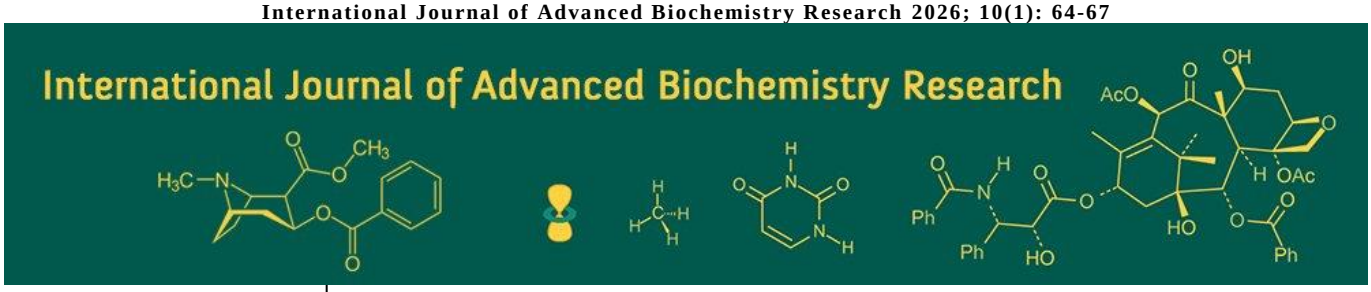

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The impact of insulin resistance on metabolic syndrome among women with polycystic ovary syndrome in Basrah Governate, Iraq

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Abstract
Polycystic ovary syndrome (PCOS) is one of the most prevalent metabolic disorders often coexist with other metabolic disturbances, including metabolic syndrome (MetS). Women with PCOS are at increased risk of insulin resistance (IR), dyslipidemia, and cardiovascular disease (CVD). The aim of study was to evaluate the impact of IR on MetS among females with PCOS in Basrah, Iraq. A case-control study was conducted at Basrah Maternity and Children's Hospital between June, 2024 throughout September, 2025. A total of 100 women with PCOS identified by Rotterdam criteria and 100 age-matched control women were enrolled. Anthropometric, physiological, and biochemical parameters including body mass index (BMI), waist circumference (WC), blood pressure (BP), fasting plasma glucose (FPG), insulin, triglycerides (TG) and high-density lipoprotein-cholesterol (HDL-C) were measured. in addition, the degree of IR (by HOMA-IR) and β-cell function (by HOMA-B) was measured. The presence of MetS was confirmed by the updated NCEP ATP III criteria. The frequency of MetS was significantly higher among women with PCOS (48%) compared to normal women (21%), $p<0.001$. The frequencies of MetS components were significantly elevated among patients with PCOS than in control women (82% vs. 49% for WC), (38% vs. 5.0% for FPG), (25% vs. 10% for BP), (72% vs. 31% for TG), $p<0.001$ and (53% vs. 33% for HDL-C), $p<0.01$. In addition, the frequencies of both HOMA-IR and HOMA-B showed significant elevation among patients with PCOS (3.53 and 113.9 respectively) than normal women (1.25 and 51.23 respectively), $p<0.001$. In conclusion, MetS is highly prevalent among women with PCOS in Basrah. These findings highlight the need for regular metabolic screening and appropriate intervention to reduce the risk long-term CVD sequel.

Keywords: Polycystic ovary syndrome, metabolic syndrome, insulin resistance, cardiovascular disease

Introduction
Polycystic ovary syndrome (PCOS) is considered among the most prevalent metabolic syndromes affecting women in reproductive age, with a estimated prevalence of about 5-15%. It is manifested by the existence of clinical or biochemical hyperandrogenism, anovulation and polycystic ovarian morphology [1]. Beside reproductive abnormalities, PCOS is strongly linked to a variety of metabolic disturbances, including insulin resistance (IR), overweight and obesity, dyslipidemia, non-alcoholic fatty liver disease (NAFLD and a high risk of metabolic syndrome (MetS) [2-6]. Mets is as a cluster of several cardio-metabolic risk factors, including central obesity, hypertension, impaired glucose tolerance or diabetes, hypertriglyceridemia, and reduced high density lipoprotein-cholesterol (HDL-C) [7]. Females with PCOS are at substantial risk of having MetS, which predisposes to the development of type 2 diabetes (T2D) and cardiovascular disease (CVD) and their adverse consequences [8-10]. The frequency of MetS among women with PCOS is highly variable among different populations, with figures ranging from 8% to 47% [11]. Hyperinsulinemia (HI) it is metabolic disturbance existing in women with PCOS, representing a secondary phenomenon to the occurrence of IR in which tissues become insensitive or less sensitive to the effects of insulin. This in turn, resulting in an increased insulin production by the pancreas as a compensate mechanism [12]. The increased insulin secretion leads to a diverse manifestation, including weight gain, menstrual disturbances, in addition to increased risk of T2D [13]. Furthermore, HI accentuates the hormonal abnormalities in PCOS, notably androgen hypersecretion, leading to hirsutism, acne and alopecia [12].

