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ASSOCIATION OF SERUM LEPTIN LEVELS WITH METABOLIC SYNDROME (MetS) AND ITS COMPONENTS IN ADULT POPULATION OF SAURASHTRA, GUJARAT.

Biochemistry

KEYWORDS: Metabolic Syndrome, Serum leptin, Obesity, Saurashtra region

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ABSTRACT

Background: Metabolic syndrome (MetS) is a common metabolic disorder resulting from the increasing prevalence of obesity; hyperleptinemia/leptin resistance is thought to be a cause of obesity. The present study is aimed to assess the association of serum leptin levels with MetS in the population of Saurashtra region, Gujarat.

Methods: In this cross-sectional observational study, 297 individuals aged between 20 to 80 years were included. The details of demographics, personal and family history of diseases, addiction/s etc. were collected from each participant. Blood samples were analyzed for lipid profile, fasting blood sugar and serum leptin levels.

Results: Serum leptin levels were found significantly high in the individuals with MetS and leptin level were positively correlated with BMI, WC, FBS, SBP and DBP. The ability of serum leptin levels to predict MetS was assessed by ROC curve; AUC were 0.737 and 0.769 in males and females respectively. Binary logistic regression analysis suggested the relative risk of developing MetS, independent of age, BMI and tobacco usage as OR 1.20 and 1.10 in males and females respectively.

Conclusion: Leptin level increases with increase in components of MetS and is found to be associated with MetS. Independent of age, BMI and tobacco usage, leptin levels are associated with abdominal obesity, high BP and high FBS which are known risk factors for cardiovascular diseases. Hence, leptin can be used as a biomarker for MetS and cardiovascular risk.

INTRODUCTION:

The obesity is a global health concern and has received a great deal of attention in past few decades. The metabolic syndrome (MetS) is a common metabolic disorder that results from the increasing prevalence of obesity[1] which is characterized by a clustering of metabolic abnormalities leading to increased cardiovascular diseases (CVD), causing increased morbidity and mortality.[2]

Leptin, a 16 KDa cytokine hormone is produced mainly by adipocytes in amount proportional to total adipose tissue mass.[3] The leptin level acts as sensor of the energy balance and communicates information on energy store to the hypothalamus by supporting food intake and stimulating energy expenditure.[4] Most overweight and obese individuals have an elevated level of leptin (hyperleptinemia), that are considered as leptin resistant in which appetite is not suppressed and/or there is failure to normalize adipose tissue mass. Leptin resistance is believed to be a fundamental pathology in obesity.[5]

A marked shift in lifestyle, affluence in urbanization, and oil and sugar rich dietary habits have been responsible for making ethnic Gujarati people prone to higher risk of CVD.[6] Hence in this ethnic population, potential relationship between serum leptin levels and development of MetS needs to be assessed towards early detection of CVD and prevention of subsequent morbidity and mortality. The present study is aimed to assess the association of serum leptin levels with MetS and access predictive value of serum leptin for diagnosis of MetS in population of Saurashtra region, Gujarat.

METHODS:

A cross sectional observational study was conducted on 297 individuals aged over 20 and less than or equal to 80 years, who willingly participated in the free health checkup camp, arranged by institute and fulfilled the inclusion criteria. Individuals with self-reported pregnancy state, with active liver or kidney disease, on treatment of hypo or hyper thyroidism and needing hospitalization were excluded. Among them, 155 individuals were males and 142 were females. The research project was approved by the Institutional Ethics Committee on Human Research. Signed informed consent was obtained from each participant. The details of demographics, personal and family history of diseases, addiction/s and medication in use were collected from each participant.

Laboratory Tests: Venous blood samples were collected in the morning after an overnight fasting. Serum separation was carried out by centrifugation and the serum specimens were immediately stored at -20°C until analyzed. Serum leptin levels were determined by enzyme-linked immunosorbent assay (Diagnostics Biochem Canada Inc., Canada). The other biochemical parameters were measured with fully automated biochemistry analyzer (Siemens Dimension R x L Max). Serum glucose was measured by hexokinase method. Serum total cholesterol (TC), serum triglycerides (TGs), serum LDL-cholesterol (LDL-C) and serum HDL-cholesterol (HDL-C) concentrations were measured by International Federation of Clinical Chemistry (IFCC) approved enzymatic methods using commercially available kits.

The body mass index (BMI) was calculated from the formula, BMI = weight in kg / height in meters^2. Hip circumference (HC) and waist circumference (WC) were measured using standard methods, in centimeters. Blood pressure (BP) was measured using a properly calibrated sphygmomanometer by standard method.

