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Article

Prevalence and Clinical Patterns of Polycystic Ovary Syndrome among Reproductive-Age Women Attending Babylon Educational Hospital for Gynecology and Pediatrics, Iraq

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Abstract: Background: Polycystic Ovary Syndrome (PCOS) is the most prevalent endocrine disorder affecting women of reproductive age, yet robust epidemiological data from Iraq remain sparse. Babylon Governorate, a densely populated central Iraqi province, has not previously been the subject of a dedicated hospital-based PCOS prevalence study. **Objectives:** To determine the prevalence of PCOS and to characterize its clinical, hormonal, and sonographic patterns among reproductive-age women attending Babylon Educational Hospital for Gynecology and Pediatrics, Hilla, Iraq. **Methods:** A cross-sectional descriptive study was conducted between January 2025 and December 2025. A total of 450 women aged 18–45 years attending the outpatient gynecology clinic were enrolled using a systematic random sampling approach. Diagnosis of PCOS was established according to the revised Rotterdam criteria (2003), which require at least two of the following three features: oligo-ovulation or anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology on pelvic ultrasound. Standardised clinical assessment, hormonal profile, fasting glucose and insulin, lipid panel, and transvaginal or transabdominal pelvic ultrasound were performed for all participants. **Results:** PCOS was diagnosed in 82 out of 450 women, yielding a prevalence of 18.2% (95% CI: 14.8%–22.1%). The mean age at diagnosis was 24.7 ± 5.3 years. The most prevalent diagnostic subtype was subtype B (oligo/anovulation + polycystic ovarian morphology, no biochemical hyperandrogenism) observed in 34.1% of PCOS cases, followed by subtype A (all three criteria) in 29.3%. Oligomenorrhea was the dominant menstrual complaint (73.2%). Clinical hyperandrogenism, manifested as hirsutism, acne, or androgenic alopecia, was present in 58.5% of PCOS women. Insulin resistance, defined by a HOMA-IR ≥2.5, was detected in 56.1% of diagnosed cases. Dyslipidaemia was identified in 48.8%, and the prevalence of metabolic syndrome among PCOS women was 24.4%. **Conclusions:** PCOS is highly prevalent among reproductive-age women in Babylon Governorate, with a hospital-based prevalence exceeding estimates reported for the general Iraqi population. The high burden of metabolic comorbidities underscores the urgent need for integrated screening, early diagnosis, and multidisciplinary management protocols within Iraqi healthcare settings.

Keywords: polycystic ovary syndrome; prevalence; Iraq; Babylon; hyperandrogenism; insulin resistance; Rotterdam criteria; reproductive-age women; metabolic syndrome

Introduction

Polycystic Ovary Syndrome (PCOS) is widely recognised as the most common endocrine and metabolic disorder affecting women of reproductive age, with a global prevalence estimated to range between 5% and 20% depending on the diagnostic criteria applied and the population studied [1][2].

First described by Stein and Leventhal in 1935 as a triad of amenorrhoea, hirsutism, and bilateral polycystic ovaries, our understanding of PCOS has evolved dramatically over subsequent decades [3]. The condition is now recognized as a complex, heterogeneous endocrinopathy characterised by the interplay of androgen excess, disordered gonadotrophin secretion, insulin resistance, and chronic low-grade inflammation [4][5].

The introduction of the revised Rotterdam Consensus criteria in 2003 by the European Society of Human Reproduction and Embryology (ESHRE) and the American Society for Reproductive Medicine (ASRM) represented a pivotal milestone in PCOS diagnostics [6]. These criteria require at least two of the following three features for a diagnosis: (i) oligo-ovulation or anovulation; (ii) clinical or biochemical signs of hyperandrogenism; and (iii) polycystic ovarian morphology (PCOM) on ultrasound. Compared to earlier National Institutes of Health (NIH) criteria of 1990 and the subsequent Androgen Excess Society (AES) criteria of 2006, the Rotterdam framework identifies a broader spectrum of PCOS phenotypes, enabling the recognition of patients who lack frank androgen excess [7][8].

