RESEARCH PAPER

Correlation of Insulin Resistance with Magnesium and Iron in Patients with Polycystic Ovarian Syndrome -An Observational Case Control Study

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ABSTRACT

Introduction: Polycystic ovarian syndrome (PCOS) is a multifactorial endocrine disorder sturdily associated with insulin resistance (IR) and metabolic dysregulation. Evidence advocates that trace elements such as magnesium and iron curb the action of insulin through enzymatic pathways, oxidative stress , and its role in glucose homeostasis. Our study aims to evaluate the correlation of serum magnesium ,iron and ferritin levels with IR in women with PCOS.

Material and Methods: Our observational case–control study was done on clinically diagnosed PCOS subjects and age-matched healthy controls. Fasting venous blood samples were analysed for serum insulin ,blood glucose lipid profile ,magnesium iron and ferritin levels using standard biochemical methods. Insulin resistance was calculated using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR). Statistical analysis was performed to compare groups and assess correlations between trace elements and HOMA-IR.

Results: Women with PCOS demonstrated significantly elevated HOMA-IR values compared to controls, confirming increased insulin resistance. Significant variation was observed in serum magnesium and ferritin level while iron levels does not show any significant changes between the PCOS women and healthy subjects. Correlation analysis revealed statistically significant association between magnesium and IR.

Conclusion: Despite the established presence of insulin resistance in PCOS, serum magnesium exhibited a significant correlation with insulin resistance while serum iron and storage form does not show a significant association with IR. Our findings propose that the role of iron and ferritin in the pathophysiology of insulin resistance in PCOS might be influenced by other factors.

Keywords: Polycystic ovarian syndrome, Insulin resistance, trace elements

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INTRODUCTION

Polycystic ovarian syndrome is progressively recognized endocrine and metabolic disorder among adolescent female population, twinning lifestyle modification, urbanized social culture leading to increasing adolescent obesity. The prevalence of PCOS has been found to be 10-22% in India, with 8-20% prevalence among south Indian population[1]. Insulin resistance (IR) stood extensively acknowledged as a key pathogenic driver of PCOS and found to manifest at early stage of adolescence. Studies among Indian population have demonstrated a strong association between an increase in body mass index and waning IR in PCOS, pointing to the release of adipokines, impaired insulin signalling, driving to overproduction of androgen and worsening PCOS symptoms[2].

Micronutrient imbalances and nutritional deficiencies also manifest as metabolic dysfunction of PCOS. Magnesium is essential for insulin receptor autophosphorylation and post-receptor signalling; henceforth hypomagnesemia may results in IR. Macro element iron is another latent contributor to insulin resistance in PCOS women mainly due to early onset oligomenorrhea, and insulin-mediated intestinal absorption of iron[3,4]. A deep insight into the effect of BMI, IR and role of micronutrients may support the development of targeted nutritional and lifestyle interventions at mitigating consequences of PCOS in adolescent population. Our study was aimed to the correlate IR with serum level of magnesium, and iron among women with PCOS.

MATERIAL AND METHODS

An observational cross sectional study was conducted on adolescent women of reproductive age 18-45 years who attended the outpatient department of Obstetrics and Gynaecology in Al Azhar Medical college and super speciality hospitals. Women with complaints of unwanted hair growth, irregular menstrual cycles and who fulfilled the Rotterdam 2003 criteria for the diagnosis of PCOS were selected[5]. Women with history of oligomenorrhea, anovulation, Clinical hirsutism (Ferriman Gallwey score ≥8) and Ultrasonographic evidence of polycystic ovaries (presence of 12 follicles or more) and exclusion of congenital adrenal hyperplasia, androgen secreting tumor, Cushing's syndrome were included. The subjects excluded were having history of systemic chronic disorders mainly diabetes, thyroid disease, adrenal, hepatic or cardiovascular disorders, hypertension, PCOS with specific endocrinopathy including hyperprolactenemia, Cushing syndrome, Congenital adrenal hyperplasia and pregnancy. The Institutional ethics committee approval was obtained (IEC No-AAMC/IEC/2023-2024/3-5). After getting informed consent from the subjects, sociodemographic details, anthropometric data including name, physical and clinical characteristics were recorded. Healthy volunteers of age group 18-45 years women with normal reproductive cycle were included as control group(GroupII).