Identification of Metabolic Syndrome: MetS has been defined as the presence of ≥ 3 of the 5 risk factors established by the Joint Interim Statement (JIS) 2009 consensus guidelines for defining MetS with Asian Indians cutoffs for WC.[7] **Abdominal obesity:** (WC ≥90 cm in males and ≥80 cm in females), **Hypertriglyceridemia:** (TGs ≥150 mg/dl or Rx to lower TGs), **Low HDL-C:** (<40 mg/dl in males and <50 mg/dl in females or Rx to increase HDL-C), **High BP** {systolic blood pressure (SBP) ≥130 mmHg and/or diastolic blood pressure (DBP)

≥85 mmHg or antihypertensive Rx}, **High fasting blood sugar** (FBS) {≥100 mg/dl or reported Type 2 Diabetes Mellitus (T2DM)}.

Data Analysis: Values of serum leptin levels obtained from participants were categorized in tertiles according to leptin levels for males (T1 <4.80 ng/ml, T2 ≥4.80 to <10.30 ng/ml, T3 ≥10.30 ng/ml) and females (T1 <9.50 ng/ml, T2 ≥9.50 to <24.50 ng/ml, T3 ≥24.50 ng/ml). The serum leptin value of >9 ng/ml in males and >20 ng/ml in females were considered as high for the analysis.

Statistical analysis was performed with SigmaPlot 12.0 for Windows (Systat Software Inc., California, USA) and SPSS 16.0 for Windows(SPSS Inc., Chicago, USA).The unpaired t test and analysis of variance (ANOVA) were used for comparisons of means of continuous variables with normal distribution whereas the Mann–Whitney and the Kruskal-Wallis test were used for non-normal distribution. The chi-square ($\chi 2$) test was used for comparisons of means of categorical variables. Correlations between data sets were calculated by Spearmen's rank correlation. A binary logistic regression model was used to evaluate association of serum leptin levels with MetS and its components. The similar association was also evaluated after adjusting serum leptin levels for age and age, BMI, tobacco usage respectively. The odds ratios (OR) were calculated to estimate the relative risk of developing MetS and individual components of MetS. Additionally, receiver operating curves (ROC) were carried out for leptin level as a predictor of MetS and cutoff levels were identified. Conversely, ROC analyses for components of MetS as a predictor of high serum leptin levels were carried out. A p-value <0.05 was considered statistically significant.

RESULTS:

In this study total 155 male and 142 female participants were included having mean age of 47.96 ± 13.81 years with no significant difference between the sexes. Almost 94% participants had at least one abnormal MetS component and found overall prevalence of MetS as 40.06% with no significant difference between genders.

Table-1. Tertiles of leptin concentration and gender wise characteristics of the study population.

