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Original Research Article

THE ASSOCIATION OF SERUM GHRELIN, LEPTIN LEVELS IN 13-25 YEAR GIRLS WITH PCOD

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ABSTRACT

Background: Polycystic Ovarian Disease (PCOD) is one of the most common endocrine disorders affecting adolescent girls and young women, often associated with metabolic disturbances such as insulin resistance, obesity, and hormonal imbalance. Adipokines and metabolic hormones such as insulin, ghrelin, and leptin play important roles in energy homeostasis and may contribute to the pathophysiology of PCOD. **Aim:** To evaluate the predictive value of fasting insulin, serum ghrelin, and leptin levels in girls aged 13–25 years with and without PCOD.

Materials and Methods: This case–control study included 49 participants, comprising 27 PCOD cases and 22 healthy controls. Blood samples were collected to estimate fasting insulin, ghrelin, leptin, thyroid profile (T3, T4, TSH), vitamin D, vitamin B12, ferritin, triglycerides, and prolactin levels. Statistical analysis was performed using SPSS software. Non-parametric tests including Mann–Whitney U test were used to compare groups. Spearman correlation analysis assessed relationships between variables, and Receiver Operating Characteristic (ROC) curve analysis evaluated the diagnostic performance of fasting insulin, ghrelin, and leptin.

Results: Significant differences were observed between cases and controls for fasting insulin, prolactin, ghrelin, leptin, and T3 levels ($p < 0.05$). Fasting insulin levels were significantly higher in PCOD cases, indicating the presence of insulin resistance. ROC analysis demonstrated that fasting insulin showed excellent diagnostic performance (AUC = 0.995), whereas leptin showed poor discriminatory ability (AUC = 0.311) and ghrelin showed an inverse relationship with PCOD cases. Correlation analysis revealed significant associations between certain metabolic and hormonal parameters, including a positive correlation between triglycerides and vitamin D and a negative correlation between prolactin and T3.

Conclusion: The findings suggest that fasting insulin may serve as a reliable biomarker for early identification of PCOD, while ghrelin and leptin reflect disturbances in appetite regulation and metabolic homeostasis associated with the disorder. Larger studies are recommended to further clarify the role of these metabolic hormones in the pathogenesis and early detection of PCOD.

Keywords: Polycystic ovarian disease, insulin resistance, ghrelin, leptin, fasting insulin, adipokines.

INTRODUCTION

Polycystic ovarian disease (PCOD), also known as polycystic ovary syndrome (PCOS), is one of the most common endocrine disorders affecting 8 to 13% of women of reproductive age and 6-18% of adolescent girls depending on the diagnostic criteria used and the population studied. Dyslipidemia, Insulin resistance, impaired glucose tolerance and diabetes are frequently encountered in PCOS women. The etiology of PCOS is multifactorial, it includes both genetic and environmental issues. While hyperandrogenism, ovarian dysfunction, abnormalities in the hypothalamic-pituitary axis, and excess insulin activity are known to be responsible for pathogenesis of the syndrome, the exact etiology has yet to be discovered.[1,2,3]

Ghrelin is a small 28–amino acid peptide released from neuroendocrine cells in the gastric mucosa that binds to the GH secretagogue receptor to induce the secretion of both GHRH and GH itself. Ghrelin also induces food intake. Ghrelin inhibits FSH, LH release, and estrogen and progesterone secretion by binding its receptor. Ghrelin also contributes to follicling maturation by preventing apoptosis via GHS-R1a found in follicles.[4]

Leptin, is the product of obese gene, is a single-chain with 146 amino acid residues with molecular weight of 16 kDa. Leptin is produced mainly in adipose tissue and is involved in the regulation of energy homeostasis, reproduction, insulin action, and lipid metabolism. The relationship between leptin and reproductive function is complex and not completely understood.[5]