Epidemiologically, reported PCOS prevalence varies substantially across geographic regions, ethnic groups, and the clinical setting in which women are recruited. Using Rotterdam criteria, community-based studies have yielded estimates of 10–13% in European populations and 15–20% in certain South and Southeast Asian populations [9][10][11]. A landmark meta-analysis by Bozdag and colleagues in 2016 encompassing 49 studies reported a pooled global prevalence of 10% (95% CI: 9%–10%) using Rotterdam criteria across unselected populations [12]. A subsequent systematic review by Siddiqui et al. highlighted significant between-study heterogeneity attributable to differences in recruitment settings, age ranges, body mass index distributions, and diagnostic thresholds [13].

The clinical consequences of PCOS extend far beyond reproductive dysfunction. Women with PCOS carry a significantly elevated lifetime risk of type 2 diabetes mellitus, with some estimates suggesting a fivefold increased risk compared to age-matched controls [14]. Insulin resistance is identified in 50–75% of PCOS women regardless of body weight, and represents a central pathophysiological driver linking ovarian androgen excess to metabolic complications including dyslipidaemia, hypertension, and non-alcoholic fatty liver disease [15][16]. Women with PCOS also demonstrate a twofold higher risk of metabolic syndrome compared to age- and BMI-matched women without the condition [17]. Beyond metabolic concerns, the psychological burden of PCOS is substantial: anxiety, depression, and diminished health-related quality of life are disproportionately prevalent in this population [18].

In the reproductive domain, PCOS is the leading identifiable cause of anovulatory infertility globally, accounting for approximately 70–80% of cases [19]. The condition is also associated with elevated risks of gestational diabetes mellitus, pregnancy-induced hypertension, preterm birth, and miscarriage, implicating PCOS management in maternal and neonatal health outcomes [20][21]. Endometrial cancer risk is estimated to be threefold higher in women with PCOS, attributable primarily to chronic anovulation and unopposed oestrogen exposure [22].

In the Middle Eastern and Arab context, where dietary patterns, rates of obesity, and genetic backgrounds differ from Western populations, PCOS prevalence and clinical phenotype may exhibit

distinctive characteristics [23]. Epidemiological studies from neighbouring countries have reported divergent findings: a prevalence of 17.8% in Saudi Arabia using Rotterdam criteria, 12.3% in Kuwait, and 14.6% in Jordan [24][25][26]. Within the Arab world, consanguineous marriages, high-carbohydrate dietary habits, physical inactivity, cultural constraints on outdoor activities for women, and rapidly rising rates of obesity may collectively exacerbate the metabolic expression of PCOS [27].

Iraq, as one of the most populous nations in the Arab world, has a substantial reproductive-age female population that may be significantly affected by PCOS. Despite this, epidemiological data from Iraq on PCOS prevalence and clinical patterns remain fragmented and limited. The few published studies from Iraq have largely been single-centre reports from Baghdad or Mosul, often employing heterogeneous diagnostic criteria, small sample sizes, or selected clinical subgroups such as infertile women, rendering their findings not representative of the broader female population [28][29].

A study conducted at Al-Yarmouk Teaching Hospital, Baghdad, by Al-Rubaie et al. (2018) reported a PCOS prevalence of 15.4% among women attending a fertility clinic using NIH criteria, which would underestimate true prevalence relative to Rotterdam-based estimates [29]. Conversely, Hameed and Talib (2020), using Rotterdam criteria in a Basra-based sample, identified PCOS in 19.1% of reproductive-age women attending a general gynaecological clinic, suggesting a potentially higher burden in southern Iraqi governorates [30].

Babylon Governorate, located in central Iraq with a population exceeding 2.2 million, lacks dedicated epidemiological data on PCOS. Babylon Educational Hospital for Gynecology and Pediatrics (BEH-GP) in Hilla city is a major tertiary referral centre serving not only Babylon Governorate but also the surrounding provinces of Karbala, Wasit, and Najaf. Despite its role as a major reproductive health facility, no systematic study has examined PCOS burden among women presenting to this institution.

The 2023 International Evidence-Based Guideline for the Assessment and Management of PCOS, developed by Teede and colleagues, calls for locally relevant epidemiological data to guide resource allocation, training of healthcare providers, and the design of population-specific management pathways [31]. In the Iraqi context, where primary healthcare infrastructure is still recovering from years of conflict and economic disruption, accurate local prevalence data are indispensable for policy formulation.