Venous blood samples were collected after 12-hour fast during the early follicular phase of spontaneous or progesterone withdrawal bleeding for complete blood count, Fasting blood glucose (FBS), fasting insulin, lipid profile, iron and magnesium assay. Hormonal evaluation was done on 2nd or 3rd days of menstrual cycle. CBC, serum insulin, lipid profile, FBS, total cholesterol, triglycerides, high density lipoprotein , low density lipoprotein was estimated using gold standard methods[6]. Serum iron was measured using chromazurol method,[7] ferritin by Nephelometric method[8] and magnesium by Xylidyl blue with Advanced Technical Cleaning Solution/Technique (ATCS) The BMI and HOMA-IR was categorized as Optimal: <1, Fair/Normal: 1–2.5 and Insulin Resistance: >2.5[10].

Statistical analysis

The data were entered in Microsoft excel and analysed by using SPSS version 27.0. Continuous variable were represented as mean ± SD and categorical variables were expressed as frequency and percentage. Normality of continuous variables was assessed using the Shapiro–Wilk test. As the variables were found to be approximately normally distributed, Pearson's correlation coefficient was used to evaluate the relationship between serum iron, ferritin, and magnesium levels with HOMA-IR. Unpaired t test was used to compare the mean difference between healthy subjects and control groups. A p value < 0.05 was considered statistically significant.

RESULTS AND DISCUSSION

Table 1: Socio demographic details of the study population (n=86)

Parameter	PCOS group (n= 43) Mean ± SD	Control group (n= 43) Mean ± SD	t	df	p value
Age (years)	23.12±3.76	22.07±3.81	1.61	84	0.867

BMI (kg/m²)	23.8±3.92	21.97±1.56	2.93	84	0.004*
Systolic Blood Pressure (mmHg)	123.02±4.64	119.53±2.13	4.47	84	<0.001*
Diastolic Blood Pressure(mmHg)	80.0±0.00	79.53±2.13	1.43	84	0.156
HOMA IR	12.69±17.78	0.822±1.02	4.36	84	<0.001*
Serum Total cholesterol (mg/dl)	173.81±28.9	171.7±16.8	0.401	84	0.689
Serum Triglyceride (mg/dl)	120.45±47.75	103.01±29.72	2.03	84	0.04*
Serum HDL cholesterol (mg/dl)	48.93±8.86	48.91±8.5	0.01	84	0.992
Serum LDL cholesterol (mg/dl)	107.12±22.33	83.94±12.46	5.94	84	0.001*
Serum VLDL (mg/dl)	24.09±9.55	20.60±5.94	2.03	84	0.04*
Serum Iron(ug/dl)	73.29±38.09	73.11±22.81	-0.022	84	0.983
Serum Ferritin (ng/ml)	82.8±36.10	37.34±14.61	7.66	84	<0.001*
Serum Magnesium (mg/dl)	1.6±0.474	2.13±.307	-6.04	84	<0.001*

P value <0.05 was significant

The mean age of PCOS women Group I was 23.12 ± 3.76 compared to 22.07 ± 3.81 years for Group I. Significant difference was observed in BMI between the two groups (23.8±3.92 vs 21.97±1.56 kg/m²;at p value 0.004). Systolic blood pressure was significantly higher among Group I compared to Group II (123.02±4.64 vs 119.53±2.13mmHg; at $p < 0.001$). However, diastolic blood pressure did not differ significantly between the groups ($p = 0.156$).

Fasting insulin and HOMA IR was found to be significantly high in PCOS women. Serum ferritin level was significantly higher in the PCOS group compared to the Group II ($p < 0.001$). No statistically significant differences were observed between the groups with respect to total cholesterol, HDL, and serum iron, levels ($p > 0.05$). Table II showed that around 50% of PCOS population had IR ,2.3% were in early stage of IR , while 47.7% were within in the normal limits.

