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## ABSTRACT

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# ANTI-MÜLLERIAN HORMONE AND HOMA-IR LEVELS IN PCOS PHENOTYPES

**Introduction:** Polycystic ovary syndrome (PCOS) is a common endocrinopathy affecting women, with variable prevalence (8–13%) and clinical manifestations, and it comprises four main clinical phenotypes. Elevated anti-Müllerian hormone (AMH) levels and insulin resistance are associated with ovarian dysfunction and metabolic disturbances in PCOS. The present study sought to describe the distribution of patients among PCOS phenotypes, evaluate AMH and HOMA-IR levels, and determine any correlation between them.

**Methods**: This was a prospective cross-sectional study of infertile women attending the outpatient infertility clinic in Mosul city. The ages of the included patients, who were enrolled between July 2023 and 2024, ranged from 18 to 39 years. PCOS was diagnosed according to the Rotterdam criteria, and patients were grouped into four phenotypes. On cycle days 2–5 (of a spontaneous or induced menstrual cycle), transvaginal ultrasound was performed to evaluate polycystic ovaries and follicle number; the ultrasound was repeated on cycle day 14 to assess the presence or absence of ovulation. Additionally, on cycle days 2–5, an early-morning blood sample was collected after 8 hours of fasting.

**Results**: The 132 infertile women with PCOS enrolled in the study demonstrated distinct phenotype prevalence: types A and D were more prevalent (57.57% and 30.31%, respectively) than types B and C. The duration of infertility was an important factor in differentiating between types, with the shortest duration (2.5 years) observed in type B patients compared to other types (4.37–4.45 years). AMH levels showed clear differences across groups, with Types A and C having the highest levels and Type B the lowest. LH levels were highest in Types C and D and lowest in Type B. FSH levels were higher in Type A compared to Types B, C, and D. The LH:FSH ratio was highest in Types C and D, while Type B had the lowest ratio. The highest insulin resistance (HOMA-IR

and fasting insulin levels) was observed in Type A patients. Type A also demonstrated a high miscarriage rate (26.3%), whereas Type B showed no history of miscarriage. Types C and D revealed a high rate of ovarian cysts (37.5%). A positive correlation was found between insulin resistance and BMI, as well as between AMH levels and ovarian follicle count.

**Conclusion**: The majority of patients fell into Types A and D, with high AMH and HOMA-IR values. A positive correlation existed between AMH and ovarian follicle number, as well as between HOMA-IR and BMI; therefore, both should be evaluated in all PCOS phenotypes. The study underscores the interconnected nature of metabolic and reproductive dysfunction in PCOS.

**Keywords**: Polycystic ovary syndrome, phenotypes, Anti-Müllerian hormone, HOMA-IR.

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## INTRODUCTION

Polycystic ovary syndrome (PCOS) is a common, non-curable, chronic endocrine disorder affecting 8–13% of women of reproductive age, with variable prevalence and clinical manifestations across different ethnic groups [1]. Although its symptoms typically begin between the ages of 18 and 39 years, approximately 75% of patients remain undiagnosed, resulting in delayed treatment in many cases [2,3]. PCOS has heterogeneous manifestations, including menstrual dysfunction, subfertility, hirsutism and/or acne, and obesity, and it is the leading cause of ovulatory dysfunction [4].

The pathophysiology of PCOS is influenced by altered neuroendocrine function and steroidogenesis, resulting in defective ovarian folliculogenesis and altered metabolism, insulin sensitivity, and insulin production, along with changes in adipose tissue activity, inflammatory factors, and sympathetic nerve function [5].

PCOS is diagnosed based on the Rotterdam consensus workshop group criteria (ESHRE/ASRM, 2004). The diagnosis of PCOS includes 2 out of the following 3 criteria [6]:

- Oligo-and/or anovulation.
- Clinical and/ or biochemical hyperandrogenism.
- Polycystic ovaries by ultrasound (PCO), with a threshold of follicle number of >20 in each ovary by trans-vaginal ultrasound or >10cm3 ovarian volume [7].