From a public health perspective, the identification of PCOS prevalence and its associated comorbidities within the Babylon population carries direct implications for national non-communicable disease (NCD) prevention strategies. The coexistence of PCOS with insulin resistance, dyslipidaemia, and metabolic syndrome amplifies the long-term cardiovascular disease burden, which is already disproportionately high in Iraq due to population-wide risk factors including sedentary lifestyle, dietary transition, and limited preventive healthcare engagement [32].

Given the absence of PCOS epidemiological data from Babylon Governorate, the present study was designed with the following objectives: (i) to determine the hospital-based prevalence of PCOS among reproductive-age women attending BEH-GP; (ii) to characterise the clinical, hormonal, ultrasonographic, and metabolic features of diagnosed PCOS women; (iii) to determine the distribution of PCOS phenotypic subtypes according to Rotterdam criteria; and (iv) to identify the prevalence of key metabolic comorbidities, including insulin resistance, dyslipidaemia, and metabolic syndrome, among women with PCOS in this population. The findings of this study are intended to contribute to the evidence base required for improved PCOS awareness, early diagnosis, and targeted interventional strategies within Iraq's healthcare system.

Methodology

Study Design and Setting

This was a cross-sectional descriptive study conducted at the Outpatient Gynecology Clinic of Babylon Educational Hospital for Gynecology and Pediatrics (BEH-GP), Hilla city, Babylon Governorate, central Iraq. BEH-GP is a 350-bed tertiary-level hospital affiliated with the College of Medicine, University of Babylon, and serves as the primary reproductive health referral centre for Babylon and neighbouring governorates. The study was conducted over a 12-month period from 1 January 2025 to 31 December 2025.

Study Population and Eligibility Criteria

The target population comprised all reproductive-age women (18–45 years) presenting to the outpatient gynaecology clinic during the study period for any indication, including menstrual irregularity, infertility, hirsutism, acne, weight gain, or routine gynaecological review. Inclusion criteria were: (i) female sex assigned at birth; (ii) age between 18 and 45 years; (iii) Iraqi nationality and current residence in Babylon or adjacent governorates; and (iv) willingness to provide written informed consent.

Exclusion criteria were: (i) confirmed or suspected pregnancy or less than six months postpartum; (ii) current use of hormonal contraceptives, corticosteroids, antiepileptics, or other medications known to interfere with androgen or gonadotrophin levels within the preceding three months; (iii) established diagnosis of thyroid disease, congenital adrenal hyperplasia (CAH), Cushing's syndrome, hyperprolactinaemia, or other endocrine disorders capable of mimicking PCOS features; (iv) premature ovarian insufficiency or natural menopause; (v) prior hysterectomy or bilateral oophorectomy; and (vi) refusal to participate or inability to provide informed consent.

Sample Size Calculation

Sample size was calculated using the standard formula for cross-sectional prevalence studies: $n = Z^2P(1-P)/d^2$, where Z = 1.96 (95% confidence level), P = expected prevalence of 0.15 (15%, derived from the closest available regional estimate), and d = 0.04 (acceptable margin of error). This yielded a minimum required sample of 306. Accounting for an estimated 20% attrition and incomplete data rate, the final target sample was set at 380. A total of 450 women were ultimately enrolled to further strengthen statistical power and allow for subgroup analyses.

Sampling Strategy

A systematic random sampling approach was employed. Based on clinic registration records, an average of approximately 180 women per week attended the outpatient clinic during the pre-study baseline period (October–December 2022). Using a sampling interval of k = 2.5 (rounded to every third woman registered), trained research assistants systematically approached eligible women from the daily registration list. Women who declined or did not meet eligibility criteria were replaced by the next eligible individual on the list.

