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Polycystic Ovary Syndrome (PCOS): Epidemiology, Pathophysiology, Clinical Management and Regional Perspectives from the Middle East and Iraq: A Narrative Literature Review

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Abstract

Introduction: Polycystic ovary syndrome (PCOS) is a common endocrine and reproductive disorder among women of child-bearing age worldwide. Well known to possess clinical heterogeneity, the disorder manifests through a range of combinations hyperandrogenism, ovulatory dysfunction and polycystic ovarian morphology leading to both short- and long-term reproductive, metabolic and psychological aftermaths. This narrative review will shed light on up-to-date knowledge regarding the epidemiology, pathophysiology, diagnostic criteria, clinical features, complications and management of PCOS based on recent international guidelines as well as published systematic reviews and meta-analyses performed in a timeframe predominantly extending from 2019 to October 2023. We thus examined this condition in the context of variable reported prevalence, sociocultural, diagnostic and health-system factors that influence recognition and management, focusing upon regional data from Iraq and throughout the Middle East. Despite the substantial global consensus around use of the Rotterdam diagnostic criteria and 2023 International Evidence-Based Guideline for assessment and management, this review documents ongoing population-level epidemiological knowledge gaps-in particular from lower- and middle-income countries such as Iraq-and ends with local research priorities.

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Keywords: Polycystic ovary syndrome, PCOS, epidemiology, Iraq, Middle East region, insulin resistance, hyperandrogenism

1. Introduction

Polycystic ovary syndrome (PCOS) is one of the most prevalent endocrine disorders in reproductive-age women, with worldwide prevalence estimates being broadly dispersed from ~5% by 1990 National Institutes of Health (NIH) criteria ^[1], to greater than 20% by more broadly applicable diagnostic criteria in various populations ^[2]. PCOS was originally described in 1935 as a narrow, gynecological condition but has since been recognized as a complex multisystem disorder with reversible and irreversible reproductive, metabolic, dermatological and psychological consequences through life ^[3]. Note on nomenclature: In 2026, a multistep global consensus process led by the same International PCOS Network group recommended renaming PCOS as Polyendocrine Metabolic Ovarian Syndrome (PMOS) ^[4], so that its endocrine, metabolic and reproductive complexity is better represented by its name and to mitigate the stigma attached to the 'cyst' terminology. Since this change in nomenclature occurred after most of the literature synthesized here was published and since regional and Iraqi clinical practice, training materials and patient-facing resources have not yet adopted the new terminology, we continue to use PCOS throughout this review for clarity with the referenced evidence base. Nevertheless, future regional research and clinical communication in Iraq should keep in mind a shift toward using PMOS.

Clinical presentation is highly variable. In women, the clinical presentation varies from intention mainly menstrual irregularity and sterility to indications of androgen excess (hirsutism and acne), obesity, metabolic syndrome features and insulin resistance ^[5]. Because individual diagnostic frameworks refer to only partially overlapping groups of affected women, such heterogeneity has complicated their epidemiological interpretation over the past decades ^[6].

Interest on PCOS has grown dramatically in the past 3 – 5 years, alongside expanding literature addressing its molecular pathophysiology as well as long-term cardiometabolic consequences and psychosocial burden ^[7]. The vast majority of the research carried out in this area across high-income countries has been through large epidemiological and interventional studies, while for Middle Eastern populations, the evidence base is both much smaller and also arguably less developed ^[8,9,10,11]. This review provides a summary of all the international data on PCOS, synthesizing what is known (and not known) about this disease process as it exists in Iraq.

This review aims to (1) tackle recent comprehension on the pathophysiology and epidemiology of PCOS, (2) summarize total diagnostic including clinical, paraclinical manifestations and evidence-based management approaches; and 3 critically appraise local results from the Middle East as well as Iraq for detecting gaps which should be addressed by future research locally.

2. Methodology of This Review

Design This is a narrative literature review, not a systematic review or meta-analysis (i.e. primary data extraction was not required as patient sampling and subsequent statistical pooling of results were beyond the scope of this study). This includes references to original journal articles (with a peer-reviewed format), systematic review analysis, international clinical guidelines followed by regionally relevant studies published in Iraqi and Middle Eastern journals (e.g. Iraqi Journal of Science; IAR Journal of Clinical & Medical Biochemistry; Global Journal of Research in Medical Sciences). Secondary hand search of key reviews and guidelines to identify additional regionally relevant studies not identified by the primary search terms

This narrative review was guided by principles based on the Scale for the Assessment of Narrative Review Articles (SANRA), which include an explicitly stated aim, description of the literature search strategy, and transparent discussion of limitations; this does not indicate systematic-review-level methodological rigor or formal SANRA scoring.

For the sake of transparency, here is a short description of

search parameters for finding sources related to this review. Databases searched were PubMed/MEDLINE, PubMed Central (PMC), ScienceDirect, Scopus and Google Scholar alongside targeted searches of Iraqi and Middle Eastern journal repositories.

Search period: focus on original date of publication between January 2019 and July 2026 with retention of a limited number of seminal papers (eg, original work on insulin resistance as an etiological factor in PCOS) where these papers provide the basis for current understanding.

ICD-10: polycystic ovary syndrome; **Keywords:** epidemiology, prevalence, insulin resistance, hyperandrogenism, pathology and pathway diagnosis management pregnancy outcomes cardiovascular disease depression anxiety Iraq Gulf Cooperation Council.

Data: Exclusion criteria included none but for the purposes of focus, we concentrated on peer-reviewed systematic reviews/meta-analyses and large population-based or GBD analyses (as appropriate, regardless of design), as well as relevant international guidelines. It included medically-oriented hospital- or clinic-based studies for which population-level data were absent, as long as these studies were published in the English language and relevant to Iraq/Middle Eastern studies.

Exclusion: material not peer-reviewed (i.e. because of existing of its peer-reviewed equivalent, preprints, institutional page from internet/web, grey literature), with the exception of cardinal source types (conference abstracts, theses or any other kind of institutional-repository sources) as these have been included only if they have explicitly noted that to be understood in this way in text due to few publications from Iraq.

With regards to structured database searches (PubMed/MEDLINE, Scopus), search terms were combined using Boolean operators: ("polycystic ovary syndrome" OR "PCOS") AND (epidemiology OR prevalence OR "insulin resistance" OR hyperandrogenism OR pathophysiology OR diagnosis OR management) AND ('pregnancy outcomes' OR 'cardiovascular disease' or depression or anxiety) AND (Iraq or 'Middle East' Or 'Gulf Cooperation Council'). For both Google Scholar and ScienceDirect, we conducted search using equivalent Boolean strings which were adapted to the specific syntax of each platform.