Variables (Unit)	Leptin Tertile 1	Leptin Tertile 2	Leptin Tertile 3	P-value
Males	Mean±SD / Median (inter-quartile range) / N (%)			
Age (years)	44.52±14.18	47.29±13.10	53.53±12.38	**0.002**
BMI (kg/m^2)	22.73±4.04	25.60±3.37	28.03±3.26	**<0.001**
SBP (mmHg)	124 (116-130)	128 (122-134)	130 (126-136)	**0.001**
DBP (mmHg)	79.94±4.38	83.40±5.51	84.28±4.85	**<0.001**
WC (cm)	89.25 (80.75-95)	95 (89.63-104.5)	104 (99-108)	**<0.001**
LDL-C (mg/dl)	117.29±33.54	116.42±28.71	116.42±36.86	0.988
HDL-C (mg/dl)	42.93±9.18	43.57±9.54	38.21±8.95	**0.007**
TC (mg/dl)	182.25±38.02	182.35±33.08	180.20±47.65	0.954
TGs (mg/dl)	102 (84-139.75)	125.5 (86-168.5)	140 (96-179)	**0.042**
FBS (mg/dl)	91.5 (87.25-97.75)	98 (90-110.5)	102 (93-116)	**<0.001**
Tobacco Use (%)	19 (36.54)	24 (46.15)	23 (45.10)	0.554
On Antihypertensive (%)	05 (9.62)	12 (23.10)	20 (39.22)	**0.002**
On Hypoglycemic (%)	08 (15.39)	12 (23.10)	15 (29.41)	0.234
MetS (%)	08 (15.39)	19 (36.54)	31 (60.78)	**<0.001**
Females	Mean±SD / Median (inter-quartile range) / N (%)			
Age (years)	41.70±13.40	50.15±14.34	51±12.33	**0.001**
BMI (kg/m^2)	20.86 (18.57-24.33)	26.56 (24.67-29.93)	30.80 (28.05-33.75)	**<0.001**
SBP (mmHg)	123.63±13.01	128.38±11.45	131.54±11.44	**0.007**
DBP (mmHg)	81.04±6.78	81.71±6.52	84.58±5.99	**0.019**
WC (cm)	78.5 (67-90.5)	95 (89.25-102.75)	100 (93.25-107.75)	**<0.001**
LDL-C (mg/dl)	109.39±32.48	120.16±28.02	126.25±31.17	**0.029**
HDL-C (mg/dl)	42.2 (38-49.13)	44.95 (39.7-52.85)	40.95 (37.15-44.97)	**0.038**
TC (mg/dl)	169 (144.75-199.25)	190 (168.5-204.75)	191 (172-203)	**0.050**
TGs (mg/dl)	97 (86.75-110.50)	125 (88.25-151.25)	117 (98.25-141.50)	**0.006**
FBS (mg/dl)	81 (72-94)	91 (83.5-101)	97.5 (87.25-107.75)	**<0.001**
Tobacco Use (%)	10 (21.74)	5 (10.42)	5 (10.42)	0.193
On Antihypertensive (%)	5 (10.87)	17 (35.42)	20 (41.67)	**0.003**
On Hypoglycemic (%)	3 (6.52)	4 (8.33)	12 (25.00)	**0.014**
MetS (%)	8 (17.39)	22 (45.83)	31 (64.58)	**<0.001**

N (%) for categorical variables, mean±SD for continuous variables with normal distribution and median (inter-quartile range) for non-normal distribution

Abdominal obesity, low HDL-C and high BP were found the most prevalent components of MetS. Age, BMI, TGs and FBS were found significantly high in group of participants having MetS comparing to group of participants not having MetS. No significant differences in the values of LDL-C and TC were observed between these groups in both the sexes.

The mean leptin level for study participants was 10.08±9.09 ng/ml in males and 21.80±18.6 ng/ml in females. Leptin levels were found to be significantly high in individuals with MetS comparing to individuals with no MetS [males: 11.27(6.64–15.99) vs. 5.27(2.7–8.93) ng/ml, P <0.001 and females: 25.4(14.13–40.78) vs. 10.1(6.5–21.49) ng/ml, P <0.001]. In study participants, leptin levels significantly increased with increasing number of MetS components (P <0.001) in both males and females.

Table-1 shows the characteristics of male and female participants stratified by leptin tertiles. In both sexes, values of study variables rose as leptin levels rose from tertile one to three with exception of LDL-C, HDL-C, TC in males and HDL-C, TGs in females. Most of these parameters were statistically significant, particularly for BMI, WC and FBS in both sexes. Similarly, percentage of participants with MetS, taking antihypertensive drugs and hypoglycemic drugs also showed significant rise from tertile one to three in both sexes.

Table-2: Gender specific, Spearmen's rank correlation coefficient between leptin and MetS components

MetS Components	Male (n=155) R-value	P-value	Female (n=142) R-value	P-value
Age (years)	0.248	0.002	0.268	0.001
BMI (kg/m^2)	0.597	0.000	0.703	0.000
SBP (mmHg)	0.308	0.000	0.288	0.000
DBP (mmHg)	0.369	0.000	0.257	0.002
WC (cm)	0.646	0.000	0.593	0.000
HDL-C (mg/dl)	-0.192	0.017	-0.0609	0.471
TGs (mg/dl)	0.242	0.003	0.235	0.005
FBS (mg/dl)	0.280	0.000	0.337	0.000

Spearman's rank correlation analysis of leptin level with age and BMI showed significant positive correlation. Likewise, correlations of leptin levels with components of MetS were also found significantly positive in both sexes except for HDL-C. As expected, HDL-C showed negative correlation with leptin, but values were not found statistically significant in males and females (Table-2).

Furthermore, ROC analysis was explored to evaluate the ability of serum leptin levels to predict MetS in both sexes (Figure-1). After adjusting serum leptin values for concomitant parameters, at the cutoff of 9.38 ng/ml leptin predicted MetS with sensitivity of 74% and specificity of 59% in males (area under curve-AUC=0.737) and at the cutoff of 21.01 ng/ml leptin predicted MetS with sensitivity of 77% and specificity of 67% in females (AUC=0.769). Conversely, ROC analysis was also explored to evaluate the ability of each component of MetS to predict high leptin levels (Table-3).

