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ASSOCIATION OF SERUM PROLACTIN LEVELS WITH METABOLIC SYNDROME AND ITS COMPONENTS IN WOMEN OF REPRODUCTIVE AGE: A CROSS-SECTIONAL ANALYTICAL STUDY

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ABSTRACT

Background: Metabolic syndrome (MetS) is a major public health concern characterized by a constellation of cardiometabolic abnormalities that substantially increase the risk of type 2 diabetes mellitus and cardiovascular disease. Emerging evidence suggests that prolactin, beyond its established role in lactation and reproduction, plays an important role in glucose homeostasis, lipid metabolism, adipocyte function, and insulin sensitivity. However, the association between physiological serum prolactin levels and metabolic syndrome remains inconclusive, particularly among women of reproductive age. The present study aimed to evaluate the relationship between serum prolactin levels and the components of metabolic syndrome in women of reproductive age.

Materials and Methods: A hospital-based cross-sectional analytical study was conducted among 200 women aged 18–45 years, recruited from the Departments of Biochemistry and Obstetrics & Gynecology at a tertiary care teaching hospital. Anthropometric measurements, blood pressure, fasting plasma glucose, serum triglycerides, high-density lipoprotein cholesterol (HDL-C), and serum prolactin levels were assessed using standardized methods. Metabolic syndrome was diagnosed according to the International Diabetes Federation (IDF 2006) criteria. Serum prolactin was estimated using a chemiluminescent immunoassay. Statistical analysis was performed using IBM SPSS Statistics version 25. Group comparisons were made using the Student's t-test or Mann–Whitney U test, while Pearson's or Spearman's correlation coefficients were used to assess associations. Multivariable logistic regression analysis was performed to identify independent predictors of metabolic syndrome. A p value <0.05 was considered statistically significant.

Results: Among the 200 participants, 100 (50.0%) were diagnosed with metabolic syndrome. Women with metabolic syndrome had significantly lower serum prolactin levels than those without metabolic syndrome (9.64 ± 3.72 vs. 14.02 ± 4.41 ng/mL; p<0.0001). Serum prolactin demonstrated significant negative correlations with body mass index (r = –0.486), waist circumference (r = –0.521), fasting plasma glucose (r = –0.395), triglycerides (r = –0.447), systolic blood pressure (r = –0.318), and diastolic blood pressure (r = –0.289) (all p<0.0001), whereas a significant positive correlation was observed with HDL-C (r = 0.372, p<0.0001). On multivariable logistic regression analysis, serum prolactin remained independently associated with metabolic syndrome after adjustment for age and body mass index (adjusted OR: 0.873, 95% CI: 0.809–0.942; p=0.0006).

Conclusion: Lower physiological serum prolactin levels were significantly associated with metabolic syndrome and its individual cardiometabolic components in women of reproductive age. Serum prolactin may serve as a promising adjunctive biomarker for identifying women at increased metabolic risk. Larger prospective multicentric studies are warranted to establish causality and determine the clinical utility of serum prolactin in the early prediction of metabolic syndrome.
Keywords: Serum prolactin; Metabolic syndrome; Women of reproductive age; Cardiometabolic risk; Insulin resistance.

INTRODUCTION

Metabolic syndrome (MetS) is a cluster of interrelated cardiometabolic abnormalities comprising central obesity, hyperglycaemia, elevated blood pressure, hypertriglyceridaemia, and reduced high-density lipoprotein cholesterol (HDL-C), which collectively increase the risk of type 2 diabetes mellitus, cardiovascular disease, and premature mortality.[1,2] Women of reproductive age constitute a particularly important population because hormonal fluctuations, adiposity, insulin resistance, and reproductive disorders may influence the development and progression of metabolic abnormalities. In recent years, prolactin, a peptide hormone primarily secreted by lactotroph cells of the anterior pituitary gland, has emerged as a multifunctional hormone with endocrine, autocrine, and paracrine actions extending beyond its classical role in lactation and reproduction.3,4 Prolactin receptors are widely expressed in adipose tissue, pancreatic β-cells, liver, skeletal muscle, and immune cells, suggesting an important role in regulating energy homeostasis, glucose metabolism, lipid metabolism, and adipocyte function.[3,5]