Figure I: Homa IR Classification

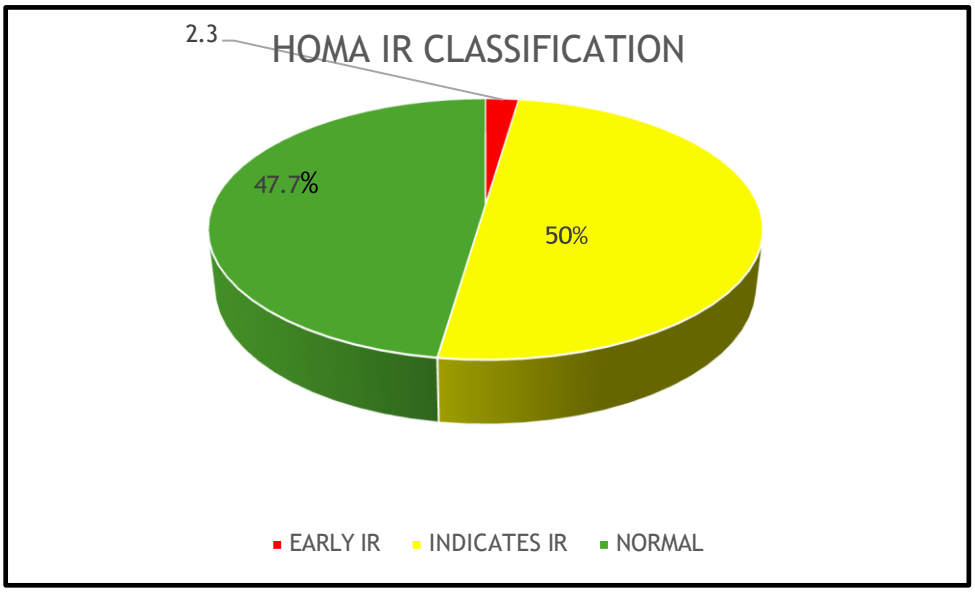


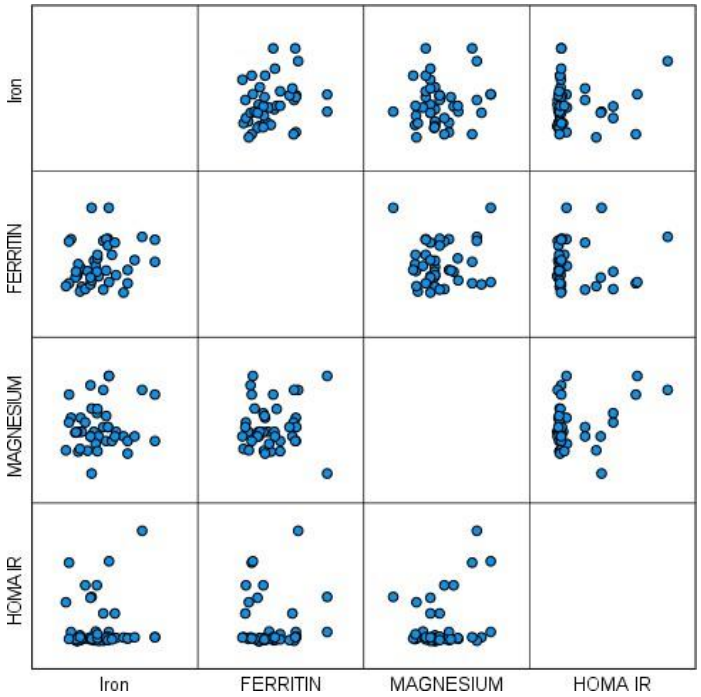
Table:II. Correlation between HOMA IR, Serum Iron, Ferritin and Magnesium in Group I and Group II

PCOS Group (Group I)				**Control Group (Group II)**		
HOMA IR	Mean± SD	r	p value	Mean± SD	r	p value
Iron	73.29±38.09	0.039	0.806	73.4±22.8	0.363	0.01*
Ferritin	82.85±36.11	-0.004	0.977	37.3±14.61	0.081	0.608
Magnesium	1.6±0.474	0.331	0.03*	2.13±0.307	0.302	<0.04*

Homeostatic Model Assessment of Insulin Resistance; Iron,Ferritin,Magnesium.*significant ($p<0.05$).

In Group I serum iron and ferritin levels did not show any significant correlation with insulin resistance while serum magnesium showed a significant assosciation with IR. In Group II, serum iron and magnesium showed a strong and significant positive correlation with HOMA-IR, while ferritin was not having a significant association with IR.

Figure II. The scatter plot matrix between HOMA-IR and serum ferritin, iron, and magnesium in PCOS .



PCOS is a heterogeneous endocrine condition characterized by hyperandrogenism, dysfunctional ovulation, and polycystic ovarian morphology, accompanied by metabolic irregularities expressed as IR and obesity[11]. The prime mechanisms leading to IR are epigenetically mediated dysregulation of metabolic pathways, genetic modifications, elevated androgenesis, impaired insulin signal transduction, inflammation and increased body mass. A review analysis by Amisi in 2022 states IR as the most promising metabolic feature in 35%-80% of PCOS women, independent of body fat and mass index (BMI). The exact cause of IR in PCOS is still unclear and the prime mechanism needs to be explicated[12].