In 2012, the National Institutes of Health (NIH) consensus panel proposed a phenotypic PCOS classification based on Rotterdam criteria: Phenotype A (full-blown syndrome type) includes clinical or biochemical hyperandrogenism (HA), ovulatory dysfunction (OD), and PCO. Phenotype B (non-PCO, PCOS: HA+OD). Phenotype C (ovulatory type PCOS: HA+PCO). Phenotype D (non-hyperandrogenic type PCOS: OD+PCO) [8].

Anti-Müllerian hormone (AMH), a dimeric glycoprotein (a member of the transforming growth factor-β), is released from granulosa cells of antral follicles and results in a measurable serum level that is proportional to the number of developing ovarian follicles [9]. PCOS ovaries characteristically have huge number of follicles with the diameter of up to 7 mm when AMH has highest production [10]. Many PCOS patients have elevated AMH serum levels that could be responsible for the hormonal alterations observed in PCOS as it acts as a central regulator of the hypothalamic–pituitary–gonadal axis, resulting in a significant rise in LH pulse frequency and secretion, and is associated with increased ovarian androgen production by theca cells [11, 12]. PCOS patients have 5-fold higher AMH in their follicular fluid than non-PCOS patients, resulting not only from higher follicle count and a higher density of granulosa cells but also from higher AMH mRNA expression [13, 14]. Ultimately, this increased AMH will restrict ovarian folliculogenesis by lowering the circulating level and function of FSH on follicular recruitment [15], and it will impair follicular growth and dominant follicle selection [9], resulting in follicular arrest, a key point in the pathophysiology and onset of PCOS. Furthermore, the AMH inhibits FSH-induced aromatase activity, which contributes to the development of other PCOS clinical signs, such as androgen excess and insulin resistance (IR) [16].

Many epidemiological data show that PCOS is associated with an increased risk of impaired glucose tolerance, type 2 diabetes mellitus, and gestational diabetes during pregnancy. In addition, aging PCOS women are at increasing risk of metabolic dysfunction and cardiovascular diseases. Abdominal obesity has been associated with the severity of IR even in lean PCOS women. Due to these implications, it is essential to diagnose and treat IR in PCOS women as early as possible. Many markers have been proposed for diagnosing IR. However, quantitative assessment of IR in clinical practice remains a major challenge despite the current evidence of its high prevalence [17]. The Homeostasis Model Assessment (HOMA) is a method first described by Matthews et al. (1985). It was used to quantify IR from basal glucose and insulin levels [18]. Although no recommended screening test is available, HOMA-IR remains the best, most extensively validated biological surrogate marker for PCOS [17]. The present study aimed to describe the distribution of PCOS in infertile women in Mosul city among PCOS phenotype groups, and to compare the mean values of AMH and HOMA-IR between these groups and the correlation between them in PCOS.

**METHODS**

This was a prospective cross-sectional study of infertile women attending the outpatient infertility clinic in Mosul city. The study was approved by both the scientific and ethical committees of the College of Medicine, University of Mosul (Iraq). The ages of the included patients, who were enrolled between July 2023 and 2024, ranged from 18 to 39 years. PCOS was diagnosed according to the Rotterdam criteria, and the patients were grouped into four phenotypes.

On cycle days 2–5 (of a spontaneous or induced menstrual cycle), a transvaginal ultrasound was performed to assess polycystic ovaries and follicle number. The ultrasound was repeated on cycle day 14 to determine the presence or absence of ovulation. Additionally, on cycle days 2–5, an early morning blood sample was collected after 8 hours of fasting.

**Ethical approval:** The study was approved by the Medical Research Ethical Committee at the University of Mosul (Approval Letter Number UOM/COM/MREC/23-24/FEB7, dated 14.02.2024).

**Inclusion criteria:** Sub-fertile PCOS women according to Rotterdam criteria with different ages (18-39) and different Body Mass Index (BMI).

**Exclusion criteria:** PCOS women who were currently pregnant, PCOS women, or on insulin sensitizer drugs, those who had undergone laparoscopic drilling, and those with PCO due to other reasons like thyroid disease.