The existence of IR in association with PCOS has also been implicated in the increased risk of CVD [14]. IR is critical metabolic derangement in the development and progression of MetS in PCOS [15]. The aim of the present work was to determine the influence of IR on MetS among females with PCOS in Basrah, Iraq.

Methodology

A case-control study was carried out at Basrah Maternity and Children's Hospital, Basrah, Iraq from June, 2024 to September, 2025. It included 200 women, comprising 100 patients diagnosed with PCOS in accordance to the Rotterdam criteria which needs the existence of two out of three findings, oligo-ovulation or anovulation, biochemical and/or clinical hyperandrogenism, and polycystic ovaries [16]. In addition, 100 age-matched healthy controls. Exclusion criteria include thyroid dysfunction, hyperprolactinemia, Cushing's syndrome, androgen-secreting tumors and congenital adrenal hyperplasia. Physiological and anthropometric parameters including body mass index (BMI), waist circumference (WC), systolic blood pressure (SBP) and diastolic blood pressure (DBP), and biochemical parameters, fasting plasma glucose (FPG), insulin, triglycerides (TG) and high-density lipoprotein-cholesterol (HDL-C) were measured. IR was measured via the HOMA-IR index, while pancreatic β-cell function was quantified by HOMA-B using on the following equations [17, 18]:

HOMA-IR = FPG X Insulin/405
HOMA-B = 360 X Insulin/FPG-63

Where, glucose is measured in mg/dL with insulin in µIU/ml. The normal value of HOMA-IR ≤ 2.5, while the normal value of HOMA-B = 100%.

MetS was identified using the updated NCEP ATP III definition which needs the existence of three or more of the following: WC ≥88 cm, TG ≥150 mg/dL, HDL-C <50 mg/dL, BP ≥135/85 mmHg or the treatment of hypertension, or FPG ≥100 mg/dL or treatment of diabetes [7].

Results

Table 1, presents the physiological and anthropometric characteristics of females with PCOS and control females. Patients with PCOS had significantly elevated BMI, WC, DBP, and SBP than control women, $p<0.001$.

Table 1: Physiological and anthropometric characteristics

Characteristic	Patients with PCOS (n = 100)	Control group (n = 100)	P-value
Age (year)	27.20±5.65	26.90±5.85	0.71
BMI (kg/m^2)	27.06±2.24	24.04±2.17	<0.001
WC (cm)	89.05±4.37	85.28±3.70	<0.001
SBP (mm.Hg)	128.31±13.69	119.15±15.36	<0.001
DBP (mm.Hg)	90.69±3.24	79.33±3.49	<0.001

Data are expressed as Mean ± SD

As illustrated in Table 2, the frequency of MetS was significantly elevated among women with PCOS patients compared to normal women with frequencies of 48% and 21% respectively, $p<0.001$.

Table 2: Frequency of metabolic syndrome

MetS	Patients with PCOS (n = 100)		Control group (n = 100)		P-value
Present	48	48 %	21	21%	<0.001
Absent	52	52%	89	89%	

Table 3, presents the frequencies of MetS components among the study groups, where these components were significantly higher among patients with PCOS compared to normal women (82% vs. 49% for WC), (38% vs. 5.0% for FPG), (25% vs. 10% for BP), (72% vs. 31% for TG), $p<0.001$ and (53% vs. 33% for HDL-C), $p<0.01$.

Table 3: Frequency of metabolic syndrome components

MetS component	Patients with PCOS (n = 100)		Control group (n = 100)	
	No.	%	No.	%
WC (cm)	82**	82%	49	49%
FPG (mg/dL)	38**	38%	5.0	5.0%
BP (mm.Hg)	25**	25%	10	10%
TG (mg/dL)	53*	53%	33	33%
HDL-C (mg/dL)	72**	72%	72	31%

*: $p<0.01$, **: $p<0.001$

The frequencies of FPG, insulin, HOMA-IR and HOMA-B were significantly higher among patients with PCOS than in control women, $p<0.001$. The comparative frequencies of both HOMA-IR and HOMA-B among patients with PCOS and control women were (3.53 and 113.9) vs. (1.25 and 51.23), Table 4.