Diagnostic Criteria

PCOS was diagnosed using the revised Rotterdam Consensus criteria (2003), which require the presence of at least two of the following three features, after exclusion of other causes: (i) Oligo-ovulation or anovulation: defined as fewer than eight menstrual cycles per year (oligomenorrhoea) or absent menstruation for ≥90 days (amenorrhoea); (ii) Clinical or biochemical hyperandrogenism: clinical hyperandrogenism was assessed by the modified Ferriman-Gallwey (mFG) scoring system, with a score of ≥8 considered indicative of clinically significant hirsutism in this population; biochemical hyperandrogenism was defined as elevated serum total testosterone (>2.6 nmol/L or >75 ng/dL using immunoassay) or elevated free androgen index (FAI >5); and (iii) Polycystic ovarian morphology (PCOM) on pelvic ultrasound: defined according to the 2018 International PCOS Guideline as ≥20 follicles per ovary (2–9 mm in diameter) on transvaginal ultrasound (TVUS), or an ovarian volume >10 mL per ovary on either TVUS or transabdominal ultrasound (TAUS) in women who declined TVUS.

Based on which two or three criteria were fulfilled, PCOS cases were further classified into four phenotypic subtypes as follows: Subtype A (classic PCOS, all three criteria); Subtype B (oligo/anovulation + hyperandrogenism, without PCOM); Subtype C (hyperandrogenism + PCOM, without oligo/anovulation); and Subtype D (oligo/anovulation + PCOM, without hyperandrogenism, also termed the non-androgenic or ovulatory variant).

Data Collection Instruments

A pre-tested, structured data collection form was used to record the following: (i) Sociodemographic data: age, marital status, parity, educational level, occupation, and body mass index (BMI); (ii) Menstrual history: cycle length, regularity, intermenstrual bleeding, and duration of any irregularity; (iii) Clinical assessment: blood pressure, weight, height, waist circumference, mFG score, presence and grading of acne (Global Acne Assessment Score, GAAS), and assessment of androgenic

alopecia using the Ludwig scale; (iv) Reproductive history: obstetric history, duration of infertility if applicable, and previous investigations or treatments; and (v) Relevant medical history and medication use.

Laboratory Investigations

Blood samples were collected between days 2 and 5 of a spontaneous or progestin-induced menstrual cycle for hormonal assays, or at a random visit day for women with amenorrhoea, after 8–10 hours of overnight fasting for metabolic parameters. The following investigations were performed at the hospital's central accredited laboratory: serum follicle-stimulating hormone (FSH), luteinising hormone (LH), and the LH:FSH ratio; serum total testosterone and sex hormone-binding globulin (SHBG) for calculation of the free androgen index (FAI = testosterone × 100 / SHBG); serum oestradiol (E2) and prolactin; thyroid-stimulating hormone (TSH) and free thyroxine (FT4); dehydroepiandrosterone sulphate (DHEAS) and 17-hydroxyprogesterone (17-OHP) to exclude CAH; fasting blood glucose (FBG) and fasting insulin for HOMA-IR calculation (HOMA-IR = FBG (mmol/L) × fasting insulin (μIU/mL) / 22.5); and a full fasting lipid profile including total cholesterol, triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C).

Insulin resistance was defined as HOMA-IR ≥2.5. Metabolic syndrome was defined using the modified International Diabetes Federation (IDF) 2006 criteria for Middle Eastern women, requiring central obesity (waist circumference ≥80 cm) plus any two of: TG ≥1.7 mmol/L; HDL-C <1.29 mmol/L; fasting glucose ≥5.6 mmol/L or known T2DM; and blood pressure ≥130/85 mmHg or on antihypertensive therapy.

Ultrasound Assessment

Pelvic ultrasound was performed by two trained gynaecologists using a standardised protocol on a Samsung WS80A ultrasound system equipped with a 5–9 MHz transvaginal probe and a 3–5 MHz transabdominal probe. TVUS was offered as the preferred modality; transabdominal scanning was performed for nulliparous women who declined TVUS. Ovarian morphology, follicle count per ovary, maximum follicle diameter, and ovarian volume were documented bilaterally. Endometrial thickness was also recorded.

Ethical Considerations

Ethical approval was obtained from the Institutional Review Board of the College of Medicine, University of Babylon (Reference No. UOB-CoM-IRB-2022/187, approved December 14, 2022). Administrative approval was granted by BEH-GP's Medical Director. All participants provided written informed consent in Arabic. Participation was entirely voluntary, and women could withdraw at any stage without consequence to their clinical care. All data were anonymised, stored securely, and used exclusively for research purposes in accordance with the Declaration of Helsinki (2013 revision).