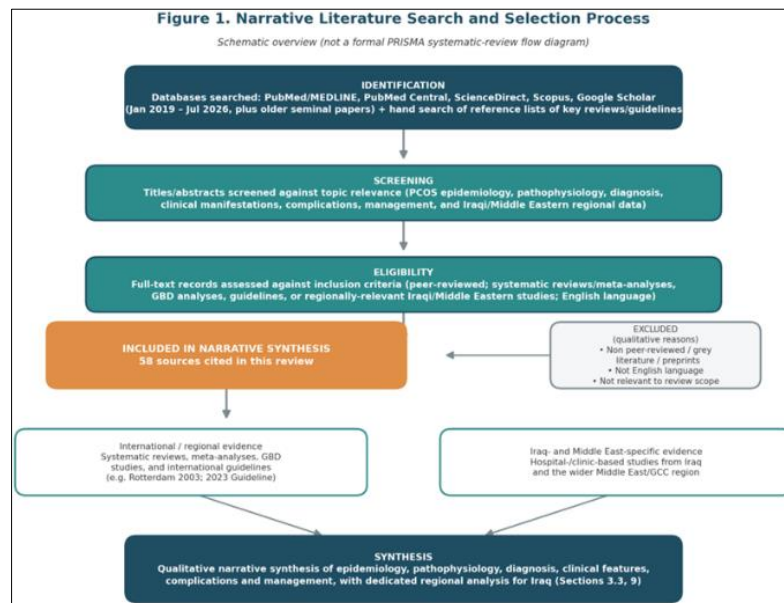


Fig 1: Narrative literature search and selection process across the databases and criteria described above.

Its findings should be considered to provide qualitative synthesis of the literature, not quantitative pooling (this review did not develop or follow an explicit PRISMA search protocol nor assess non-comparatively risk-of-bias or quality-appraisal methodology for inclusion and exclusion criteria). Table 1 presents a summary of the key epidemiological studies (design, population and reported prevalence) from Iraq and the wider region, intended to give the reader a flavor of both regional evidence base and its heterogeneity as discussed in Section 3.

3. Epidemiology and Global Burden

3.1. Global Prevalence

Almost half of the authors used this definition but more than thirty published different diagnoses, resulting in wide variation in prevalence estimates for polycystic ovary syndrome (PCOS). Reported prevalence varies considerably by diagnostic framework, commonly ranging from approximately 5–10% under the strict 1990 NIH criteria to approximately 8–20% under the broader 2003 Rotterdam and 2006 Androgen Excess-PCOS (AE-PCOS) Society criteria, depending on the population and methodology used [2,12,13]. The Nomenclature variation corresponds with population cohort, ethnicity, screening method and study design which all can influence the diagnostic threshold used [1].

This gap is striking considering GBD modelling has estimated a steady increase in the incidence and prevalence of polycystic ovary syndrome (PCOS) since 1990, with a GBD age-standardised incidence rate in women of approximately 64 per 100000 population at the study date (2021) [14,15]. This knowledge base links PCOS with a substantial downstream disease burden from endometrial carcinoma, type 2 diabetes and cardiovascular disease with quantifiable economic cost to health systems [16].

GCC → 3.2 Regional Prevalence of the Middle East

PCOS is an extremely common hormonal disturbance among Middle-Eastern and South-Asian women. Approximately 12% pooled prevalence is reported using Rotterdam criteria in Middle Eastern and refugee populations, and some meta-analyses report estimates of up to 16% [17]. Regional estimates give a wider range of 8–30% but suggest around one quarter to one third Middle Eastern or South Asian women

commenced in the GCC cohorts [18]. A systematic review and meta-analysis of pooled prevalence including only women with infertility in particular GCC nations revealed a 30% PCOS prevalence [9]. Further regional data from specific subpopulations, including medical and health-sciences university students and a large Iranian population-based cohort, similarly demonstrate wide variability in estimated prevalence depending on the diagnostic criteria and sampling frame used [19,20,21]. Based on regional GBD modelling, the burden of cardiovascular disability-adjusted life-years attributed to PCOS is similarly higher in the Middle East compared with Western Europe. This is a marker of both higher biological risk burden, and reflected differential access to chronic-disease management [22].

3.2. Iraq: Prevalence and Possible Origins

There is limited and heterogeneous evidence from Iraq which in most cases is derived from hospital-/clinic-based samples rather than population-representative surveys. A case-control study from Mosul examining physiological and sociodemographic characteristics found PCOS to be a common contributor to female infertility in the region, though no population prevalence estimate was calculated [10]. Methods A retrospective analysis of hospital-, community- and school-based datasets from populations within rural and suburban Baghdad was conducted over approved five-year periods between 1995 and 2020. Yearly prevalence estimates were 5.8%, however, depending on study design they range between 13% and 21% for at least one episode and <5.2% for recurrent [8].

Population burden has been evaluated in several overseas studies including Iraq however, most of the Iraqi studies have focused solely on hormonal and biochemical profiling. A comparative study from an infertility and IVF referral center in Basra found significantly elevated levels of luteinizing hormone (LH) and/or LH/FSH ratios along with higher body mass index in PCOS cases than controls [8]. A third, Baghdad based study found significant differences in hormonal levels as well as hyperandrogenism features typical for Rotterdam criteria between women diagnosed with PCOS and healthy women recruited from an infertility clinic [11]. Although meta-analytic methods cannot be employed due to non-

representativeness of respective small samples, available Iraqi studies support the regional pattern of increased PCOS burden and its close association with infertility presentations that dominate in Iraqi clinical practice ^[8,11]. Iraq's Ministry of Health or Central Statistics Organization features a consistently failing to publish nationally

representative data on PCOS specifically but do provide indicators of reproductive health and fertility at large (eg total fertility rate, infertility-clinic attendance), no disease specific prevalence estimate exists for the presence of PCOS in Iraq. Development in section 9.3: Evidence gaps—Absence of systematic national level surveillance.

Table 1: Summary of key epidemiological studies on PCOS in Iraq and the wider Middle East

Study (ref.)	Country / setting	Design & population	Diagnostic criteria	Reported PCOS prevalence
Mnajed ^[8]	Iraq (rural/outskirts Baghdad)	Cross-sectional, outpatient clinic attendees, n=3,370 screened	Rotterdam	~3.9% of screened attendees (131/3,370)
Ataallah & Altaï ^[10]	Iraq (Mosul)	Case-control, hospital-based, 60 cases / 60 controls	Not uniformly stated	Case-control study; no population prevalence estimate calculated
Mohamad ^[11]	Iraq (Baghdad, infertility clinic)	Case-control, 80 cases / 20 controls	Rotterdam	Clinic-based hyperandrogenism cohort (not a population prevalence)
Alam <i>et al.</i> ^[9]	GCC countries (Saudi Arabia, Qatar, Kuwait, Oman)	Systematic review & meta-analysis, 7 studies, infertile women	Mixed (studies pooled)	Pooled 30.0% among infertile women
Ding <i>et al.</i> ^[17]	Middle East (pooled, Iranian/Turkish cohorts)	Systematic review & meta-analysis	1990 NIH / 2003 Rotterdam / 2006 AES	6.1% (NIH) to 16.0% (Rotterdam)
Mousa <i>et al.</i> ^[18]	Middle East (regional synthesis)	Systematic review & meta-analysis of gynecological conditions	Mixed	~8–30%, varying by population

Note: Prevalence estimates of all conditions are not directly comparable because each study sampled a different population (general population versus infertility clinic and hospital population) and/or studied over varying time periods. The following table provides an overview of study design and reported estimates, as opposed to a pooled quantitative comparison.