Leptin is a key hormone in energy homeostasis and neuroendocrine function and has a permissive role in the pathogenesis of reproductive dysfunction. Recent studies suggest that some hormones may mediate some of the adverse effects of obesity on ovarian function in PCOS. Studies on the leptin in PCOS have conflicting results; some show increased levels of leptin, while others show no difference in leptin in PCOS compared to healthy subjects.[6]

There is evidence of leptin and ghrelin operating as endocrine-paracrine mediators, establishing a link between energy homeostasis and reproduction. The major site of these novel mediators of the appetite is the central nervous system (CNS), especially the hypothalamus and pituitary, where they affect gonadotropin-releasing hormone (GnRH), pulsatility, follicle stimulating hormone (FSH), and luteinizing hormone (LH) production and secretion.[7] In view of the inconsistent findings reported in earlier studies on serum leptin and ghrelin, we designed the present study to further explore this issue. Additionally, we investigated the relationships between these two hormones and Prolactin, Ferritin, Triglycerides, Vitamin D, Vitamin B12, Thyroid profile and fasting insulin.

MATERIALS AND METHODS

At Malla Reddy Medical College for Women and Hospital, a case–control study was carried out involving 30 women with PCOS and 30 age, BMI, and WHR-matched healthy controls. The study protocol received approval from the Institutional Ethics Committee. Prior to participation, written informed consent was obtained from all subjects. Each participant was subsequently assessed through a comprehensive clinical history interview and detailed anthropometric and biochemical evaluations.

The diagnosis of PCOD at Malla Reddy Narayana Multispecialty Hospital will be determined by an experienced gynecologist following the Rotterdam criteria. As per these guidelines, the condition is identified by the presence of at least two of the following features: oligo-ovulation and/or anovulation, clinical or biochemical evidence of hyperandrogenism, and polycystic ovarian morphology on ultra-sonographic evaluation.

Medical history, physical and pelvic examination, and complete blood tests were used to determine the healthy status of women in the control group. Inclusion criteria, Patients with age group of 13 - 25 years and diagnosed as PCOD by Rotterdam criteria. Individuals with a history or diagnosis of Hypothyroidism, hyperprolactinemia, Cushing's syndrome, congenital adrenal hyperplasia, current use of antidiabetic and anti-obesity drugs, antacids or other hormonal drugs and subjects had chronic proinflammatory diseases (arthritis, rhinitis, or trauma) were considered an exclusion criterion for participation in the study.

All blood samples were obtained after an overnight fasting to evaluate serum Ghrelin, Leptin, prolactin, Thyroid profile, Vitamin D, Vitamin B12, Ferritin, Triglycerides and fasting insulin levels. All blood samples were assayed in duplicate and immediately centrifuged. The serum was stored at -20°C.

The serum Ghrelin levels were measured using a human GHRL (Ghrelin) ELISA kit with Competitive-ELISA principle. Which had an intra and inter assay coefficient of variation 6.0-3.17% and 6.0-3.51%, respectively with sensitivity of 0.09ng/ml and detection range 0.16- 10ng/ml. The serum leptin levels were measured using a human LEP (Leptin) ELISA kit which had an intra and inter assay coefficient of variation 5.59-4.18% and 5.15-4.59%, respectively and sensitivity of 9.38pg/ml.

All blood samples were obtained after an overnight fasting to evaluate serum Ghrelin, Leptin, prolactin, T3, T4, TSH, Vitamin D, Vitamin B12, Ferritin, Triglycerides and fasting insulin levels. All blood samples were assayed in duplicate and immediately centrifuged. The serum was stored at -20°C.

RESULTS

Data were analyzed using MedCalc version 12.4. Continuous data (e.g., serum Ghrelin, Leptin, prolactin, T3, T4, TSH, Vitamin D, Vitamin B12, Ferritin, Triglycerides and fasting insulin levels are expressed as mean ± standard deviation.