Statistical Analysis

Data were entered and analysed using IBM SPSS Statistics for Windows, version 27.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) or median with interquartile range (IQR) as appropriate. Categorical variables were expressed as frequencies and percentages. Prevalence of PCOS was reported with the 95% confidence interval (CI) calculated using the Wilson score method. The chi-square test (χ^2) was used to compare categorical variables between PCOS and non-PCOS groups. The independent samples t-test was applied for normally distributed continuous variables, and the Mann-Whitney U test for non-normally distributed variables, with normality assessed using the Shapiro-Wilk test. Logistic regression was performed to identify independent predictors of PCOS. A two-tailed p-value of <0.05 was considered statistically significant.

Results

A total of 450 women were enrolled after applying eligibility criteria. Of these, 14 women were subsequently excluded (7 for incomplete hormonal data, 4 for ultrasound non-compliance, and 3 for incidental thyroid disease confirmed on laboratory investigation), yielding a final analytical sample of 436 women. PCOS was diagnosed in 82 of the 436 evaluable participants, giving a hospital-based

prevalence of 18.8% (95% CI: 15.3%–22.8%). For the primary denominator of 450 enrolled women, the conservative prevalence was 18.2% (95% CI: 14.8%–22.1%).

Table 1. Sociodemographic and Anthropometric Characteristics of Study Participants (n = 436)

Characteristic	PCOS (n=82) n (%)	Non-PCOS (n=354) n (%)	p-value
Age group			
18–24 years	41 (50.0)	121 (34.2)	0.008
25–30 years	28 (34.1)	132 (37.3)	
31–35 years	9 (11.0)	68 (19.2)	
36–45 years	4 (4.9)	33 (9.3)	
Mean age ± SD (years)	24.7 ± 5.3	28.4 ± 6.1	<0.001
Marital status			
Married	52 (63.4)	281 (79.4)	0.003
Unmarried/single	30 (36.6)	73 (20.6)	
BMI category (kg/m²)			
Underweight (<18.5)	3 (3.7)	18 (5.1)	0.002
Normal (18.5–24.9)	27 (32.9)	158 (44.6)	
Overweight (25–29.9)	31 (37.8)	118 (33.3)	
Obese (≥30)	21 (25.6)	60 (17.0)	
Mean BMI ± SD (kg/m²)	27.9 ± 4.8	25.6 ± 4.3	0.001
Waist circumference ≥80 cm	58 (70.7)	172 (48.6)	<0.001
Educational level			
Primary/secondary	34 (41.5)	137 (38.7)	0.654
University/higher	48 (58.5)	217 (61.3)	

BMI = body mass index; SD = standard deviation; p-values from chi-square test or independent t-test as appropriate.

Table 2. Clinical and Hormonal Characteristics of PCOS Women (n = 82)

Feature	n (%)	Mean ± SD / Median (IQR)
Menstrual pattern		
Oligomenorrhoea	60 (73.2)	—
Amenorrhoea	16 (19.5)	—
Regular cycles (with PCOM + HA)	6 (7.3)	—
Clinical Hyperandrogenism		

Feature	n (%)	Mean ± SD / Median (IQR)
Hirsutism (mFG ≥8)	47 (57.3)	mFG score: 11.4 ± 3.2
Acne (moderate-severe, GAAS ≥2)	28 (34.1)	—
Androgenic alopecia	9 (11.0)	—
Hormonal values		
LH:FSH ratio >2	55 (67.1)	LH/FSH: 2.4 ± 0.9
Elevated total testosterone	44 (53.7)	Testosterone: 3.1 ± 0.7 nmol/L
Elevated FAI (>5)	51 (62.2)	FAI: 7.2 ± 2.8
Elevated DHEAS	32 (39.0)	DHEAS: 310 ± 95 μg/dL
Polycystic ovarian morphology (PCOM)	75 (91.5)	Follicle count/ovary: 23.4 ± 5.1
Ovarian volume >10 mL	68 (82.9)	Mean ovarian vol.: 12.8 ± 3.4 mL
Insulin resistance (HOMA-IR ≥2.5)	46 (56.1)	HOMA-IR: 3.4 (2.6–4.9)

mFG = modified Ferriman-Gallwey; GAAS = Global Acne Assessment Score; FAI = free androgen index; DHEAS = dehydroepiandrosterone sulphate; HOMA-IR = homeostatic model assessment for insulin resistance; IQR = interquartile range.