Table 2: Summary of PCOS studies conducted within Iraq (subset of Table 1)

Study (ref.)	City / setting	Design & sample	Diagnostic criteria	Key finding
Mnajed ^[8]	Rural/outskirts of Baghdad	Cross-sectional, outpatient clinic attendees, n=3,370 screened	Rotterdam	~3.9% of screened attendees (131/3,370)
Ataallah & Altaï ^[10]	Mosul	Case-control, hospital-based, 60 cases / 60 controls	Not uniformly stated	Case-control study; no population prevalence estimate calculated
Mohamad ^[11]	Baghdad (infertility clinic)	Case-control, 80 cases / 20 controls	Rotterdam	Clinic-based hyperandrogenism cohort (not a population prevalence)

Note: all three available Iraqi studies are small, single-center, hospital- or clinic-based samples; none is population-representative at a national scale (see Section 3.3 and Section 9.3).

4. Pathophysiology

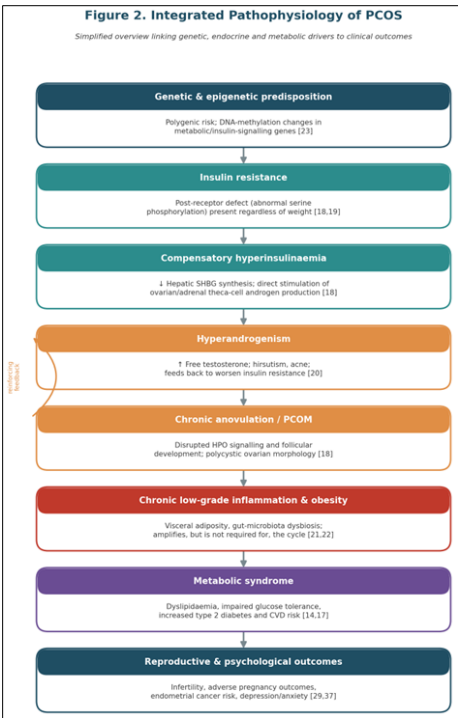


Fig 2: Integrated pathophysiology of PCOS, from genetic predisposition through to reproductive and psychological outcomes (see Sections 4.1–4.4 for supporting evidence).

Sections 4.1–4.4 below detail the mechanistic evidence underlying each step of this cascade.

4.1. Hormonal and Neuroendocrine Mechanisms

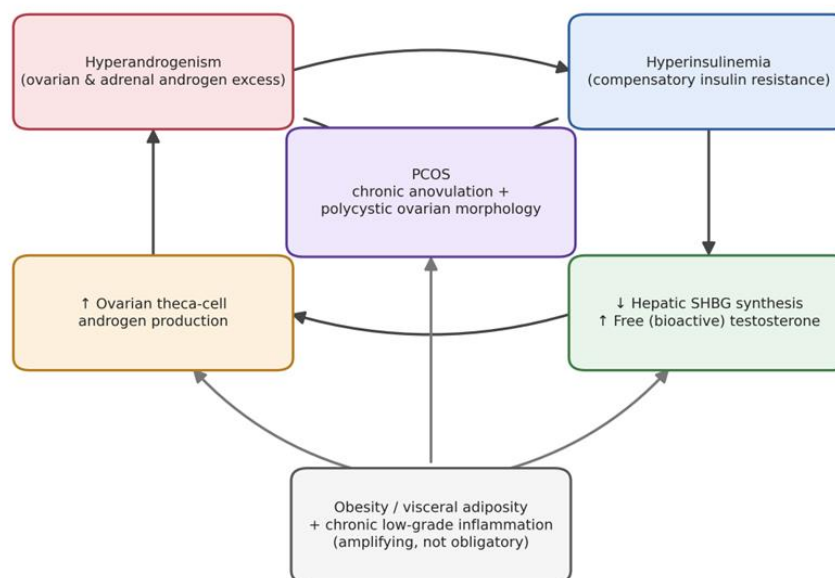
The clinical manifestations of PCOS have been postulated to be the result of a multifactorial pathophysiology that is incompletely defined but commonly accepted as a functional derangement in hypothalamic–pituitary–ovarian (HPO) axis signaling, associated with changes in gonadotropin-releasing hormone (GnRH) pulsatility and an increased ratio of luteinising hormone to follicle-stimulating hormone [23]. Persistent anovulation and polycystic ovarian morphology create a self-perpetuating cycle of chronic elevation of abnormal gonadotropin secretion that in turn increases androgen production at the ovaries, causes dysregulation of normal follicular development and additional luteal phase defects [23].

4.2. Insulin Resistance and Hyperinsulinemia

It is now believed that insulin resistance is one of the

foundings and overall unifying aspects of PCOS pathophysiology, with most women with this condition demonstrating features of it irrespective of obesity [23]. While these studies confirmed the metabolic actions of insulin through its own receptor, they also demonstrated that insulin promotes ovarian and adrenal androgen production as well as pituitary LH release; abnormal serine phosphorylation of the insulin receptor may account for both the insulin resistance and hyperandrogenism common in many patients [24]. In response, compensatory hyperinsulinemia stimulates ovarian theca cells to produce greater amounts of androgens while simultaneously decreasing hepatic production of sex hormone-binding globulin (SHBG) [23]. This produces a vicious cycle in which hyperandrogenism, hyperinsulinemia and low sex hormone-binding globulin worsen each other [25]. The clinical phenotype also influences the severity of insulin resistance: the highest degrees are usually present in those females with both hyperandrogenism and ovulatory dysfunction, but even mild phenotypes can have demonstrable insulin insensitivity [25].

Figure 1. Proposed “vicious cycle” model linking hyperandrogenism, hyperinsulinemia, and reduced SHBG in PCOS pathophysiology



Adapted schematically from the insulin-resistance/hyperandrogenism model described by Dunaif (1997) and Diamanti-Kandarakis & Dunaif (2012); obesity amplifies but is not required for the cycle.

Fig 3: Vicious-cycle model of hyperandrogenism, hyperinsulinemia and SHBG in the pathophysiology of PCOS.