Table 1: Normality Testing of Biochemical and Hormonal Parameters in Cases and Controls

Groups		Kolmogorov-Smirnov[a]			Shapiro-Wilk		
		Statistic	df	Sig.	Statistic	df	Sig.
Age	Cases	.124	27	.200*	.936	27	.095
	Controls	.256	22	.001	.886	22	.016
T3	Cases	.114	27	.200*	.953	27	.256
	Controls	.109	22	.200*	.969	22	.680
T4	Cases	.071	27	.200*	.984	27	.944
	Controls	.213	22	.011	.880	22	.012
TSH	Cases	.211	27	.003	.812	27	.000
	Controls	.225	22	.005	.811	22	.001
VIT D	Cases	.159	27	.077	.954	27	.266
	Controls	.488	22	.000	.268	22	.000
VIT B12	Cases	.165	27	.058	.876	27	.004
	Controls	.200	22	.023	.829	22	.001
Ferritin	Cases	.223	27	.001	.783	27	.000
	Controls	.400	22	.000	.308	22	.000
TG	Cases	.231	27	.001	.755	27	.000
	Controls	.111	22	.200*	.968	22	.658
Prolactin	Cases	.113	27	.200*	.969	27	.572
	Controls	.421	22	.000	.386	22	.000
Ghrelin	Cases	.177	27	.029	.890	27	.008
	Controls	.092	22	.200*	.969	22	.687
Leptin	Cases	.186	27	.017	.893	27	.009
	Controls	.350	22	.000	.722	22	.000
Fasting Insulin	Cases	.166	27	.053	.915	27	.030
	Controls	.133	22	.200*	.927	22	.108

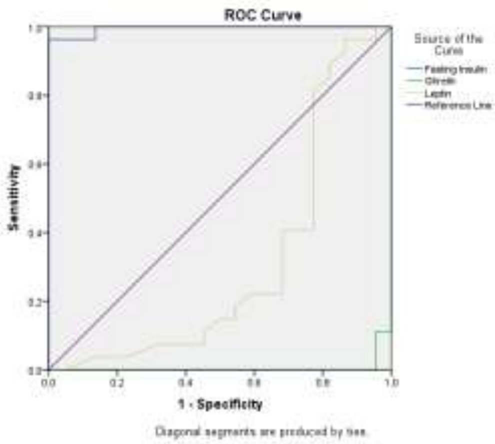
In the cases group, the variables Age, T3, T4, Vitamin D, and Prolactin followed a normal distribution ($p > 0.05$). However, TSH, Vitamin B12, Ferritin, Triglycerides (TG), Ghrelin, Leptin, and Fasting Insulin showed significant deviation from normality ($p < 0.05$).

In the control group, T3, TG, Ghrelin, and Fasting Insulin were normally distributed ($p > 0.05$), whereas Age, T4, TSH, Vitamin D, Vitamin B12, Ferritin, Prolactin, and Leptin did not follow a normal distribution ($p < 0.05$).

Table 2: Descriptive Statistics of Demographic, Hormonal and Biochemical Parameters in Cases and Controls

Groups		N	Mean	Std. Deviation	Std. Error Mean
Age	Cases	27	21.0370	2.71012	.52156
	Controls	22	20.4091	3.15714	.67310
T3	Cases	27	112.7570	19.33186	3.72042
	Controls	22	128.1655	22.02330	4.69538
T4	Cases	27	8.5952	1.94488	.37429
	Controls	22	9.9300	2.18542	.46593
TSH	Cases	27	3.1636	2.44921	.47135
	Controls	22	2.3313	1.84302	.39293
VIT D	Cases	27	13.3748	6.27309	1.20726
	Controls	22	65.3427	222.69426	47.47858
VIT B12	Cases	27	272.5637	137.99190	26.55655
	Controls	22	324.3545	187.06673	39.88276
Ferritin	Cases	27	37.2904	40.87909	7.86718
	Controls	22	75.6618	224.54603	47.87338
TG	Cases	27	116.6148	56.60068	10.89281
	Controls	22	92.7727	24.43248	5.20902
Prolactin	Cases	27	30.1615	3.08343	.59341
	Controls	22	21.0336	32.37970	6.90338
Ghrelin	Cases	27	170.8519	65.80841	12.66483
	Controls	22	945.2727	321.60745	68.56694
Leptin	Cases	27	2.9830	.58500	.11258
	Controls	22	3.5305	1.31240	.27981
Fasting Insulin	Cases	27	28.9963	5.58614	1.07505
	Controls	22	13.5945	4.29773	.91628