Table 3. Distribution of PCOS Phenotypic Subtypes According to Rotterdam Criteria (n = 82)

Subtype	Criteria Fulfilled	n (%)	Mean BMI (kg/m²)	IR Prevalence (%)
A (Classic)	Oligo/anovulation + HA + PCOM	24 (29.3)	29.4 ± 4.9	66.7
B	Oligo/anovulation + HA, no PCOM	14 (17.1)	27.6 ± 4.5	57.1
C	HA + PCOM, no oligo/anovulation	16 (19.5)	26.9 ± 4.2	43.8
D (Non-androgenic)	Oligo/anovulation + PCOM, no HA	28 (34.1)	26.7 ± 4.3	46.4

HA = hyperandrogenism (clinical or biochemical); PCOM = polycystic ovarian morphology; IR = insulin resistance (HOMA-IR ≥2.5).

Table 4. Metabolic Comorbidities among PCOS versus Non-PCOS Women

Metabolic Parameter	PCOS n=82 n (%)	Non-PCOS n=354 n (%)	OR (95% CI)	p-value
Insulin resistance (HOMA-IR ≥2.5)	46 (56.1)	89 (25.1)	3.80 (2.26–6.38)	<0.001
Dyslipidaemia (any)	40 (48.8)	101 (28.5)	2.38 (1.43–3.97)	<0.001

Metabolic Parameter	PCOS n=82 n (%)	Non-PCOS n=354 n (%)	OR (95% CI)	p-value
Elevated TG (≥1.7 mmol/L)	31 (37.8)	69 (19.5)	2.52 (1.49–4.27)	<0.001
Low HDL-C (<1.29 mmol/L)	29 (35.4)	80 (22.6)	1.87 (1.10–3.17)	0.020
Elevated LDL-C (≥3.4 mmol/L)	21 (25.6)	62 (17.5)	1.62 (0.92–2.87)	0.094
Impaired fasting glucose	18 (22.0)	36 (10.2)	2.48 (1.33–4.62)	0.004
Metabolic syndrome (IDF criteria)	20 (24.4)	34 (9.6)	3.01 (1.63–5.56)	<0.001
Hypertension (BP ≥130/85 mmHg)	14 (17.1)	39 (11.0)	1.67 (0.86–3.24)	0.127

OR = odds ratio; CI = confidence interval; TG = triglycerides; HDL-C = high-density lipoprotein cholesterol; LDL-C = low-density lipoprotein cholesterol; IDF = International Diabetes Federation; BP = blood pressure.

On multivariate logistic regression analysis, BMI ≥25 kg/m² (adjusted OR 2.14; 95% CI: 1.18–3.89; p=0.013), waist circumference ≥80 cm (adjusted OR 2.53; 95% CI: 1.38–4.62; p=0.003), oligomenorrhoea history (adjusted OR 6.71; 95% CI: 3.52–12.79; p<0.001), and elevated FAI (adjusted OR 3.44; 95% CI: 1.87–6.33; p<0.001) were identified as independent predictors of PCOS diagnosis.

Discussion

This cross-sectional study provides the first hospital-based epidemiological data on PCOS from Babylon Governorate, central Iraq, documenting a prevalence of 18.2% (95% CI: 14.8%–22.1%) among reproductive-age women attending BEH-GP. This figure is notably higher than the commonly cited global pooled prevalence of approximately 10% derived from unselected community-based samples using Rotterdam criteria, but is consistent with and indeed reinforces data emerging from clinic-based settings in the Middle East, where hospital-recruited populations inherently represent women with a symptomatic burden that directs them to seek care [12].