4.3. Chronic inflammation, adipose tissues and obesity

Obesity correlates well with PCOS through overlapping pathophysiological mechanisms consisting of insulin resistance, compensatory hyperinsulinemia and the activation of renin–angiotensin–aldosterone system [26]. Obesity, especially with further deposition of visceral fat tissue promotes a state of low-grade chronic inflammation which is a contributing factor to insulin resistance and hyperandrogenism [26,27]. Gut microbiota dysbiosis is a further contributing factor: diet-induced obesity is associated with changes in gut microbiota composition, with increased intestinal permeability leading to translocation of bacterial lipopolysaccharides into the systemic circulation, inducing systemic inflammation and augmenting insulin resistance [28]. Importantly, insulin resistance is a key feature of the pathophysiology of PCOS which may exist even in those with normal weight, and thus it helps to explain both lean women

with (and without) more exaggerated features of the syndrome [26].

4.4. Genetic, Epigenetic and Immune Contributions

PCOS has an important genetic background but epigenetic modulation appears to have a fine-tuning role on disease vulnerability; DNA methylations associated with metabolism and insulin signalling characterization genes may become phenotypic expression or susceptibility biomarkers [29]. On the other hand, immune dysregulation has also been a relevant pathogenetic mechanism suggested where low-grade chronic inflammation represented by high numbers of macrophages and pro-inflammatory cytokines (e.g. TNF- α) or cellular insulin resistance and hyperandrogenaemia interacts to exacerbate poor metabolic and reproductive outcomes [30]. Collectively, these findings support a polygenic model for the contribution of recurring interactions

among genetic risk, endocrine dysfunction, immune–metabolic dysregulation and environmental or lifestyle factors to the pathogenesis of PCOS [3,30].

5. Diagnostic Criteria and Challenges

Over the past 30 years, three sets of diagnostic criteria for PCOS have been widely used: the NIH'90 criteria, Rotterdam '03 and AE-PCOS Society (AE-PCOS) [6]. Aside from the classification system established by NIH, Rotterdam criteria

are utilized globally and were established during a consensus of experts in reproductive medicine; it utilizes at least two out of three features: oligo-anovulation, clinical or biochemical hyperandrogenism and polycystic ovarian morphology on ultrasound [3,5]. In contrast, Rotterdam criteria identify a larger, more heterogeneous population of women because they do not mandate menstrual irregularity and this can explain the greater prevalence estimates usually reported using Rotterdam-based studies rather than NIH criteria [2,6].

Table 3: Comparative Summary of PCOS diagnostic frameworks

Feature	1990 NIH criteria	2003 Rotterdam criteria	2006 AE-PCOS Society criteria	2023 International Evidence-Based Guideline
Required features	Both required: (1) hyperandrogenism (clinical/biochemical) and (2) chronic anovulation	2 of 3: (1) oligo/anovulation, (2) hyperandrogenism, (3) PCOM	Hyperandrogenism required, plus either ovarian dysfunction (anovulation) or PCOM	Formally endorses the 2003 Rotterdam criteria (2 of 3), with structured exclusion of mimicking disorders
Role of ultrasound (PCOM)	Not used	Can substitute for either anovulation or hyperandrogenism	Requires hyperandrogenism; PCOM can substitute for anovulation	Transvaginal ultrasound only ≥8 years post-menarche; AMH not yet recommended to replace ultrasound
Approximate prevalence yield	≈6%	≈20%	≈15%	~10–13%
Clinical implication	Narrowest phenotype; excludes normo-androgenic, ovulatory PCOM (“Phenotype D”)	Most comprehensive and widely accepted; basis of current international guidelines	Centres hyperandrogenism as a key feature; excludes Phenotype D	Reinforces Rotterdam as the international standard; adds lifestyle-first management, routine mental-health and cardiovascular screening, and ethnic-specific BMI thresholds

Sources: [1,2,6,31,32,33].

Diagnosis is primarily clinical (focused on menstrual history and examination for signs of hyperandrogenism [hirsutism, acne] with androgen measurement/transvaginal ultrasound added only when necessary) [6]. The clinical role of anti-Müllerian hormone (AMH) as a potential surrogate marker for polycystic ovarian morphology has not yet been settled, but remains more an uncertainty than a definitive conclusion [3]. Diagnosis of PCOS in adolescence is rare due mainly to the characteristics of the peripubertal transition, which often

includes amenorrhea and milder acne [31], with most professional bodies advising that persistently abnormal menstrual cycles and hyperandrogenism be used at younger ages, while ultrasounds based on diagnosis are deferred until gynecological maturity has been reached [5]. Noticeably whilst commonly taught in practice based on the Rotterdam criteria, trainees within obstetrics and gynecology have frequently cited use of all 3 elements however without being able to accurately enumerate all three constituents [6].

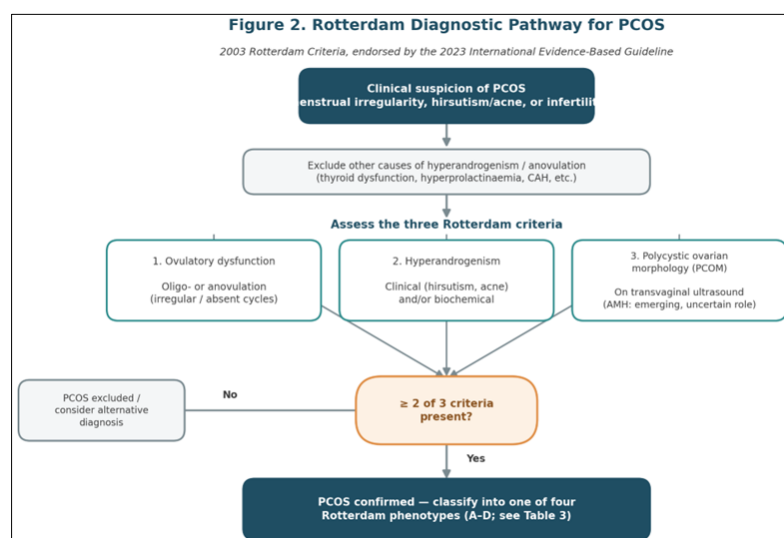


Fig 4: Rotterdam criteria: PCOS diagnostic pathway and 2023 International Evidence-Based Guideline

Four acknowledged clinical phenotypes are derived via the Rotterdam criteria (summarized in Table 4 and Figure 5), which differ with respect to hormonal, reproductive, and

metabolic profiling; nevertheless, these phenotypes are being more commonly used to individualize risk assessment and management.

Table 4: PCOS phenotypes based on the Rotterdam criteria

Phenotype	Defining features (H = hyperandrogenism, O = ovulatory dysfunction, PCOM = polycystic ovarian morphology)	Typical severity of insulin resistance	Relative reproductive/metabolic risk
A	Classic phenotype: H + O + PCOM	Most pronounced	Maximum metabolic and reproductive risk combined
B	NIH-equivalent phenotype: H + O (PCOM negative)	Marked	High metabolic risk; NIH-equivalent phenotype
C	Ovulatory phenotype: H + PCOM (ovulatory, O negative)	Mildest	Predominantly dermatological presentation; lower metabolic risk
D	Non-hyperandrogenic phenotype: O + PCOM (H negative)	Variable, generally mild–moderate	Rotterdam-only; not met by NIH or AE-PCOS criteria

Sources: [1,6,25].

