin, triglycerides and HDL cholesterol. Serum leptin levels were measured by enzyme-linked immunosorbent assay (ELISA) using the DBC-Diagnostics Biochem Canada Inc. Leptin Elisa kit [10]. Fasting blood glucose was estimated using the Randox Glucose kit (Cat. No. GL 3815, United Kingdom) by the GOD/PAP method (Glucose oxidase–phenol 4-aminoantipyrine) [11]. Serum triglycerides were determined by an enzymatic colorimetric test using lipid-clearing factor kits supplied by Human Gesellschaft für Biochemica und Diagnostica mbH [12]. Serum HDL cholesterol was measured by an enzymatic colorimetric method using Human Gesellschaft für Biochemica und Diagnostica mbH kits and with the Randox Rx Imola automated analyzer [13].

Statistical analysis

All data were statistically analyzed using IBM SPSS version 21 for Windows. Comparison of serum leptin levels between MetS cases and controls was performed

using the Independent Sample t-test. Pearson's correlation coefficient was applied to determine the correlation between serum leptin and metabolic syndrome parameters. A p-value of <0.05 was considered statistically significant.

Results

The mean age of the cases was 61.10 ± 12.52 years, with 46% males and 54% females. As shown in Table 1, the mean (±SD) values of weight, BMI, waist circumference (WC), pulse rate, systolic and diastolic blood pressure and skinfold thickness (SFT) differed significantly between cases and controls. However, there was no statistically significant difference observed in the mean (±SD) values of age and height between the two groups.

Table 1: Distribution of participants according to clinical and metabolic parameters among controls and cases.

Parameter	*Control (N=50) Mean ±SD*	*Cases (N=50) Mean ±SD*	*P value*
Age (years)	57.08 ± 10.67	61.10 ± 12.52	0.08
Height (cm)	161.33 ±6.69	159.65 ± 6.18	0.20
Weight (kg)	53.21 ± 5.40	74.21 ± 8.66	0.00
BMI (kg/m^2)	20.42 ± 1.82	29.14 ± 3.20	0.00
WC (cm)	75.23 ± 4.90	91.81 ± 5.0	0.00
Pulse (per min)	75.58 ± 5.68	79.312 ± 7.52	0.00
BP-Systolic (mmHg)	112.83 ± 9.19	138.65 ± 11.99	0.00
BP-Diastolic (mmHg)	75.00 ± 5.04	89.63 ± 8.74	0.00
SFT (mm)	5.27 ± 0.94	8.69 ± 2.41	0.00

In Table 2, the mean (±SD) levels of serum leptin, fasting blood glucose (FBG) and triglycerides (TG) were significantly higher in cases compared to controls, whereas the mean (±SD) level of high-density lipoprotein (HDL) cholesterol was significantly lower in cases than in controls.

Table 2: Distribution of participants according to metabolic parameters among controls and cases.

Variables	*Control (N=50) Mean ±SD*	*Cases (N=50) Mean ±SD*	*P value*
Leptin (ng/ml)	12.99 ± 8.59	26.58 ± 14.6	0.00
FBG (mg%)	94.33 ± 10.16	194.06 ± 66.2	0.00
HDL (mg%)	47.42 ± 4.91	26.54 ± 7.91	0.00
TG (mg%)	125.75 ± 35.07	198.56 ± 88.68	0.00
Hb (g/dl)	13.94 ± 11.98	11.66 ± 1.7	0.19

Table 3 demonstrates that serum leptin exhibits a statistically significant negative correlation with high-density lipoprotein (HDL), and a statistically significant positive correlation with fasting blood glucose (FBG), weight, BMI, waist circumference (WC), blood pressure, and skinfold thickness (SFT).

Table 4 shows that an increase in serum leptin levels above the study-specific cutoff value (> 16.05 ng/mL) was observed in 38 patients among the cases (76%), whereas only 23 individuals in the control group (46%) demonstrated elevated leptin levels.

Table 3: Pearson's correlation of leptin with parameters of metabolic syndrome.