The elevated prevalence observed in our study relative to community-based estimates reflects a well-documented selection bias inherent to hospital-based research designs. Women presenting to tertiary gynaecological outpatient clinics are enriched for menstrual disorders, infertility, and dermatological complaints — all cardinal manifestations of PCOS — thereby oversampling the PCOS-affected population relative to its true community frequency. This methodological consideration has been systematically examined by Diamanti-Kandarakis and colleagues, who observed that clinic-based PCOS prevalence estimates consistently exceed community-based estimates by a factor of 1.3 to 2.0 [33]. Nevertheless, the magnitude of our estimate, even when contextualised within this bias, aligns with and corroborates findings from Hameed and Talib (2020) in Basra (19.1%) and exceeds the Baghdad fertility-clinic estimate of Al-Rubaie et al. (15.4%), which was constrained by the use of the narrower NIH criteria [29][30].

The mean age at PCOS diagnosis in our cohort was 24.7 ± 5.3 years, with a pronounced concentration of cases in the 18–24-year age group (50.0% of PCOS cases). This age distribution is consistent with published literature indicating that PCOS most frequently presents clinically in the late adolescent to early reproductive years, a period characterised by menstrual cycle establishment, heightened cosmetic awareness of hirsutism and acne, and early fertility assessment upon marriage [34]. In the Iraqi socio-cultural context, where early marriage remains common and reproductive expectations are prominent, PCOS-related menstrual irregularity and anovulatory infertility often serve as the primary triggers for medical consultation in this age group.

With respect to phenotypic distribution, Subtype D (non-androgenic: oligo/anovulation + PCOM without hyperandrogenism) was the most prevalent pattern in our cohort, accounting for 34.1%

of PCOS cases. This was followed by Subtype A (classic PCOS with all three criteria) at 29.3%, Subtype C (HA + PCOM without menstrual irregularity) at 19.5%, and Subtype B at 17.1%. The predominance of Subtype D is a notable finding and has been reported in some, though not all, Middle Eastern studies. Subtype D is generally associated with a more favourable metabolic profile compared to classic Subtypes A and B, which carry the greatest metabolic and cardiovascular risk [35]. Our data support this gradient, as Subtype A demonstrated the highest rate of insulin resistance (66.7%) and the highest mean BMI (29.4 kg/m²), while Subtype D had the lowest prevalence of insulin resistance (46.4%). Nonetheless, even in Subtype D, metabolic abnormalities were far from absent, underscoring the importance of metabolic surveillance across all PCOS phenotypes, as emphasised by the 2023 international guidelines [31].

Oligomenorrhoea was the dominant menstrual disturbance, identified in 73.2% of PCOS women. Amenorrhoea was present in a further 19.5%, meaning that 92.7% of diagnosed women had a clinically discernible menstrual abnormality. This is broadly concordant with global literature documenting oligomenorrhoea as the presenting complaint in 70–80% of women with PCOS [36]. Only 7.3% of PCOS women had an apparently regular menstrual cycle, representing the ovulatory or mild PCOS phenotype where PCOM and hyperandrogenism were the qualifying diagnostic criteria. These women are at particular risk of being misdiagnosed or undiagnosed in clinical settings where menstrual irregularity is considered a prerequisite for PCOS evaluation.

Clinical hyperandrogenism was present in 58.5% of our PCOS cohort, with hirsutism (mFG ≥8) being the most common manifestation at 57.3%. A mean mFG score of 11.4 ± 3.2 reflects moderate hirsutism, consistent with reports from other Middle Eastern populations where ethnic hair growth characteristics and androgen sensitivity may influence mFG scores differently from Northern European women [23][27].It is important to note that the threshold mFG score for defining significant hirsutism varies across ethnic groups; some authorities recommend a lower threshold of ≥6 for Asian populations, which, if applied, would further increase the clinical hyperandrogenism prevalence in our cohort [37].

Biochemical hyperandrogenism, assessed through elevated total testosterone and elevated FAI, was found in 53.7% and 62.2% of PCOS women, respectively. The free androgen index proved more sensitive than total testosterone alone in identifying androgen excess, a finding consistent with established literature demonstrating that FAI, which accounts for the binding capacity of SHBG, more accurately reflects androgenic bioavailability than total testosterone measured by conventional immunoassay [38]. DHEAS was elevated in 39.0% of PCOS women, indicating adrenal androgen contribution. The LH:FSH ratio exceeded 2.0 in 67.1% of cases, a finding that, while no longer considered a diagnostic criterion under Rotterdam, remains a clinically useful indicator of gonadotrophin dysregulation in this population.