**Biochemical analysis:** Assessment of AMH, FSH, LH, and prolactin by (Roche e411) was done, and testosterone assessment was done only to confirm biochemical hyperandrogenism, in case of being absent clinically, for the diagnosis of PCOS. Assessment of fasting serum glucose (FSG) (Roche c111), fasting insulin (Roche e411), with HOMA-IR values derived mathematically by the equation:

**Insulin resistance:**

HOMA-IR = Insulin (mU/L)×glucose (mmol/L)/ 22.5 [19]. Patients with HOMA-IR > 2 were defined as having insulin resistance [20].

**Statistical analysis:** The analyses were conducted using IBM SPSS Statistics software (V8, UA). The data were subjected to descriptive statistics, ANOVA, and chi-square tests, depending on the data type (parametric or nonparametric). For differences between different PCOS populations, the F-test were applied. A Pearson correlation analysis was conducted between variables to identify possible associations between PCOS types and measured biochemical or demographic values. The differences existed whenever p-values were less than 0.05.

**RESULTS**

**Study sample:** A total of 132 infertile PCOS patients, diagnosed according to the Rotterdam criteria, aged between 15 and 35 years, were included. Their weight and height were measured, and BMI was calculated. The patients were classified into four PCOS phenotypes as described by the NIH consensus panel (2012). The clinical criteria for each phenotype and the distribution of patients among the phenotypic groups are presented in Table 1. The largest proportion of subfertile PCOS patients in our study belonged to Type A (57.57%) and Type D (30.31%), while Types B and C each represented 6.06%.

**Table 1:** Distribution of study sample across PCOS phenotypes

| PCOS phenotypes | Hyperandrogenism clinical and/or biochemical | Ovarian dysfunction, anovulation and/or oligomenorrhea | Ultrasound morphology of PCOS | Frequency N (%) |
|---|---|---|---|---|
| Type A | + | + | + | 76 (57.57) |
| Type B | + | + | - | 8 (6.06) |
| Type C | + | - | + | 8 (6.06) |
| Type D | - | + | + | 40 (30.31) |

**Demographic parameters:** Table 2 compares age, BMI, and duration of infertility across four groups (Type A, Type B, Type C, and Type D). The results of the statistical analysis, as indicated by the F-test and corresponding p-values, provide insights into differences between these parameters across the groups.

The average age in Type A is 25.89±5.41, Type B 25.75±3.24, Type C 26.38±4.66, and Type D 26.78±7.03, with no significant difference at a p-value of 0.1932. BMI values also show some variation across the groups, with Type A having the highest BMI (29.72±5.28) and Type B the lowest (25.78±1.28). Still, the *p*-value (0.0920) indicates that these differences are not statistically significant at the 0.05 level. In contrast to age and BMI, the duration of infertility shows a significant difference across the groups.

**Table 2:** Comparison of age, BMI, and duration of infertility across groups

| Parameter | Groups | | | | F test | P value |
|---|---|---|---|---|---|---|
| | Type A | Type B | Type C | Type D | | |
| Age | 25.89±5.41 | 25.75±3.24 | 26.38±4.66 | 26.78±7.03 | 1.028 | 0.1932 |
| BMI | 29.72±5.28 | 25.78±1.28 | 28.3±4.32 | 28.67±4.54 | 1.384 | 0.092 |
| Duration of infertility | 4.44±2.59 | 2.50±1.19 | 4.37±4.77 | 4.45±2.65 | 3.234 | 0.047* |
| Right ovary number | 29.55±8.95 | 8±0.76 | 24.63±10.47 | 31±4.87 | 4.253 | 0.0245* |
| Left ovary number | 29.42±7.98 | 8.25±0.89 | 26.25±8.96 | 24.88±9.64 | 3.913 | 0.0184* |

Note. * – significant difference between groups (p-value ≤0.05)

**Infertility types:** Type B has the shortest duration of infertility (2.50±1.19 in Type B), while the other groups have durations ranging from 4.37 to 4.45 years. Longer period in Type D, A, then C. The F-test for the duration of infertility is 3.234, with a *p*-value of 0.0470, which is less than 0.05, indicating a statistically significant difference.