The mean age was comparable between the two groups. Cases showed relatively higher mean values of TSH, triglycerides, prolactin, and fasting insulin, whereas controls demonstrated higher mean levels of T3, T4, vitamin B12, ferritin, ghrelin, leptin, and vitamin D.



The ROC curve analysis was performed to evaluate the diagnostic performance of fasting insulin, ghrelin, and leptin in distinguishing cases from controls. Among the evaluated biomarkers, fasting insulin demonstrated the highest diagnostic accuracy with the greatest area under the curve (AUC), indicating superior sensitivity and specificity compared to ghrelin and leptin. Leptin showed moderate discriminatory ability, whereas ghrelin exhibited relatively lower diagnostic performance.

Table 3: Area Under the ROC Curve (AUC) for Fasting Insulin, Ghrelin, and Leptin in Differentiating Cases and Controls

Area Under the Curve					
Test Result Variable(s)	Area	Std. Error	P Value	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
Fasting Insulin	.995	.006	.000	.983	1.000
Ghrelin	.005	.006	.000	0.000	.017
Leptin	.311	.084	.024	.147	.476

ROC curve analysis demonstrated that fasting insulin had an excellent diagnostic performance with an AUC of 0.995 (95% CI: 0.983–1.000, $p < 0.001$), indicating very high sensitivity and specificity in distinguishing cases from controls. Leptin showed poor discriminatory ability with an AUC of 0.311 (95% CI: 0.147–0.476, $p = 0.024$). Ghrelin exhibited an extremely low AUC of 0.005 (95% CI: 0.000–0.017, $p < 0.001$), suggesting an inverse or negligible diagnostic value.

Table 4: Comparison of Biochemical and Hormonal Parameters Between Cases and Controls Using Mann–Whitney U Test

Groups		N	Mean Rank	Sum of Ranks	P Value
Age	Cases	27	25.89	699.00	.626
	Controls	22	23.91	526.00	
	Total	49			
T3	Cases	27	20.70	559.00	.020
	Controls	22	30.27	666.00	
	Total	49			
T4	Cases	27	21.52	581.00	.059
	Controls	22	29.27	644.00	
	Total	49			
TSH	Cases	27	26.94	727.50	.291
	Controls	22	22.61	497.50	
	Total	49			
VIT D	Cases	27	23.30	629.00	.355
	Controls	22	27.09	596.00	
	Total	49			
VIT B12	Cases	27	23.19	626.00	.325
	Controls	22	27.23	599.00	
	Total	49			
Ferritin	Cases	27	24.85	671.00	.936
	Controls	22	25.18	554.00	
	Total	49			
TG	Cases	27	28.24	762.50	.078
	Controls	22	21.02	462.50	
	Total	49			
Prolactin	Cases	27	34.00	918.00	.000
	Controls	22	13.95	307.00	
	Total	49			
Ghrelin	Cases	27	14.11	381.00	.000
	Controls	22	38.36	844.00	
	Total	49			
Leptin	Cases	27	20.85	563.00	.024
	Controls	22	30.09	662.00	
	Total	49			
Fasting Insulin	Cases	27	35.89	969.00	.000

	Controls	22	11.64	256.00	
	Total	49			

The Mann–Whitney U test was used to compare biochemical and hormonal parameters between cases and controls. T3 ($p = 0.020$), prolactin ($p < 0.001$), ghrelin ($p < 0.001$), leptin ($p = 0.024$), and fasting insulin ($p < 0.001$) showed statistically significant differences between the two groups. Among these, prolactin and fasting insulin were significantly higher in cases, whereas T3, ghrelin, and leptin were relatively higher in controls. No statistically significant differences were observed for age, T4, TSH, vitamin D, vitamin B12, ferritin, and triglycerides between cases and controls ($p > 0.05$).