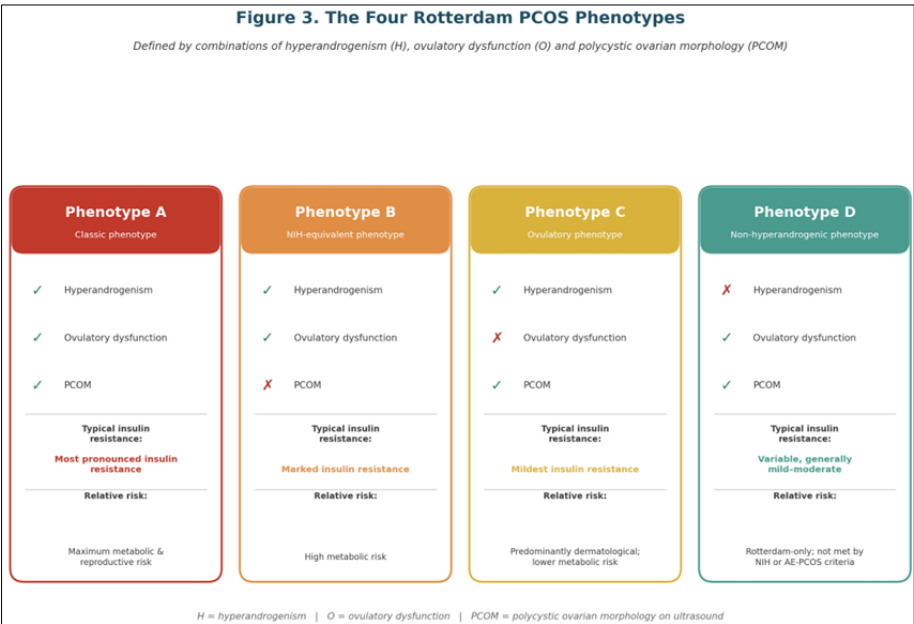


Fig 5: The Rotterdam criteria identify four PCOS phenotypes:

6. Clinical Manifestations

6.1. Reproductive and Menstrual Features

The commonest reproductive phenotype of PCOS is oligomenorrhea or amenorrhea due to chronic anovulation, which can result in anovulatory infertility; another smaller subgroup exhibit menorrhagia [5].

6.2. Dermatological Manifestations

Hirsutism, defined as excessive terminal hair growth in a male-pattern distribution has been one of the most recognized androgen-related features of PCOS and is more specifically correlated with circulating levels of multiple androgens [34,35]. The dermatological signs are often related to the first reason for which women come for consultation; such symptoms usually correlate with increased psychopathology and decreased self-esteem [35].

6.3. Metabolic Features

The vast majority of women with PCOS are at least overweight or obese, have insulin resistance and dyslipidaemia together with impaired glucose tolerance although a smaller proportion present as lean [26,31]. Most of these metabolic derangements are responsible for most of the chronic disease burden described in Section 7.

7. Complications and Comorbidities

7.1. Infertility

PCOS is a common international cause with anovulatory infertility [10]. Due to the very different mechanism and clinical characteristic of ovulatory dysfunction in PCOS and the differences in management according to a woman's fertility intent, it has been recommended that for those patients who desire a pregnancy diagnosis should be achieve early [5].

7.2. Pregnancy and Obstetric Complications

Women with PCOS have a significantly increased risk of adverse pregnancy outcomes, as shown in meta-analyses and systematic reviews including gestational diabetes mellitus (GDM), gestational hypertension/pre-eclampsia, miscarriage and caesarean delivery compared to controls [36]. In a similar way, both preterm delivery and either normal or (in some studies) low birthweight are increased by similar amounts, with higher neonatal unit admission rates [37]. These associations are mostly due to the PCOS diagnosis criteria threshold among source studies, maternal age as well as pre-pregnancy obesity and mode of conception (e.g. natural conception versus assisted reproductive technology) [36]. Interest has focused particularly on the incidence of GDM in women with PCOS (21). Four systematic reviews included in

our analysis demonstrate that women with PCOS have nearly three-fold increased relative risk of gestational diabetes mellitus (GDM), and this effect is even greater in those with a higher pre-conception body-mass index, existing insulin resistance and certain hyperandrogenic phenotypes^[38,39,40,41]. The findings are not totally consistent though. A large Norwegian cohort found that GDM was associated with increased risk of late miscarriage but did not independently confer increased risks for other pregnancy complications once maternal age, BMI, parity and gestational weight gain were controlled for. This pattern has been described by some authors as evidence that GDM per se is not responsible for much of the excess pregnancy-related risk associated with PCOS, but rather a common underlying insulin resistance^[42]. Discussion of variability as discussed, this variation shows cases the more general problems present in the literature on PCOS and pregnancy^[38,43] with contradictory diagnostic criteria, differences in population characteristics, and not adjusting for obesity meaning meaningful comparison between studies is difficult.

7.3. Long-term consequences: Metabolic and cardiovascular risk

This is also crucial as after reproductive age, women with PCOS can have a several fold increase in the risk for type 2 diabetes, metabolic syndrome, dyslipidemia and cardiovascular disease (including hypertension)^[6]. This risk is heterogeneous based on population. There is evidence for a gene-environment interaction based on studies conducted with GBD data between the type 2 diabetes prevalence in women with PCOS from statistically comparable obesity groups through North Asia and Northern Europe, suggesting a higher (3 to 5-fold) prevalence of the disease among similarly obese South Asian as opposed to those from Northern Europe^[16,21]. Emerging evidence also suggests that offspring of women with PCOS may themselves carry an elevated cardiovascular risk profile, highlighting a potential transgenerational dimension to this metabolic burden^[44].

7.4. Risk of Gynecological and Endometrial Cancer

PCOS involves long-standing anovulation, causing pathological and persistent unopposed estrogen exposure of the endometrium with increased lifetime risk of endometrial hyperplasia and endometrial cancer^[16]. It is difficult to standardize the classification and comparison of overweight magnitude, yet it has become clear from GBD data that there are significant population differences in endometrial cancer incidence associated with obesity among women with PCOS despite relatively comparable BMI^[22].

7.5. Psychological health and quality of life

There is a growing and considerable body of evidence that links PCOS with adverse mental health and quality of life issues. A Danish conference report suggested that approximately two-thirds of women with PCOS screened positive for anxiety and approximately three-fifths for depression, with roughly seven in ten reporting low quality of life; hormonal irregularity, overweight, infertility and hirsutism were each individually correlated with greater mental health deterioration^[45]. As this source is a conference abstract rather than a full peer-reviewed study, these figures should be interpreted with appropriate caution pending confirmation in a full publication. A recent systematic review and meta-analysis showed that many individuals with PCOS

suffer from depression and/or anxiety, and despite not being physical disorders, body-image dissatisfaction, perceived stigma and low self-esteem are significant mediators of this relationship in LAMICs^[46], where visible symptoms of a PCOS such as hirsutism or acne appear to be particularly stigmatized. Using this approach, an Arabic screening study showed that nearly as many PCOS individuals had clinically significant anxiety (41%), but predominantly unmarried women, similar some extent to the importance of sociocultural context^[47]. Elevated depressive and anxiety symptom burden has similarly been documented in broader adult PCOS populations, and psychosocial outcomes and quality of life appear to differ further by reproductive history, with distinct patterns observed among nulligravida, primigravida and multigravida women^[48,49].