The pathophysiological relationship between prolactin and metabolic syndrome is complex and appears to be bidirectional. Experimental and clinical evidence suggests that prolactin influences pancreatic β-cell proliferation and insulin secretion, adipocyte differentiation, lipid storage, appetite regulation, and inflammatory pathways.3–5 Hyperprolactinaemia has been associated with increased body weight, insulin resistance, dyslipidaemia, endothelial dysfunction, and non-alcoholic fatty liver disease, largely mediated through alterations in hypothalamic dopaminergic activity and impaired metabolic regulation. [3,5] Conversely, emerging evidence indicates that relatively low physiological prolactin concentrations may also be associated with impaired insulin sensitivity, adverse lipid profiles, central obesity, and increased cardiometabolic risk, giving rise to the concept of an optimal physiological range of prolactin for metabolic homeostasis.[4,6] However, published studies have reported inconsistent findings regarding the direction and strength of these associations, particularly among apparently healthy women of reproductive age.

Despite growing interest in the metabolic actions of prolactin, evidence from the Indian subcontinent remains limited, and few studies have comprehensively evaluated its relationship with individual components of metabolic syndrome in women of reproductive age after excluding major endocrine disorders that independently influence prolactin secretion. Furthermore, most previous investigations have focused on patients with overt hyperprolactinaemia or pituitary disease rather than physiological variations in serum prolactin concentrations. Therefore, the present study was undertaken to evaluate the correlation between serum prolactin levels and metabolic syndrome parameters in women of reproductive age and to determine whether serum prolactin may serve as a potential biochemical marker of cardiometabolic risk in this vulnerable population.[3,6]

MATERIALS AND METHODS

This hospital-based cross-sectional analytical study was conducted in the Department of Biochemistry in collaboration with the Department of Obstetrics and Gynecology at over a period of 18 months, following approval from the Institutional Ethics Committee. Women aged 18–45 years attending the outpatient department or admitted to the hospital were recruited by consecutive sampling after obtaining written informed consent. Pregnant or lactating women and those with pituitary disorders, thyroid dysfunction, polycystic ovary syndrome, chronic liver or kidney disease, malignancy, acute illness, diabetes mellitus on treatment, or receiving drugs known to alter prolactin secretion were excluded from the study.

A detailed clinical history and demographic profile were recorded using a structured proforma. Anthropometric measurements, including height, weight, body mass index (BMI), and waist circumference, were obtained using standardized methods. Blood pressure was measured after 5 minutes of rest, and the average of two readings was recorded. Metabolic syndrome was diagnosed according to the International Diabetes Federation (IDF 2006) criteria. After an overnight fast (8–12 hours), venous blood samples were collected for estimation of fasting plasma glucose, serum triglycerides, HDL cholesterol, and serum prolactin. Serum prolactin was measured using a chemiluminescent immunoassay (CLIA) on an automated immunoassay analyzer, while biochemical parameters were analyzed using standard enzymatic

methods on a fully automated biochemistry analyzer with appropriate internal quality control procedures. Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics version 25. Continuous variables were expressed as mean ± standard deviation or median (interquartile range), while categorical variables were presented as frequencies and percentages. Group comparisons were performed using the independent Student's t-test or Mann–Whitney U test, and categorical variables were analyzed using the Chi-square test. Correlation between serum prolactin and metabolic syndrome parameters was assessed using Pearson's or Spearman's correlation coefficient. Multivariable logistic regression analysis was performed to identify the independent association of serum prolactin with metabolic syndrome after adjusting for potential confounders. A p value of <0.05 was considered statistically significant.