Table-3. Gender wise ROC analysis – Age, BMI and components of MetS as predictors of high leptin levels. AUC=area under curve

Males				
Component of MetS (cut-off)	AUC (95% CI)	Sensitivity (%)	Specificity (%)	P-value
Age (49 years)	0.644 (0.556–0.732)	63	57	0.0023
BMI (25.37 kg/m^2)	0.795 (0.724–0.866)	79	65	<0.0001
WC (96.5 cm)	0.819 (0.750–0.888)	81	76	<0.0001
FBS(97 mg/dl)	0.655 (0.569–0.742)	65	62	0.0010
SBP (127 mmHg)	0.653 (0.566–0.739)	66	56	0.0013
DBP (83 mmHg)	0.679 (0.594–0.764)	66	65	0.0002
TGs (125 mg/dl)	0.633 (0.541–0.725)	63	62	0.0050
Low HDL-C (41.1 mg/dl)	0.325 (0.237–0.413)	34	38	0.0002
Females				
Age (46 years)	0.594 (0.500–0.688)	59	50	0.0603
BMI (26.34 kg/m^2)	0.826 (0.756–0.896)	85	69	<0.0001
WC(93.5 cm)	0.737 (0.656–0.819)	74	59	<0.0001
FBS(89 mg/dl)	0.678 (0.590–0.766)	70	51	0.0003
SBP (127 mmHg)	0.619 (0.525–0.712)	63	53	0.0176
DBP (81 mmHg)	0.618 (0.525–0.712)	67	44	0.0180
TGs (107 mg/dl)	0.573 (0.476–0.669)	57	52	0.1477
Low HDL-C (43.7 mg/dl)	0.412 (0.316–0.508)	41	52	0.0794

Binary logistic regression analysis was used to estimate the predictive role of serum leptin concentration as a continuous variable relating to the risk of developing MetS and its components. In Table-4, Model-A and Model-B includes serum leptin values adjusted for age and age, BMI, tobacco usage respectively. Without adjusting serum leptin values for concomitant parameters, the risk of developing MetS was likely to increase by 1.04 times in females and by 1.06 times in males. After adjusting serum leptin values for age (Model-A), this estimation was raised to 1.18 times in females and 1.20 times in males. While after adjusting serum leptin values for age, BMI and tobacco usage (Model-B) this estimation reached to 1.10 times in females and remain unaffected in males. Additionally, serum leptin levels significantly predicted the relative risk of developing components of MetS respectively abdominal obesity, high BP and high FBS, but it did not significantly predicted the relative risk of developing hypertriglyceridemia and low HDL-C.

Table-4. Relative risk of developing the MetS and specific MetS components with serum leptin concentration.

Males						
Dependent Variable	Independent Variable					
	Leptin		Model-A		Model-B	
	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P
Abdominal Obesity (n=109)	1.255 (1.131-1.392)	<0.001	1.000 (0.849-1.178)	0.998	1.684 (1.414-2.006)	<0.001
High Blood Pressure (n=73)	1.026 (0.990-1.064)	0.156	1.637 (1.352-1.981)	<0.001	1.152 (1.070-1.239)	<0.001
High Fasting Glucose (n=62)	1.056 (1.015-1.098)	0.007	1.275 (1.084-1.499)	0.003	1.093 (1.021-1.170)	0.011
Hypertriglyceridemia (n=47)	1.025 (0.989-1.063)	0.177	1.029 (0.874-1.210)	0.733	1.056 (0.985-1.132)	0.128
Low HDL Cholesterol (n=73)	1.024 (0.988-1.062)	0.188	1.041 (0.896-1.210)	0.598	1.031 (0.968-1.099)	0.340
Metabolic Syndrome (n= 58)	1.059 (1.018-1.102)	0.004	1.195 (1.019-1.401)	0.029	1.199 (1.106-1.300)	<0.001
Females						
Abdominal Obesity (n=106)	1.112 (1.055-1.172)	<0.001	1.179 (1.047-1.328)	0.007	1.458 (1.269-1.675)	<0.001
High Blood Pressure (n=78)	1.023 (1.002-1.043)	0.030	1.369 (1.208-1.551)	<0.001	1.074 (1.034-1.115)	<0.001
High Fasting Glucose (n=38)	1.026 (1.006-1.046)	0.010	1.161 (1.043-1.292)	0.006	1.064 (1.024-1.107)	0.002
Hypertriglyceridemia (n=24)	1.014 (0.992-1.036)	0.207	1.118 (0.991-1.261)	0.069	1.035 (0.994-1.077)	0.094
Low HDL Cholesterol (n=114)	1.004 (0.981-1.027)	0.379	1.019 (0.909-1.143)	0.743	0.965 (0.928-1.002)	0.065
Metabolic Syndrome (n=61)	1.038 (1.016-1.060)	<0.001	1.177 (1.065-1.301)	0.001	1.097 (1.053-1.144)	<0.001