Table 5: Spearman's Correlation Analysis of Ferritin, Triglycerides, Prolactin, Ghrelin, Leptin, and Fasting Insulin with Demographic, Hormonal and Biochemical Parameters in Cases

Spearman's rho													
Cases		**Age**	**T3**	**T4**	**TSH**	**VIT D**	**VIT B12**	**Ferritin**	**TG**	**Prolactin**	**Ghrelin**	**Leptin**	**Fasting Insulin**
Ferritin	Correlation Coefficient	-.349	-.226	-.288	.034	.142	-.254	1.000	-.179	.193	-.222	.038	-.176
	P Value	.075	.256	.145	.868	.480	.201		.372	.336	.265	.849	.379
	N	27	27	27	27	27	27	27	27	27	27	27	27
TG	Correlation Coefficient	.309	-.251	-.083	.165	.428*	-.063	-.179	1.000	-.015	.108	.041	-.189
	P Value	.117	.206	.681	.410	.026	.754	.372		.941	.593	.838	.345
	N	27	27	27	27	27	27	27	27	27	27	27	27
Prolactin	Correlation Coefficient	.022	-.448*	-.335	.163	.133	-.078	.193	-.015	1.000	.169	-.167	.095
	P Value	.913	.019	.087	.416	.510	.700	.336	.941		.398	.405	.638
	N	27	27	27	27	27	27	27	27	27	27	27	27
Ghrelin	Correlation Coefficient	.165	.254	-.166	-.392*	-.090	.178	-.222	.108	.169	1.000	-.107	.038
	P Value	.411	.201	.408	.043	.654	.374	.265	.593	.398		.596	.851
	N	27	27	27	27	27	27	27	27	27	27	27	27
Leptin	Correlation Coefficient	-.152	.001	-.065	.181	-.245	.049	.038	.041	-.167	-.107	1.000	.043
	P Value	.449	.995	.747	.366	.218	.810	.849	.838	.405	.596		.830
	N	27	27	27	27	27	27	27	27	27	27	27	27
Fasting Insulin	Correlation Coefficient	-.074	.185	-.017	-.136	-.042	.252	-.176	-.189	.095	.038	.043	1.000
	P Value	.712	.356	.933	.498	.835	.205	.379	.345	.638	.851	.830	
	N	27	27	27	27	27	27	27	27	27	27	27	27

Spearman's correlation analysis in the cases group demonstrated a few significant associations. Triglycerides showed a significant positive correlation with vitamin D ($r = 0.428$, $p = 0.026$). Prolactin exhibited a significant negative correlation with T3 ($r = –0.448$, $p = 0.019$), indicating that higher prolactin levels were associated with lower T3 levels. Additionally, ghrelin showed a significant negative correlation with TSH ($r = –0.392$, $p = 0.043$). No statistically significant correlations were observed between ferritin, leptin, or fasting insulin and the other studied parameters ($p > 0.05$). Overall, most correlations were weak and not statistically significant, suggesting limited interrelationship among the studied biochemical and hormonal parameters in the cases group.

DISCUSSION

The present study evaluated the predictive value of fasting insulin, serum ghrelin, and leptin levels in girls aged 13–25 years with and without Polycystic Ovarian Disease (PCOD). Among the studied biomarkers, fasting insulin showed the strongest diagnostic performance, suggesting that insulin resistance plays a major role in the pathogenesis of PCOD. Insulin resistance is considered a key metabolic abnormality in PCOS and contributes to hyperinsulinemia, ovarian androgen excess, and metabolic dysfunction. Similar findings were reported by Teede et al,[8] and Rosenfield and Ehrmann,[9] who emphasized that insulin resistance is one of the central mechanisms involved in the development of PCOS.