Insulin is a peptide receptor-binding hormone released by pancreatic beta cells, which binds to cell surface receptors. INSR is a heterotetramer with extracellular α- and β-subunits. The α-subunit initiates binding to the ligands while β-subunit regulates the tyrosine kinase activity across the cell membrane. Binding of insulin to receptors induces specific autophosphorylation of tyrosine, which inturn phosphorylates intracellular IRS1-4, SRC homologues, and collagen homologues (Shc) [13].This is followed by a complex intracellular cascade that inductes insulin signal transduction. Metabolic signalling pathway mediated through phosphatidylinositol 3-kinase (PI3-K) and serine/threonine kinase Akt/protein kinase B (PKB), stimulates glucose uptake by stimulating translocation of GLUT4 from intracellular vesicles to the cell surface The net result has been found to be inactivation of serine phosphorylation of glycogen synthase kinase 3 (GSK3), increased synthesis of glycogen, fatty acid, and protein. Also, mammalian target of rapamycin (mTOR) gets activated regulating protein synthesis and degradation. The second signal pathway being the mitogen-activated protein kinase-extracellular signal-regulated kinase (MAPK-ERK) pathway, which gets activated by insulin receptor-mediated phosphorylation of Shc or IRS. This progressively stimulates the translocation of cascade erk1/2 to the nucleus and phosphorylates transcription factors to stimulate cell growth and differentiation and regulate gene expression. Increased serine phosphorylation, decreased tyrosine phosphorylation of IR and IRS results in termination of insulin action, resulting in post-binding defects in insulin signal transduction and leading to insulin dysfunction in adolescent women with PCOS [14].

IR with hyperinsulinism underwrite to androgenesis in PCOS and the co-gonadotropin action of insulin on ovary, ease androgen secretion. Previous studies have proved that insulin stimulates androgenesis in normal ovarian in vitro models. PCOS thecal cells have high androgen responsiveness to insulin compared to the normal thecal cells. This may happen even with normal or reduced action of insulin in PCOS condition. Various studies have shown other theories for a deep understanding of PCOS indices, including primary enzymatic default in adrenal and ovarian steroidogenesis. Defective gonadotropin-

releasing hormone excretion indorsing leuterising hormone emission , altered insulin movements resulting in IR are other features [15].Glucose and insulin metabolism in PCOS are strongly related to mineral imbalances, with evidences of decreased magnesium (Mg) and reliable evidences for the potential variation in iron (Fe), copper (Cu), and calcium (Ca) levels, contributing to IR . Mg deficiency has been found to reduce glucose uptake and impairs insulin action[16, 17]. In our study, statistically significant correlation was observed between serum magnesium levels and IR with r value 0.331 and p value 0.03 for magnesium. Mg a perilous mineral, a cofactor for major enzymes involved in regulating nucleic acid synthesis, carbohydrate, lipid and protein metabolism, integrity of cell membrane.

Studies indicate that chronic Mg deficiency might be manifested with a high risk of various clinical manifestations including obesity, insulin IR, T2DM, hypertension (HTN), changes in lipid metabolism, and high risk of cardiovascular diseases[18]. The pathophysiology behind was altered lipid metabolism, cardio vascular manifestations are chronic low-grade inflammation. Pro inflammatory cytokines induces intracellular shift and uptake of magnesium. IR mediated renal loss of magnesium due to decreased tubular reabsorption and increased excretion might be the another reason for hypomagnesemia. Magnesium acts as cofactor for synthesis and regeneration of **glutathione** , converting superoxide radicals ,modulates the expression of Nrf2 Hypomagnesemia may result from inadequate dietary intake in concurrence with obesity-associated metabolic alterations in PCOS women.

The studies have proved the protective role of Magnesium by improved glucose homeostasis, enhancing endothelium-dependent vasodilation, and maintaining a normal lipid profile. Any deficiency or hypomagnesemia inturn impairs insulin signalling due to defective tyrosine kinases activity of insulin receptor, leading to IR. Inflammaging process and oxidative damage results in IR and worsen PCOS. Revathi *et al*. showed that serum levels of Cu and Zn were significantly higher while Mg level was low in PCOS patients than the control group. A meta analysis study done by Yin *et al* in 2020 briefed the potential role of trace elements in PCOS occurrence and development and hence would give effective new strategies to prevent, screen and treat PCOS. Li *et al*. in a Chinese cohort study reported no significant differences in the levels of serum Zinc, Mg, and iron between PCOS and healthy control groups. A meta-analysis conducted by Spritzer and co-workers in 2015 did not offer a robust conclusion. However, most of these studies had some limitations, including lack of consistency in nutrient and formulations or dosages, different diagnostic criteria or cut-offs and outcome measures.[19,20]