OR (95% CI) = odds ratio (95% confidence interval), Model-A=serum leptin values adjusted for age, Model-B=serum leptin values adjusted for age, BMI & tobacco usage.

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DISCUSSION:

Obesity is associated with changes in many biochemical variables, especially adipokines like leptin and adiponectin.[8] Studies have confirmed that South Asians have the least favorable adipokine profile over other ethnic population.[9,10] Study conducted on migrant South Asians in USA reported a strong association between leptin and body composition including BMI, total body fat, visceral fat, and hepatic fat.[11] In light of this, present study explored the possible association between serum leptin levels and MetS and components of MetS in an adult population of Saurashtra region, Gujarat.

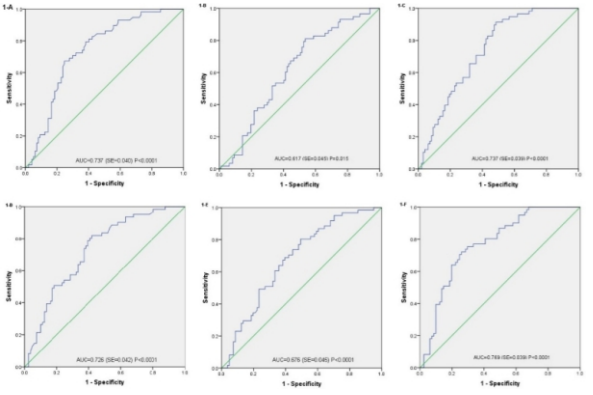
Applying the newly accepted JIS-2009 diagnostic criteria for MetS[7] to this study population, more than 40% of the individuals met this criterion with abdominal obesity, low HDL-C and high BP as the most prevalent components of MetS. Prasad et al and Ramachandran et al observed similar findings in their respective study population.[12,13] Other than the proposed recommendations for Asian Indians, this population specific high prevalence of MetS might be due to the composition of diet, affluence in urbanization and sedentary life style.[14,15,16] The observed incidences of being overweight – obesity in MetS participants highlighted this as major determinant of MetS.

Leptin and components of MetS: The increased level of serum leptin in most overweight - obese individuals may be secondary to leptin resistance; as it fails to suppress appetite and/or fails to normalize adipose tissue mass.[5] Observations of other previously reported studies[4,17,18] had highlighted such inter-relationship between leptin and obesity. In present study, significant variation in BMI and WC from tertile one to three of leptin levels and positive correlation coefficient between these components (BMI: r=0.597 in males, r=0.703 in females and WC: r=0.646 in males, r=0.593 in females) were observed. Additionally, BMI and WC strongly predicted high leptin levels through ROC analysis (Table-3). Conversely binary logistic regression analysis revealed independent association between leptin and abdominal obesity in both sexes. In humans, increased levels of circulating free fatty acids were observed in obesity and its augmented levels were found inversely correlated with insulin sensitivity.[19,20]