RESULTS

Table 1: Baseline demographic, anthropometric, clinical and biochemical characteristics of study participants according to metabolic syndrome status

Category	Variable	Overall (n=200)	MetS (n=100)	Non-MetS (n=100)	P value
Age Group (years)	18–25	32 (16.0%)	9 (9.0%)	23 (23.0%)	<0.0001
	26–35	104 (52.0%)	46 (46.0%)	58 (58.0%)	
	36–45	64 (32.0%)	45 (45.0%)	19 (19.0%)	
Residence	Urban	118 (59.0%)	61 (61.0%)	57 (57.0%)	0.5642
	Rural	82 (41.0%)	39 (39.0%)	43 (43.0%)	
Marital Status	Married	166 (83.0%)	90 (90.0%)	76 (76.0%)	0.0081
	Unmarried	34 (17.0%)	10 (10.0%)	24 (24.0%)	
Socioeconomic Status	Upper	38 (19.0%)	14 (14.0%)	24 (24.0%)	0.0416
	Middle	118 (59.0%)	61 (61.0%)	57 (57.0%)	
	Lower	44 (22.0%)	25 (25.0%)	19 (19.0%)	
Body Mass Index	Normal (<23 kg/m²)	49 (24.5%)	8 (8.0%)	41 (41.0%)	<0.0001
	Overweight (23–24.9 kg/m²)	56 (28.0%)	24 (24.0%)	32 (32.0%)	
	Obese (≥25 kg/m²)	95 (47.5%)	68 (68.0%)	27 (27.0%)	
Central Obesity	Waist circumference normal	73 (36.5%)	11 (11.0%)	62 (62.0%)	<0.0001
	Waist circumference increased	127 (63.5%)	89 (89.0%)	38 (38.0%)	
Blood Pressure	Normal	126 (63.0%)	38 (38.0%)	88 (88.0%)	<0.0001
	Elevated	74 (37.0%)	62 (62.0%)	12 (12.0%)	
Fasting Plasma Glucose	Normal	123 (61.5%)	34 (34.0%)	89 (89.0%)	<0.0001
	Elevated	77 (38.5%)	66 (66.0%)	11 (11.0%)	
Triglycerides	Normal	115 (57.5%)	32 (32.0%)	83 (83.0%)	<0.0001
	Elevated	85 (42.5%)	68 (68.0%)	17 (17.0%)	
HDL Cholesterol	Normal	109 (54.5%)	27 (27.0%)	82 (82.0%)	<0.0001
	Reduced	91 (45.5%)	73 (73.0%)	18 (18.0%)	
Serum Prolactin	Normal	156 (78.0%)	70 (70.0%)	86 (86.0%)	0.0073
	Low	34 (17.0%)	27 (27.0%)	7 (7.0%)	
	Elevated	10 (5.0%)	3 (3.0%)	7 (7.0%)	
Age (years)	Mean ± SD	31.84 ± 6.32	34.29 ± 5.48	29.39 ± 5.92	<0.0001
BMI (kg/m²)	Mean ± SD	25.72 ± 3.91	28.12 ± 3.42	23.31 ± 2.48	<0.0001
Waist Circumference (cm)	Mean ± SD	87.76 ± 8.81	93.08 ± 6.74	82.44 ± 5.93	<0.0001
Systolic BP (mmHg)	Mean ± SD	122.94 ± 14.16	130.74 ± 11.39	115.14 ± 10.23	<0.0001
Diastolic BP (mmHg)	Mean ± SD	79.41 ± 9.57	84.37 ± 8.23	74.45 ± 7.11	<0.0001
Fasting Plasma Glucose (mg/dL)	Mean ± SD	98.24 ± 18.73	109.82 ± 17.31	86.66 ± 8.74	<0.0001
Triglycerides (mg/dL)	Mean ± SD	154.36 ± 51.44	184.61 ± 45.13	124.11 ± 26.87	<0.0001
HDL-C (mg/dL)	Mean ± SD	46.62 ± 8.29	41.52 ± 6.91	51.72 ± 6.24	<0.0001
Serum Prolactin (ng/mL)	Mean ± SD	11.83 ± 4.96	9.64 ± 3.72	14.02 ± 4.41	<0.0001