Serum ferritin level has been found to be significant at p <0.001in adolescent PCOS women indicative of low-grade chronic inflammation, insulin resistance, and iron storage. The pathophysiology for this may be metabolic dysfunction and amenorrhea, than an increased uptake of iron. In our study no statistically, significant correlation was observed between serum iron, ferritin, levels and IR with r value -0.004 and p value 0.977 for iron and r value 0.039, p value 0.806 for ferritin.

Iron is a vital macro element, its metabolism is inflexibly maintained at the cellular and systemic levels. To maintain its homeostasis it is regulated at dietary level or at level of absorption. Ferritin, the storage form is the major indicator of balance of iron at cellular level and gets altered in chronic inflammatory metabolic disturbances and cancer. Body iron stores interrupt signalling pathways, induce cellular damage directly or depleting cellular energy stores due to oxidative stress, and impairs action of insulin.[21] Excess iron in tissues promote the reaction of free ferrous iron with hydrogen peroxide to produce reactive hydroxyl radicals. The consequence being disruption of cellular redox homeostasis and oxidative DNA damage which in turn activate inflammatory pathways that interfere glucose uptake. A previous study on iron metabolism and IR proved that elevated iron levels might interfere with adipocyte differentiation and lipid metabolism, reducing adiponectin levels and thereby promoting macrophage inflammation and suppress osteocalcin secretion. Hence, adiponectin secretion from adipose tissue gets impaired. The bidirectional relationship between iron and insulin resistance proves hyperinsulinemia increases iron accumulation,[22] creating a vicious cycle thereby high insulin stimulates ferritin production, leading to iron retention and decreased hepcidin levels, which, in turn, increases iron absorption and retention, worsening iron overload. On the other hand there is not enough data about the relationship between serum ferritin concentrations in PCOS and the severity of insulin resistance and their clinical features including severity of irregular menstruation. Diabetes Care in 2005 explained that body iron stores are increased in overweight and obese women with polycystic ovary syndrome. Héctor *et al* also found elevated ferritin in overweight and obese women with PCOS compared to lean subjects and concluded that an increased iron stores might pave way for insulin resistance and β-cell dysfunction.[23] This finding were consistent with that of Hitha *et al*. [24], where a positive association was observed among ferritin and metabolic parameters in obese women. Serum iron level was observed to be high among PCOS subjects, but the data were not significant. Studies have proved that in PCOS ferritin being an acute phase reactant gets elevated independent of iron overload. In PCOS the lack of association might be due to the variations in hepcidin

levels resulting from inflammation or hormonal factors which inturn disturb the metabolic action of serum iron and ferritin [24].

Previous literature showed that few studies estimated the serum Fe concentrations in patients with PCOS while several studies focused on ferritin concentrations.[25] The scatter plot matrix showed a random distribution of points without a clear linear pattern, indicating weak relationships between HOMA-IR and serum ferritin and iron in PCOS cases.

CONCLUSION

Our observational case–control study establishes that IR is highly prevalent among women with PCOS, reinforcing its key role in the pathophysiology of the endocrine condition. Body mass index showed a positive association with IR, underscoring the contribution of adiposity to metabolic dysregulation in PCOS. Our study showed a weak inverse relationship of serum magnesium with IR, portentous a potential protective role. Serum iron levels did not show a significant correlation with IR, while serum ferritin levels were significantly elevated in PCOS patients compared to healthy controls, pointing the impaired insulin signaling pathway leading to hyperinsulinemia with an altered iron metabolism.

Hence, these research points out the need for comprehensive metabolic evaluation, anthropometric and micronutrient level assessment in the therapeutic and prognostic path of PCOS. Early identification of these condition and targeted intervention addressing obesity and micronutrient imbalances might improve the physical and emotional well being of the individual. It also improves insulin sensitivity and in turn can reduce the long-term metabolic complications and congestive heart failures pertained to PCOS.

Limitation

Women with PCOS often shows dietary deficiency of micronutrients including magnesium, potassium, folate and iron compared with women without PCOS. Our study failed to assess dietary supplementation, while many other studies considered dietary supplementation as exclusion criteria. Further large-scale studies may be beneficial to elucidate the role of nutrients in PCOS .

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