Hyperleptinemia, either manifested due to age-related obesity or produced by the consumption of energy-enriched diets, is primarily responsible for inducing hyperglycemia and hyperinsulinemia; the persistence of this patho-physiologic continuation often leads to diabetes mellitus.[21] The present study has observed a significant increase in values of FBS from the first to third tertile of leptin levels. Spearman's rank correlation showed an association between leptin and FBS (males-0.280, females-0.337), and the binary logistic regression analysis (OR: males-1.093, females-1.064) showed independent relative risk of developing high FBS. On the basis of an association between leptin and increased fasting and post-load glucose levels, an eleven years' follow-up study has also shown that leptin predicts future development of diabetes mellitus.[22] These all observations are suggestive of the role of leptin in patho-physiology related to glucose metabolism and insulin activity. A complicated relationship exists between leptin and other adipokines which work collectively to impair insulin signaling, create state of inflammation, and/or interact to alter sympathetic regulation.[23] This in turn activates a sympathetic response which increases peripheral vascular resistance and renal sympathetic tone contributing towards the development of hypertension.[24,25] This study also observed a significant increase in values of SBP and DBP from the first to third tertile of leptin levels. Spearman's rank correlation between leptin and SBP and DBP (males: SBP r=0.308 DBP r=0.369, females: SBP r=0.288 DBP r=0.257) depicted positive association between leptin and BP. Moreover, binary logistic regression analysis between leptin and BP showed independent relative risk of developing high BP in both sexes (Table-4). Previous cross-sectional and prospective epidemiological studies have also observed a similar independent positive association in males.[26,27] Similarly the National Heart, Lung, and Blood Institute Family Heart Study has identified significant associations between plasma leptin levels and both BP and hypertension in women.[28]

Unlike obesity, FBS and BP, the present study found a weak relationship between leptin and lipid profile parameters. Similar findings were observed by Martins et al and Gupta et al in their respective study populations.[4,29] Most of our study population had age more than 50 years and TGs levels tends to be stable after this age.[30] Moreover, Kakuma et al has reported that leptin represses gene coding for stearoyl-CoA desaturase-1 (SCD-1), an enzyme involved in the synthesis of TGs.[31,32]

Figure-1: Receiver Operating Curve (ROC) analyses for leptin levels as predictor of MetS



Analyses for males (1-A, 1-B, 1-C) and females (1-D, 1-E, 1-F). 1-A & 1-D for leptin vs. MetS without adjustment, 1-B & 1-E for age adjusted, 1-C & 1-F for age, BMI, tobacco usage adjusted. AUC (Std. Error) were 0.737(0.040), 0.617 (0.045), 0.737 (0.039) in males and 0.726 (0.042), 0.676 (0.045), 0.769 (0.039) in females.

Leptin and MetS: Several researchers have reported higher leptin levels in individuals with MetS and even found it to be related to the components of MetS in healthy individuals.[33,34,35] The present study also supports these findings; significantly higher leptin values have been detected in individuals with MetS (P <0.001). Additionally, we had observed that increase in value of serum leptin also increases the number of determinants of MetS.

Through binary logistic regression and ROC analysis, this study explored the relationship between serum leptin levels and the risk of MetS. In both sexes, analysis of this relationship, without adjusting for the concomitant parameters, indicated that serum leptin has the ability to predict the MetS. Additionally, this relationship did not alter even after serum leptin levels were adjusted for the effect of age, BMI and tobacco usage. Cutoff values of 9.38 ng/ml in males and 21.01 ng/ml in females of serum leptin levels predicted MetS after adjusting for concomitant parameters in this ethnic population. Abdominal obesity, high BP and high FBS also predicted high leptin values through ROC analysis. These clustered features of MetS could be indicative of increasing cardiovascular risk in this population.

However, the high values of OR of MetS and components of MetS were observed in males compared to females, with and without serum leptin levels adjusted for the concomitant parameters (Table-4). This observation might be suggestive of a better correlation between serum leptin levels and the development of MetS in males compared to females. Similar observation has been made by the studies conducted on Taiwanese and Korean population.[36,37] It might be possible due to the gender differences in body fat distribution or due to the effect of sex hormones.[38,39] However, it warrants further study, to determine the role of adipose tissue distribution or sex hormones in the association between MetS and serum leptin levels.

The main limitation of the present study is the innate characteristics of a cross-sectional study; hence, longitudinal follow-up studies are important to legitimize these findings towards the causal pathway of diabetes and cardio-metabolic risk. Furthermore, analysis for specific markers of diabetes and CVD and detailed information

regarding exercise and diet could have given better insights to this research.

CONCLUSION:

Leptin has pleiotropic effects; besides its important role in energy homeostasis, it plays role in the patho-physiology of obesity-related CVDs.[40] This study confirmed that independent of age, BMI and tobacco usage, leptin levels are positively associated with abdominal obesity, high BP and high FBS which are known risk factors for CVDs. Additionally, leptin level increased with increase in components of MetS and was found to be associated with MetS. Hence, leptin can be used as a biomarker for MetS and cardiovascular risk. Therefore derived serum leptin cutoff levels should be taken as a warning sign in the development of MetS in this ethnic population. Henceforth, individuals having high serum leptin levels should be motivated for changes in dietary habits and lifestyle towards prevention of cardiovascular risk.

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