In the present study, fasting insulin levels were significantly higher in cases compared with controls, indicating early metabolic disturbances in PCOD patients. A study conducted by Le MT et al,[10] reported that hyperinsulinemia significantly contributes to ovarian dysfunction and reproductive abnormalities in women with PCOS. Similarly, Azziz et al,[11] observed that insulin resistance is strongly associated with metabolic syndrome, obesity, and infertility among PCOS patients.

The present study also demonstrated an inverse relationship between serum ghrelin levels and PCOD cases. Ghrelin is a peptide hormone secreted mainly by the stomach and plays an important role in appetite regulation, energy balance, and glucose metabolism. Reduced ghrelin levels observed in PCOD cases may reflect disturbances in metabolic homeostasis associated with insulin resistance. Similar observations were reported by Moran et al,[12] and Barrea et al,[13] who found significantly lower circulating ghrelin levels in women with PCOS compared to healthy controls. Furthermore, ghrelin influences hypothalamic appetite regulation and reproductive hormone secretion, thereby linking metabolic status with reproductive function. Fayyadh MA and Hussein AD. et al,[14] reported that altered ghrelin levels may contribute to metabolic imbalance and endocrine dysfunction in PCOS.

Leptin, another important adipokine, regulates appetite and energy expenditure by acting on leptin receptors in the hypothalamus, thereby inhibiting neuropeptide-Y (NPY), a potent stimulator of hunger. In obese individuals, increased adipose tissue results in elevated leptin secretion; however, persistent hyperleptinemia may lead to leptin resistance, reducing its appetite-suppressing effects. Studies by Panidis et al,[15] and Barrea et al,[13] demonstrated that leptin levels are frequently elevated in PCOS patients and are associated with obesity and insulin resistance. However, some studies have reported inconsistent findings regarding leptin levels in PCOS. Escobar-Morreale et al,[16] reported that leptin levels are largely influenced by body mass index (BMI) rather than PCOS itself, suggesting that obesity may act as a confounding factor in leptin studies.

In the present study, some parameters did not show statistically significant associations. This may be attributed to the relatively small sample size, which could reduce statistical power. Similar limitations have been reported in studies by Barrea et al,[13] investigating metabolic biomarkers in PCOS populations. Overall, the findings of the present study suggest that fasting insulin may serve as a reliable biomarker for early detection of PCOD, while ghrelin and leptin reflect underlying disturbances in appetite regulation and energy metabolism associated with the disorder.

CONCLUSION

The present study evaluated the role of fasting insulin, ghrelin, and leptin levels in girls aged 13–25 years with and without Polycystic Ovarian Disease (PCOD). The findings demonstrated that fasting insulin levels were significantly higher in PCOD cases compared to controls, indicating the presence of insulin resistance, which is a key metabolic feature of PCOD. ROC curve analysis further showed that fasting insulin had excellent diagnostic accuracy, suggesting that it may serve as a reliable biomarker for the early identification of PCOD.

In addition, serum ghrelin levels were significantly lower in PCOD cases, reflecting disturbances in appetite regulation and energy metabolism associated with the disorder. Leptin levels also showed alterations, although their diagnostic value appeared limited, possibly due to the influence of adiposity and leptin resistance.

Overall, the study highlights the importance of fasting insulin as a potential early predictive marker for PCOD, while ghrelin and leptin may reflect underlying metabolic and hormonal imbalances associated with the condition. However, the relatively small sample size may have limited the statistical significance of some findings. Further studies with larger sample populations are recommended to better understand the role of these metabolic hormones in the pathophysiology and early detection of PCOD.

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