The causal pathway remains unresolved: although part of the association between PCOS and anxiety and depression may be mediated by obesity (suggested by evidence from several individuals with PCOS^[50] that the excess risk was explained largely by comorbid obesity), other studies comparing severe but uncomplicated obesity to women either with or without PCOS show similar population means for some psychological symptoms in both groups. Regardless of the underlying mechanism influencing this relationship, there is substantial evidence to support the importance of incorporating mental health screening into routine care for women with PCOS^[45,51].

8. Management and Treatment

8.1. Lifestyle Interventions

International guidelines consistently recommend lifestyle modification (diet, physical activity and behavioural strategies) as the first-line management for all facets of PCOS throughout its life course, including menstrual dysfunction, metabolic risk reduction and fertility^[31,52]. Thus, there is no evidence that any one dietary or exercise prescription should be favored as a superior weight-loss tool by the health care provider, and recent guidance recommends combatting weight stigma while managing medically relevant weight-related risk^[31]. Two forms of structured exercise (HIIT included) exist, although both promise improvement in metabolic parameters and psychiatric symptoms including depression, anxiety and PCOS-specific quality of life^[1,53].

8.2. Drugs for Menstrual Disorders and Hyperandrogenism

Combined oral contraceptive pills remain the first-line pharmacological management for menstrual irregularity and hyperandrogenism, with lower-dose ethinyl estradiol formulations preferred to reduce adverse effects; however, no clear preference among individual formulations has been identified^[32]. Metformin is primarily indicated for its metabolic effects, especially in adults with a BMI ≥ 25 kg/m², and clinical benefits of the drug appear to exceed that of inositol supplementation as it has only minimal direct impact on hirsutism, ovulation or weight compared to other options^[32]. Metformin may also modestly reduce PCOS-related acne, a potential downstream effect of better hyperinsulinaemia and hyperandrogenism^[54]. Hirsutism laser hair removal is only effective in a selective patient population and anti-androgen medication can be offered externally to first line therapies usually when considered contraindicated or ineffective^[32].

8.3. Fertility Management

Letrozole is recommended as first-line pharmacological therapy for ovulation induction in women with PCOS desiring pregnancy based on data extrapolated from large randomized trials (higher live-birth rates and lower twin–pregnancy rate than clomiphene citrate) [32]. Clomiphene citrate does remain an acceptable second-line agent alone or in combination with metformin, and the addition of metformin significantly increases ovulation rates over clomiphene monotherapy [33]. E.g. Gonadotropins or surgical/exogenous ovarian stimulation by laparoscopic ovarian surgery [32].

8.4. Management During Pregnancy

Due to the higher risk of gestational diabetes and other complications during pregnancy, further screening and follow up should be considered in all phases of pregnancy in

PCOS [38]. It appears that metformin may lower the risk of preterm delivery or excessive gestational weight gain, as suggested by some data. Overall, however, strong and consistent evidence that this is consistently more effective in reducing insulin resistance or indeed the risk of gestational diabetes or pregnancy-induced hypertension is lacking (evidence should be noted as mixed), so – like most things in pregnancy it tends to be very patient specific rather than a one size fits all recommendation [32].

8.5. Novel Therapies for Obesity

Pharmacological weight-loss therapy and bariatric surgery may be available to women with PCOS and obesity based on the same criteria that apply to both sexes in the general population; however, due to their wider range of metabolic benefits beyond just body weight, GLP-1 receptor agonists have become an area of particular interest for this cohort.

Table 5: Treatment of PCOS as per modern evidence-based guideline 2023

Presenting concern	First-line management	Second-line / alternative	Key notes
General / all presentations	Lifestyle modification: diet, exercise, behavioral support	—	No specific diet or exercise regimen is superior; avoid weight stigma
Menstrual irregularity / hyperandrogenism	Combined oestrogen pill (low-dose ethinyl estradiol)	Metformin (esp. if BMI ≥ 25); anti-androgens if COCP contraindicated or insufficient	Laser treatment for hirsutism in selected patients
Metabolic dysfunction / insulin resistance	Lifestyle change + metformin (if BMI ≥ 25)	GLP-1 receptor agonists / bariatric pathway if obesity unresponsive to lifestyle	Limited direct effect of metformin on hirsutism or ovulation
Infertility / ovulation induction	Letrozole	Clomiphene citrate $\pm$ metformin; gonadotropins, laparoscopic ovarian drilling, or IVF if first-/second-line fails	Letrozole is associated with higher live-birth rate and lower twin rate than clomiphene
Pregnancy	Gestational diabetes and hypertensive-disorder screening/monitoring	Individualized metformin use	Evidence for metformin preventing GDM or pre-eclampsia is inconsistent
Psychological symptoms	Routine mental health screening	Referral to psychological support / CBT when appropriate	CBT reduced depression and anxiety scores versus other treatments in RCTs

Sources: [31,38,51,52,32,39,54,33].

9. The regional perspective: PCOS in the Middle East and Iraq

9.1. Widespread variability and limits of the regional data

PCOS prevalence estimates based on studies from Iraq and the greater Middle East (Table 1) vary widely, spanning over 15 percentage points from approximately 5% to exceeding 20%, depending greatly upon the period and location of study relative to current diagnostic criteria in use. The majority of the data from Iraq originated from small, clinic or hospital-based samples, many participating women were recruited through infertility/IVF clinics; which may produce overestimation of prevalence compared with population-level studies and limit generalisability [8,11]. These results mirror a wider regional disparity: the number of comprehensive, general population studies using internationally standardised Rotterdam criteria published in the Middle East is considerably lower than that for North America, Europe or East Asia [17].

9.2. Sociocultural and Health-System Factors

Non-medical factors certainly also play a part in the experience and management of PCOS within the region. Normative expectations of femininity, marriage and fertility in pronatalist settings can exacerbate stigma around observable signs such as hirsutism, acne or weight gain (where infertility is present), because these may clash with expected reproductive and social roles for women. Dedicated

regional research on PCOS-related stigma, infertility stigma and body-image outcomes in Middle Eastern women remains limited, and this observation is offered here as contextual interpretation rather than a claim supported by a specific regional stigma study. For instance, GBD modelling at the health-system level identified excess cardiovascular disease burden associated with PCOS in Middle Eastern countries compared to Western Europe [22], which reflects both inequities in access to chronic-disease screening and preventive care and underlying biological risk.