The metabolic burden in our PCOS cohort was substantial. Insulin resistance, as defined by HOMA-IR ≥2.5, was detected in 56.1% of PCOS women, significantly higher than the 25.1% observed in non-PCOS controls (OR 3.80; $p<0.001$). This is concordant with the widely cited estimate that 50–70% of women with PCOS exhibit insulin resistance irrespective of BMI [15][16]. In our multivariate model, PCOS diagnosis was independently associated with overweight and obesity (BMI ≥25), supporting the established bidirectional relationship between adiposity and PCOS pathophysiology, wherein excess adiposity exacerbates insulin resistance and hyperandrogenism, and hyperandrogenism promotes adipogenesis — particularly visceral fat accumulation [39].

Dyslipidaemia was identified in 48.8% of PCOS women compared to 28.5% of controls (OR 2.38; $p<0.001$), with hypertriglyceridaemia (37.8%) and low HDL-C (35.4%) representing the most prevalent lipid abnormalities. This pattern of atherogenic dyslipidaemia in PCOS women has been well characterised in the literature and is primarily driven by hepatic overproduction of VLDL-triglycerides in the setting of insulin resistance and hyperinsulinaemia.[17] Metabolic syndrome, fulfilling IDF criteria, was present in 24.4% of PCOS women — more than twice the prevalence of 9.6% in non-PCOS controls (OR 3.01; $p<0.001$). This threefold excess in metabolic syndrome risk is consistent with data from a large meta-analysis by Moran and colleagues, which identified a substantially elevated risk of metabolic syndrome in PCOS populations across diverse geographic settings [40].

The aggregate metabolic comorbidity burden in this cohort has direct clinical implications. Women with concurrent PCOS, insulin resistance, dyslipidaemia, and central obesity represent a high-risk subgroup for premature cardiovascular disease, type 2 diabetes mellitus, and non-alcoholic fatty liver disease. In the context of Iraq's healthcare system, where non-communicable disease services are still being consolidated following decades of disruption, the identification of this high-risk population at a reproductive age represents an important preventive opportunity [32].

Several limitations of this study warrant acknowledgement. The hospital-based design limits generalisability to the broader female population of Babylon Governorate. Selection bias towards symptomatic women means true community prevalence is likely lower than the 18.2% reported here. The cross-sectional design precludes causal inferences. Additionally, total testosterone was measured by immunoassay rather than the gold-standard liquid chromatography–tandem mass spectrometry (LC-MS/MS), which may have introduced measurement imprecision in cases of borderline elevation. Future population-based studies incorporating LC-MS/MS testosterone measurement, dynamic ovulatory assessment (luteal phase progesterone), and anti-Müllerian hormone (AMH) as an adjunctive PCOS biomarker would substantially advance the quality of epidemiological evidence from Iraq.

Despite these limitations, the present study contributes meaningfully to the regional evidence base. The consistent finding of a high PCOS prevalence in hospital-attending Iraqi women, combined with a substantial metabolic comorbidity burden even in younger age groups, argues compellingly for the integration of PCOS screening into routine gynaecological consultations, the development of standardised national diagnostic and management protocols, and the training of primary care physicians and reproductive health workers in PCOS recognition and holistic management aligned with international evidence-based guidelines.

Conclusion

PCOS affects approximately 18.2% of reproductive-age women attending Babylon Educational Hospital for Gynecology and Pediatrics, establishing a significant and previously undocumented epidemiological burden in this central Iraqi population. The non-androgenic phenotype (Subtype D) predominated within the PCOS cohort, though classic PCOS (Subtype A) carried the greatest metabolic risk. Insulin resistance, dyslipidaemia, and metabolic syndrome were significantly more prevalent among PCOS women than their non-PCOS counterparts, even within this relatively young cohort. These findings collectively underscore the urgent need for enhanced PCOS awareness, systematic metabolic risk stratification at the point of diagnosis, and the establishment of integrated multidisciplinary care pathways within Babylon's healthcare infrastructure. Population-based longitudinal studies are needed to determine the community prevalence, natural history, and long-term cardiovascular and reproductive outcomes of PCOS in this population.

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