Table 4: FPG, insulin, HOMA-IR and HOMA-B

Parameters	Patient with PCOS (n = 100)	Control group (n = 100)	P-value
FBS (mg/dL)	121.3±12.9	101.9±12.2	<0.001
Insulin (µIU/ml)	12.9±7.61	5.04±0.98	<0.001
HOMA-IR	3.53±2.26	1.25±0.27	<0.001
HOMA-B	113.9±69.82	51.23±20.83	<0.001

Data are expressed as Mean ± SD

Discussion

Several studies have been conducted in Basrah toward MetS and PCOS and their metabolic components [19-21]. This study revealed that nearly half of females with PCOS in Basrah met the diagnostic criteria for MetS, a prevalence considerably higher than in control women. The prevalence of MetS among women with PCOS demonstrate marked variation world-wide. This is attributed to various factors including age, ethnicity, age, in addition to the diagnostic criteria of MetS [22-26]. Ehrmann *et al.* [27] found a frequency of MetS among patients with PCOS of 46%, and it was strongly associated with the existence of IR and obesity. In addition, Lim *et al.* [28] found that patients with PCOS had a 3.35-fold elevated risk of MetS than normal women. Other studies reported lower figures of MetS than the frequency illustrated in the present study [29-31]. Khorshidi *et al.* [29]. in a

meta-analysis found an overall frequency of MetS in PCOS was approximately 30%, with considerable regional variation with higher figures in Middle East and South Asia. Local differences in aspects such as lifestyle habits, cultural, social and economic issues, in addition to the degree of urbanization all affect the development of MetS. In accordance to reports, females with PCOS were more likely to experience negative impact with regard to CVD risk factors, such as obesity, MetS presence, and hypertension [28]. Women with PCOS, both adults and adolescents, had substantial risk of developing MetS in comparison to normal women [32]. IR is an essential feature in the diverse pathophysiological mechanisms of PCOS. IR and the consequent HI accentuate ovarian androgen production with the concomitant reduction in hepatic sex hormone binding globulin. This results in exaggerating hyperandrogenism. IR is more prevalent in women with PCOS, with a prevalence ranging 12% to 60%. This is wide variation may be explained by the use of different cutoffs points in different tests among various populations [33]. Obesity potentiates IR and the secondary HI, which in turn enhances adipogenesis while inhibit lipolysis. In addition, Obesity causes sensitization of ovarian thecal cells to the enhancement of Luteinizing hormone and, also augments hyperandrogenism by up-regulating androgen secretion. Furthermore, Obesity promotes inflammatory adipokines production which, in turn, aggravate HI, which further increases obesity [34]. Both visceral obesity and IR are considered essential in the pathogenesis of MetS among women with PCOS, potentiating dyslipidemia, hypertension, and impaired glucose metabolism [16]. The presence of both central obesity in association with PCOS, in terms of increased WC, and IR, manifested by increased HOMA-IR, observed in the present study are consistent with critical role of visceral obesity and IR in the development and progression of metabolic dysfunction seen in PCOS. The high prevalence of obesity and adverse lipid profile among females with PCOS in the present study may explain the elevated frequency of MetS. Ganie *et al.* [30] reported a prevalence of MetS among PCOS women of 24.9%, with higher frequency among obese women. In addition, Moini *et al.* [31]. found that 22.7% of PCOS women fulfilled the MetS criteria based on ATP III definition, with central obesity and reduced HDL-C are the most prevalent MetS components. IR in PCOS results from defect in insulin action in several target tissues in the body. This is manifested by compensatory HI and a decreased insulin response secondary to glucose load. The effects of PCOS affected extend to wide variety of body tissues and systems. Insulin exerts different effects in diverse body tissues with regard to availability and balance of nutrients. In women with PCOS, IR preferentially affects the metabolism in common insulin target tissues such as the hepatic tissues, skeletal muscle, and adipocytes, beside other organs such as the ovaries and pituitary [35]. Moreover, androgen overproduction, excessive lipid accumulation, release of inflammatory cytokines is implicated in IR exerted in peripheral sites. In both normal women and those with PCOS, the most determinant of β-cell function is IR. This is compatible with the event of compensatory insulin hypersecretion in response to IR. Secondary HI is to maintain normoglycemia to counteract IR. However, with the persistence of IR, women with PCOS and IR are at a considerable risk of β-cell exhaustion and the subsequent development of T2D [36].

Conclusion

Women with PCOS demonstrated substantially higher prevalence of Mets. Therefore, these women are at a considerable risk of T2D and CVD and their adverse consequences. These findings focus light on the necessity of regular screening and follow up of women with PCOS for the detection and management of the associated adverse metabolic disorders with particular emphasis to Mets and its components.

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