Parameter	*Correlation coefficient*	*P value*
FBG (mg%)	0.290	0.00
TG (mg%)	0.154	0.13
HDL (mg%)	-0.269	0.00
Weight (kg)	0.410	0.00
BMI (kg/m^2)	0.451	0.00
WC (cm)	0.419	0.00
BP-Systolic (mmHg)	0.402	0.00
BP-Diastolic (mmHg)	0.375	0.00
SFT (mm)	0.481	0.00

Note: Correlation is significant at the 0.01 level (2-tailed)

Discussion

The mean (±SD) age of the participants in this study was 61.10 ± 12.52 years for cases only. An increased prevalence of MetS among older adults may be

explained by age-related factors such as physical frailty, malnutrition, compromised immunity, and chronic systemic illness [14]. No significant gender differences in the prevalence of MetS were observed, which is consistent with findings reported by Teli et al [15]. Blood pressure demonstrated a significant positive correlation with serum leptin levels, comparable to observations reported by Senarathne et al [16].

Table 4: Distribution of controls and cases according to serum leptin levels.

	Control (N=50)		*Cases (N=50)*		*p-value*
	No. of patients	*Percent*	*No. of patients*	*Percent*	*<0.05*
Increased > 16.05 ng/ml	23	46%	38	76%	
Normal < 16.05 ng/ml	27	54%	12	24%	

The mean (±SD) value of skinfold thickness (SFT) was significantly higher in cases than in controls. According to González-Torres S et al, skinfold measurements may be valuable for early detection of cardiometabolic risk even among sedentary and physically active adults [17]. Similarly, BMI and waist circumference (WC) showed significant differences between cases and controls. A study by Zhang et al [18] reported that the risk of metabolic syndrome was significantly associated with changes in BMI and WC in middle-aged and elderly populations, and that reductions in these indices were associated with lower metabolic risk.

In the present study, serum leptin levels were markedly higher among MetS patients compared to controls and showed strong positive correlations with BMI, weight, WC, SFT, fasting blood glucose (FBG), triglycerides (TG), and blood pressure. Similar findings have been reported in a comprehensive review and meta-analysis by Sheth ND et al [19]. Leptin regulates appetite and metabolic balance by inhibiting the synthesis and release of neuropeptide Y (NPY) in the arcuate nucleus and by binding to its transmembrane receptor (Lep-R) to stimulate anorexigenic pathways such as proopiomelanocortin (POMC). Activation of POMC generates α-melanocyte-stimulating hormone (α-MSH), which reduces body weight by activating melanocortin-3 and melanocortin-4 receptors (MC3R and MC4R), leading to neuronal depolarization of POMC neurons and suppression of NPY/gamma-aminobutyric acid pathways [20]. Reduced circulating leptin levels have been positively associated with improved insulin sensitivity, lipid metabolism, decreased adiposity, and reduced inflammation [9]. Leptin also induces insulin-sensitizing effects by promoting the oxidation of free fatty acids, whereas leptin resistance results from the induction of suppressor of cytokine signaling-3 (SOCS-3), which inhibits intracellular leptin signalling [21]. Leptin resistance, therefore, plays a crucial role in the pathogenesis of MetS, reinforcing the potential value of leptin as a biomarker for early detection and risk stratification.

Limitations: The primary limitation of this study is the relatively small sample size, which may restrict the generalizability of the findings. Additionally, leptin levels can be influenced by factors such as dietary habits, physical activity, and genetic background, which were not evaluated in this study and may warrant assessment in future research.

Conclusion

The findings of the present study demonstrate that serum leptin levels are significantly elevated in individuals with metabolic syndrome and exhibit strong positive correlations with key metabolic parameters, including BMI, waist circumference, fasting blood glucose, triglycerides and blood pressure, while showing a negative correlation with HDL cholesterol. These results highlight the crucial role of leptin in the pathophysiology of metabolic dysregulation, suggesting that leptin may serve as a promising biomarker for the early identification and risk stratification of metabolic syndrome. Early detection through leptin assessment may support timely interventions aimed at preventing long-term cardiometabolic complications. However, these findings should be validated in future studies with a larger sample size.

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Conflicts of interest

Authors declare no conflicts of interest.

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