9.3. Iraq-Specific Research Gaps

Global bibliometric analysis shows a marked and sustained rise in PCOS research output over the past decade, yet this growth has been unevenly distributed geographically, with Iraq and much of the surrounding region contributing only a small fraction of the published literature [55]. The particular evidence base from Iraq suggests that multiple gaps are present. The first is the lack of nation-wide multi-centre prevalence studies using population-representative, standardised international diagnostic criteria [8]; as stated in Section 3.3 and as opposed to numerous endocrinopathies for which national surveillance data are currently published from either the Iraqi Ministry of Health or Central Statistics Organisation, there presently exists no current PCOS specific national survey based-data documenting key disease parameters. Secondly, most Iraqi studies published until now

focused mainly on their hormonal and biochemistry profiling of smaller clinical populations with limited population-specific data on long-term metabolic and cardiovascular risk, mental health burden as a consequence of stress/environmental impact or response to lifestyle or pharmacological cures/intervention^[8,11]. Third, there is little data on how Iraqi sociocultural factors — such as early-marriage norms and stigma regarding infertility and gynaecological care, and mental health service provision affect rates of diagnosis-seeking, treatment adherence and psychologic outcomes. Locally led, well-funded research to fill these gaps in evidence would deepen the existing evidence base for Iraqi clinicians and policymakers and enable easier comparison to the regional and global literature summarised in this review.

9.4. Clinical Implications for Iraq

Because of the availability of no outcome studies in Iraqi people, clinicians mainly rely on the Rotterdam- diagnostic criteria and the present international guideline^[6,32] in Iraq. Such a sensible default given the body of global evidence generating for its support also UNZ likely means that management recommendations are uncalibrated against local trends in body mass index and insulin resistance or comorbidity patterns, which probably vary from those in the predominantly Western and East-Asian populations underpinning much guideline evidence^[26]. A realistic approach to aligning practice in Iraq nationally with international guidance, which accounts for local resourcing limitations, could include local investment in structured lifestyle-intervention programmes and mental health referral pathways alongside timely laboratory-based hormonal assessment^[31]. Comparable region-specific adaptation efforts have already been undertaken elsewhere, for example in a Nordic-perspective summary of the same international guideline, and could serve as a template for a similarly tailored Iraqi or Middle Eastern implementation document^[56].

Also placing efforts to enhance public health messages and clinician education will benefit from better care for PCOS in Iraq. It is likely that women with a primarily metabolic or dermatological phenotype (rather than one with a reproductive centred presentation) are poorly recognised and treated in routine practice since the majority of current Iraqi evidence comes from infertility and IVF centres rather than general Gynaecological or primary care settings^[8,11]. Increasing screening and awareness earlier in a woman's life course (for instance extending into primary care and general gynaecology) should thus allow this condition, often grouped with other conditions such as polycystic ovary syndrome (PCOS) in childhood or later authentic variation of early reproductive related metabolic and cardiovascular risk gained^[7] as discussed in Section 7; to be recognised at its natural history with consequent reduction of the long-term metabolic and cardiovascular risk outlined.

10. Discussion

10.1. Comparison with Europe, East Asia and the Americas

Contextualising the Iraqi and Middle Eastern figures within a broader international framework proves instructive. Pooled prevalence in Europe and the USA (using Rotterdam-based criteria) has recently been estimated to be approximately 19.5%^[57] broadly similar to the pooled estimate for the

Middle East of 16.0% detailed here, which is higher than that reported regionally in this review (Section 3). In comparison, estimates for East Asians were usually lower: a Chinese ethnicity-specific pooled estimate using Rotterdam criteria of 5.6%^[17], and a recent large Chinese national survey reported an increasing but still relatively low phenotypic prevalence of 7.8% in 2020^[58]. Pooled prevalence of South Asian Data from India falls in the middle, with approximately 10% under Rotterdam/AE-PCOS criteria^[59]. Rather, many reasons probably account for this geographic gradient. One reason is the differing expression of hyperandrogenism: East Asian women with PCOS show a greater tendency to have low clinical hyperandrogenism, shifting more patients into non-hyperandrogenic Rotterdam phenotype D and reducing the apparent prevalence under stricter criteria^[17]. Direct comparisons across regions are further limited by differences in body composition and obesity prevalence, varying access to transvaginal ultrasound and androgen assays, and population based versus clinic/hospital based sampling frames^[15,22]. The majority of those studies recruit samples from clinics and hospitals in the context of infertility services (see Section 3.3, Table 2) so that estimates relevant to Iraq likely overestimate prevalence when compared with population-based surveys like those from European, Chinese and Indian studies cited above. This strengthens the argument presented in Section 9.3 for obtaining population-representative Iraqi data.

10.2. Interpreting Divergent Findings

For Iraq itself, why is the range so large: ~3.9%–20% (Table 2)? This is largely the result of gaps in methodology — not biology. The outpatient-clinic screen of Mnajed^[8] recruited from a widely unselected attendee population and produced the lowest result, whereas the retrospective Baghdad analysis in Section 3.3 (Section 7)^[8] and Basra and Mosul case-control studies^[10,11] drew through infertility or hospital-referral channels that enrich PCOS cases and yield exaggerated prevalence estimates. All three of the studies from Iraq shown in table 2 are not population-representative, so none can be considered a national estimate; they should be interpreted as clinic-level signals rather than epidemiological benchmarks.

East Asia Why is the wider Middle East higher Three factors combine. Secondly, ethnic differences in androgen secretion such that Middle Eastern women are more likely to meet hyperandrogenism-based criteria than East Asian women who will instead be classified under the low-androgenic phenotype D category which leads to reduced estimates using stricter definitions^[17]. Second, Gulf and Middle Eastern populations are undergoing a rapid nutritional transition that is associated with increased prevalence of obesity and insulin resistance, which amplify hyperandrogenism-hyperinsulinaemia cycles (Section 4; Box 2) causing greater symptom severity and detection^[22,26]. Third, although the Iraqi studies are well conducted, most Middle Eastern data originate from infertility or hospital settings as opposed to community surveys — i.e., similar referral bias concerns highlighted for Iraq specifically^[8,9,11].

Nowhere, either, can the true inconsistency of the evidence base be papered over. Systematic reviews document an almost threefold relative risk for PCOS in gestational diabetes^[38], yet a recent large Norwegian cohort analysis found no independent association after adjusting for BMI, age, parity and weight gain in pregnancy^[42] — a discrepancy

that is most plausibly explained by confounding by obesity rather than non-causation but cannot be adjudicated on the basis of narrative synthesis alone. As for the strength of studies, the best evidence in this review comes from large methodologically transparent sources: national or multi-country surveys and meta-analyses with clear diagnosis criteria and reasonable sample sizes (e.g., the two-wave Chinese national survey^[58] the European/USA meta-analysis^[57]). The Iraqi hospital- and clinic-based literature itself (n=60–370 per study^[8,10,11]), which is small, single-centre

and non-representative research, provides the weakest evidence. For this reason and those discussed in turn, Section 10.5, conclusions drawn from it (and indeed from this review) should be viewed as hypothesis-generating rather than conclusive.