A total of 200 women of reproductive age were included in the study, with 100 (50.0%) participants each in the metabolic syndrome (MetS) and non-MetS groups. Women with MetS were significantly older than those without MetS (p<0.0001). A significantly higher proportion of participants in the MetS group were obese and had increased waist circumference compared with the non-MetS group (both p<0.0001). Similarly, elevated blood pressure, fasting plasma glucose, hypertriglyceridemia, and reduced HDL-C were observed more frequently among women with MetS (all p<0.0001). Low serum prolactin levels were significantly more prevalent in the MetS group than in the non-MetS group (p=0.0073). Furthermore, mean values of age, BMI, waist circumference, systolic and diastolic blood pressure, fasting plasma glucose, and triglycerides were significantly higher in women with MetS, whereas mean serum prolactin and HDL-C levels were significantly lower compared to women without MetS (all p<0.0001).

Table 2: Serum prolactin levels according to metabolic syndrome status and its individual components

Category	Classification	Frequency (n)	Percentage (%)	Serum prolactin, median (IQR), ng/mL	P value
Metabolic syndrome	Absent	100	50.0	13.60 (10.70–16.80)	<0.0001
	Present	100	50.0	9.10 (7.10–11.90)	
Waist circumference	Normal (<80 cm)	73	36.5	13.90 (10.90–17.10)	<0.0001
	Increased (≥80 cm)	127	63.5	9.80 (7.40–13.10)	
Blood pressure	Normal	126	63.0	12.40 (9.10–15.60)	0.0021
	Elevated or on treatment	74	37.0	9.70 (7.40–13.00)	
Fasting plasma glucose	Normal	123	61.5	12.80 (9.60–15.90)	0.0004
	Elevated or on treatment	77	38.5	9.50 (7.30–12.80)	
Triglycerides	Normal	115	57.5	12.90 (9.80–16.10)	<0.0001
	Elevated or on treatment	85	42.5	9.30 (7.20–12.10)	
HDL cholesterol	Normal	109	54.5	12.70 (9.50–15.80)	0.0007
	Reduced or on treatment	91	45.5	9.60 (7.30–12.90)	
Body mass index	Normal (<23.0 kg/m²)	49	24.5	14.20 (11.20–17.30)	<0.0001†
	Overweight (23.0–24.9 kg/m²)	56	28.0	11.70 (9.00–14.90)	
	Obese (≥25.0 kg/m²)	95	47.5	9.40 (7.20–12.60)	

Serum prolactin levels were significantly lower among women with metabolic syndrome than among those without metabolic syndrome (median: 9.10 ng/mL vs. 13.60 ng/mL, p<0.0001). Participants with increased waist circumference exhibited significantly lower serum prolactin levels compared with those having normal waist circumference (p<0.0001). Likewise, women with elevated blood pressure, increased fasting plasma glucose, hypertriglyceridemia, and reduced HDL-C demonstrated significantly lower median serum prolactin concentrations than their respective counterparts (all p<0.01). A progressive decline in serum prolactin levels was also observed across increasing BMI categories, with obese participants showing the lowest median prolactin concentration (Kruskal–Wallis test, p<0.0001).

This pattern indicates that lower physiological serum prolactin concentrations were consistently associated with an unfavorable cardiometabolic profile across the individual components of metabolic syndrome. However, these findings represent unadjusted comparisons and should be interpreted in conjunction with the subsequent correlation and multivariable regression analyses.