10.3. Recent Landmark Studies (2023–2026)

A few recent, methodologically well-constructed studies are highlighted in Table 6 as most relevant to the international evidence synthesized in this review.

Table 6: Selected recent landmark studies informing this review (2022–2025)

Author (ref.)	Country	Study type	Major finding	Clinical implication
Teede <i>et al.</i> ^[31]	International (39 societies, 71 countries)	2023 International Evidence-Based Guideline	Consolidated Rotterdam-based diagnosis with structured lifestyle, metabolic and psychological care pathways	Current international reference standard for diagnosis and management
Zeng <i>et al.</i> ^[7]	Global	GBD trend analysis, 1990–2021	Age-standardized PCOS incidence and prevalence have risen globally, with uneven regional health-system impact	Highlights need for region-specific surveillance, including in under-represented settings such as Iraq
Yang <i>et al.</i> ^[58]	China	Two-wave national survey (2010 vs 2020, n=28,739)	Prevalence rose from ~4.3% to 7.8% over a decade, with a more severe phenotype in 2020	Demonstrates feasibility and value of repeated population-based national surveys
Chiaffarino <i>et al.</i> ^[57]	Europe & USA	Systematic review & meta-analysis	Rotterdam-based prevalence ~19.5%, similar across Europe and the USA but phenotype frequency differs	Confirms high-income-region comparability is possible with standardized criteria
Atinga <i>et al.</i> ^[46]	Low- and middle-income countries	Systematic review & meta-analysis	Depression and anxiety are substantially elevated in PCOS across LMIC settings	Supports routine mental-health screening in PCOS care, including in Iraq
Patten <i>et al.</i> ^[53]	Australia	Randomized controlled trial	High-intensity interval training improved metabolic parameters and PCOS-specific quality of life	Provides trial-level evidence for structured exercise as a management option

10.4. Summary of General Conclusions

Such a body of literature brings out some general conclusions. Certainly, PCOS is an inherently heterogenous disorder and as a result reported rates in the literature are highly variable depending on both population studied and diagnostic criteria employed^[5] thus making clear regional and global comparisons — compared even with other regions of Iraq (tables 3 & 4) -- methodologically difficult. Insulin resistance and hyperandrogenism are two well-established pathophysiological phenomena in PCOS that are believed to support each other^[23,25], and although obesity is not an obligatory phenotypic feature of this syndrome it has repeatedly been found to aggravate both conditions^[24]. Third, reproductive morbidity in PCOS women is only a portion of the clinical landscape: Those long-term (metabolic, cardiovascular, oncological and psychological) consequences should optimally be addressed inter-disciplinarily from gynecology with endocrinology to mental health practitioners^[5,7]. Fourth, although international evidence-based guidelines (the 2023 recommendation included) provide a good basis for diagnosis and management, strong proof-of-concept for the adaptation of these recommendations into lower resource or culturally different settings such as Iraq is still limited. This translation is still a work in progress, grounded in ongoing local investment in health systems and implementation research^[31,52].

This shortcoming is true for much of the Iraqi and regional literature: reliance on small, clinic- or hospital-based samples rather than population-based sampling; diagnostic criteria applied inconsistently across studies. All of these elements by themselves, or in combination limit the reliability and comparability of prevalence estimates^[8+1]. Further, larger

population-based studies in both international and Iraq samples — notably for standardized lean PCOS phenotypes^[46] — which include mental health outcomes as part of routine clinical or epidemiological assessment are needed.

10.5. Limitations of This Review

This is a narrative review—it does not follow a formal systematic search protocol, quality-appraisal framework or PRISMA reporting structure, nor is it an original collection of new data, sampling of patients or statistical pooling. It should therefore be considered as a summary of previously published literature and not as new primary research or a systematic review. Due to the majority of Iraqi and Middle Eastern literature referenced being small, single-center studies based in tertiary centers that may not be representative of the population-level burden of PCOS within Iraq, conclusions derived from this regional overview must be regarded as hypothesis generating. As an additional, definitional limitation, this review was completed shortly after the October 2023 international consensus to rename PCOS as Polyendocrine Metabolic Ovarian Syndrome (PMOS)^[4]; because the evidence base cited almost exclusively predates this change, the clinical and research implications of this new nomenclature for Iraqi and regional practice could not be evaluated here and will require dedicated attention in future work.

11. Conclusion

Objective: Polycystic ovary syndrome (PCOS) is a common and heterogenous disorder with implications for reproductive, metabolic and psychological health. This includes common international diagnosis and management frameworks for example, the Rotterdam diagnostic criteria

and current guidelines. Although the regional data primarily accrued from those in Iran and Iraq suggested a high burden of PCOS, robust evidence is lacking as available studies are limited by small size, non-representative sampling and methodological heterogeneity. Establishing larger and more standardized community-based research in Iraq – compounded directly and/or indirectly by the shifting sociocultural understanding of the condition, as well as the provision of increased mental health services for women affected by this highly prevalent disorder – is therefore a key near- to medium-term priority for optimal detection, management and longer-term outcomes associated with PCOS within an Iraqi population.

To be followed through the above gaps are four sets of near-term priorities.

Research priorities: multi-center, population-representative prevalence studies using standardized Rotterdam criteria; longitudinal data on metabolic, cardiovascular and psychological outcomes; and structured evaluation of lean and non-hyperandrogenic (phenotype D) presentations which are likely to be especially neglected in Iraq [25,46].

Clinical priorities: Routine screening for depression/anxiety with concomitant metabolic assessment at diagnosis. Inclusion of 2023 recommendation in training curricula for obstetrics and gynecology trainees as gaps documented in application of three Rotterdam components [6]. Earlier recognition/metabolic/dermatological presentations (rather than infertility clinics alone) targeted at primary care and general gynecology.

Policy priorities – inclusion of PCOS-specific indicators in national reproductive-health surveillance conducted by the Iraqi Ministry of Health and Central Statistics Organization (section 3.3) and a dedicated funding mechanism for locally led, multi-center research due to the near-complete lack of population-based Iraqi data detected by this review.

Based on the above-informed attitude towards PCOS in Iraq, we recommend policies to expand this further: structured PCOS education is important within undergraduate and postgraduate obstetrics and gynecology curricula [6] as documented difficulty among trainees include correctly dissecting different aspects of the three Rotterdam components; and public-facing awareness materials addressing such stigma surrounding hirsutism, acne and infertility should be explicitly developed to instigate earlier care-seeking (section 9.2).

Combining those priorities is realistic within the health-system resourcing currently in Iraq, and, data generated from population-level implementation will generate a national (rather than simply clinical) evidence base for locally-calibrated management thresholds which align Iraqi practice with international evidence.

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