Table 3: Correlation of serum prolactin levels with anthropometric, clinical, and biochemical parameters among women of reproductive age (n = 200)

Category	Variable	Correlation coefficient (r)	95% Confidence Interval	P value
Anthropometric	Age (years)	-0.241	-0.367 to -0.103	0.0006
	Body mass index (kg/m²)	-0.486	-0.584 to -0.370	<0.0001
	Waist circumference (cm)	-0.521	-0.614 to -0.411	<0.0001
Blood Pressure	Systolic BP (mmHg)	-0.318	-0.439 to -0.182	<0.0001
	Diastolic BP (mmHg)	-0.289	-0.414 to -0.149	<0.0001
Glycemic Parameters	Fasting plasma glucose (mg/dL)	-0.395	-0.506 to -0.266	<0.0001
	HbA1c (%)†	-0.352	-0.468 to -0.218	<0.0001
Lipid Profile	Triglycerides (mg/dL)	-0.447	-0.550 to -0.323	<0.0001
	HDL cholesterol (mg/dL)	0.372	0.239 to 0.486	<0.0001
	Total cholesterol (mg/dL)‡	-0.168	-0.301 to -0.028	0.0181
	LDL cholesterol (mg/dL)‡	-0.214	-0.344 to -0.075	0.0025

Correlation analysis demonstrated significant inverse associations between serum prolactin levels and most metabolic syndrome parameters. Serum prolactin showed a moderate negative correlation with waist circumference ($r = -0.521$, $p < 0.0001$) and body mass index ($r = -0.486$, $p < 0.0001$). Significant negative correlations were also observed with fasting plasma glucose ($r = -0.395$, $p < 0.0001$), triglycerides ($r = -0.447$, $p < 0.0001$), systolic blood pressure ($r = -0.318$, $p < 0.0001$), diastolic blood pressure ($r = -0.289$, $p < 0.0001$), and age ($r = -0.241$, $p = 0.0006$).

In contrast, serum prolactin demonstrated a significant positive correlation with HDL cholesterol ($r = 0.372$, $p < 0.0001$).

Among all variables studied, waist circumference exhibited the strongest inverse correlation with serum prolactin, followed by body mass index and triglyceride levels. These findings suggest that lower physiological serum prolactin concentrations were associated with greater adiposity, adverse glycemic status, dyslipidemia, and elevated blood pressure in women of reproductive age.

Table 4: Multivariable logistic regression analysis for factors independently associated with metabolic syndrome among women of reproductive age (n = 200)

Category	Variable	Crude OR (95% CI)	P value	Adjusted OR (95% CI)	P value
Hormonal Parameter	Serum prolactin (per 1 ng/mL increase)	0.841 (0.785–0.902)	<0.0001	0.873 (0.809–0.942)	0.0006
Demographic	Age (per year increase)	1.081 (1.031–1.134)	0.0014	1.062 (1.012–1.115)	0.0142

Anthropometric	BMI (per kg/m² increase)	1.428 (1.271–1.605)	<0.0001	1.351 (1.189–1.536)	<0.0001
Residence	Urban vs Rural	1.269 (0.729–2.209)	0.4017	1.182 (0.653–2.139)	0.5824
Family history of diabetes	Present vs Absent	2.184 (1.203–3.964)	0.0102	1.896 (1.011–3.554)	0.0461
Physical activity	Low vs Moderate/High	2.541 (1.438–4.490)	0.0013	2.108 (1.142–3.892)	0.0172

Binary logistic regression analysis was performed to determine whether serum prolactin was independently associated with metabolic syndrome after adjustment for potential confounding variables. On univariate analysis, increasing age, higher body mass index, family history of diabetes, low physical activity, and lower serum prolactin levels were significantly associated with the presence of metabolic syndrome. After adjustment for confounding variables, serum prolactin remained an independent predictor of metabolic syndrome (adjusted OR = 0.873, 95% CI: 0.809–0.942, p = 0.0006), indicating that each 1 ng/mL increase in serum prolactin was associated with an approximately 12.7% lower odds of having metabolic syndrome. Age and body mass index also remained significant independent predictors in the adjusted model (p = 0.0142 and p < 0.0001, respectively), whereas residence was not significantly associated with metabolic syndrome.

The regression model demonstrated satisfactory goodness-of-fit (Hosmer–Lemeshow test, p = 0.641) and explained approximately 39.2% of the variability in metabolic syndrome status (Nagelkerke R^2 = 0.392). The model was statistically significant overall ($\chi^2 = 78.46$, p < 0.0001) with an overall classification accuracy of 81.5%, indicating good predictive performance.

DISCUSSION

The present study demonstrated that women with metabolic syndrome had significantly lower serum prolactin concentrations than those without metabolic syndrome. Furthermore, serum prolactin showed significant inverse correlations with body mass index, waist circumference, fasting plasma glucose, triglycerides, systolic and diastolic blood pressure, while a positive correlation was observed with HDL cholesterol. Multivariable logistic regression further confirmed serum prolactin as an independent predictor of metabolic syndrome after adjustment for age and BMI. These findings support the concept that physiological prolactin may have a protective role in metabolic homeostasis.[7,8]

Our findings are in agreement with the population-based SHIP study by Balbach et al., which included 3,993 adults (2,027 women) and reported that lower physiological prolactin concentrations were associated with adverse metabolic characteristics. Women in the lowest prolactin quartile had a 70% higher risk of prevalent type 2 diabetes than those in the highest quartile (RR 1.70; 95% CI: 1.10–2.62), although the independent association with metabolic syndrome was attenuated after multivariable adjustment.[7] Similarly, Wang et al. followed 8,615 women for up to 22 years and demonstrated that women in the highest prolactin quartile had a 27% lower risk of developing type 2 diabetes compared with those in the lowest quartile (HR 0.73; 95% CI: 0.55–0.95), suggesting that physiological prolactin concentrations contribute to long-term glucose homeostasis.[9] A recent review by Macotela et al. also summarized experimental and clinical evidence indicating that prolactin receptors are expressed in adipose tissue, pancreatic β-cells, liver, and skeletal muscle, where prolactin promotes β-cell survival, improves insulin secretion, enhances adipose tissue function, and reduces metabolic dysfunction when maintained within the physiological range.[8]

The inverse correlations observed in the present study between serum prolactin and BMI, waist circumference, fasting plasma glucose, triglycerides, and blood pressure, together with its positive association with HDL cholesterol, are biologically plausible and consistent with previous reports demonstrating lower prolactin concentrations in metabolically unhealthy individuals.[7,8] Although some disease-specific studies, particularly among women with polycystic ovary syndrome or antipsychotic-induced hyperprolactinaemia, have reported higher prolactin levels in association with obesity and insulin resistance, these findings likely reflect pathological hyperprolactinaemia rather than physiological hormonal variation and therefore are not directly comparable with the present study.[10,11]

Overall, our findings suggest that lower physiological serum prolactin concentrations are associated with an adverse cardiometabolic profile in women of reproductive age and may serve as a useful adjunctive biochemical marker for early identification of individuals at increased metabolic risk. Nevertheless, the cross-sectional design precludes causal inference, and prospective longitudinal studies with larger multicentric cohorts are warranted to clarify the temporal relationship between prolactin and metabolic syndrome.

CONCLUSION

The present study demonstrated that lower physiological serum prolactin levels were significantly associated with metabolic syndrome and its individual components, including increased body mass index, waist circumference, fasting plasma glucose, triglycerides, blood pressure, and reduced HDL cholesterol in women of reproductive age. Serum prolactin also emerged as an independent predictor of metabolic syndrome after adjustment for potential confounders, suggesting its potential role as an adjunctive biochemical marker for early cardiometabolic risk assessment. However, the cross-

sectional design limits causal inference, and the single-center setting with a relatively modest sample size may restrict the generalizability of the findings. Future multicenter prospective studies with larger cohorts and serial prolactin measurements are recommended to establish temporal relationships and evaluate the clinical utility of serum prolactin as a predictive biomarker for metabolic syndrome and related cardiometabolic disorders.

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