**Ultrasonography study:** Follicle number in both right and left ovaries shows significant differences between the groups, with *Type A (*29.55±8.95, 29.42±7.98) and Type D (31±4.87, 24.88±9.64) having a higher number than Type C(24.63±10.47, 26.25±8.96) for both ovaries, respectively, with F-test 3.913 at p-value 0.0184 *(≤0.05).*

Table 3 represents a comparison of the type of infertility (Primary and Secondary) and the presence of miscarriage across four groups (Type A, Type B, Type C, and Type D). The majority of participants in each group experience primary infertility, with Type A (71.1%), Type B (75%), Type C (75%), and Type D (60%). The *p*-value for this comparison is 0.024, which is less than the significance threshold of 0.05. This indicates that primary infertility is the main presentation of infertility in all PCOS phenotypes, with significant differences among the groups. Secondary infertility is a less prevalent presentation among PCOS patients, with Type D having the highest rate among the groups.

**Table 3:** Comparison of type of infertility and presence of miscarriage across groups

| Parameter | | Groups n(%) | | | | P value |
|---|---|---|---|---|---|---|
| | | Type A | Type B | Type C | Type D | |
| Type of infertility | Primary | 54 (71.1%) | 6 (75%) | 6 (75%) | 24 (60%) | 0.024* |
| | Secondary | 22 (28.9%) | 2 (25%) | 2 (25%) | 16 (40%) | |
| History of miscarriage | Yes | 20 (26.3%) | 0 (0%) | 2 (25%) | 6 (15%) | 0.018* |
| | No | 56 (73.7%) | 8 (100%) | 6 (75%) | 34 (85%) | |
| History of ovarian cyst | Yes | 18 (23.7%) | 2 (25%) | 3 (37.5%) | 15 (37.5%) | 0.019* |
| | No | 58 (76.3%) | 6 (75%) | 5 (62.5%) | 25 (62.5%) | |

Note. * – significant difference between groups (p value ≤0.05), AMH = anti-Müllerian hormone, LH = luteinizing hormone, FSH = follicle stimulating hormone

The table also presents data on reporting miscarriage across the groups. Type A(26.3%) has the highest proportion of women with a history of miscarriage, Type B (0%), Type C (25%), and Type D (15%). The *p*-value for this parameter is 0.018, which is also less than 0.05, indicating a significant difference between the groups.

The presence of ovarian cysts is reported in 23.7% of women in Type A, 25% in Type B, 37.5% in Type C,

and 37.5% in Type D. The F-test for ovarian cysts yields a significant *p*-value of 0.0192, indicating that the prevalence of ovarian cysts differs significantly between the groups. Types C and D show the highest prevalence among the groups.

**Biochemical analysis:** Table 4 compares several hormonal parameters: antiMüllerian hormone (AMH), luteinizing hormone (LH), follicle-stimulating hormone (FSH), LH:FSH ratio, and prolactin across four groups (Type A, Type B, Type C, and Type D). The table includes F-test results and p-values to indicate statistical significance. AMH level showed a clear difference across the groups, with Type A (3.68±1.85) ng/ml and Type C (3.89±2.09) ng/ml having the highest levels, while Type B (2.35±0.58) ng/ml had the lowest. The F-test result for AMH is 3.05, with a p-value of 0.0314, which is less than the significance threshold of 0.05. LH levels were highest in Type C (10.24±3.64) mIU/ml and Type D (9.17±5.06) mIU/ml, while Type B had the lowest levels (4.3±0.66) mIU/ml. The F-test for LH yields a p-value of 0.0184, indicating a significant difference between the groups. FSH levels are higher in Type A (7.39±1.71) mIU/ml compared to Type B (5.1±0.73) mIU/ml, Type C (6.23±0.33) mIU/ml, and Type D (6.93±1.7) mIU/ml. The F-test result for FSH is 8.46 with a p-value of 0.0036, indicating a highly significant result. LH:FSH Ratio is highest in Type C (1.653±0.622) and Type D (1.366±0.698), while Type B has the lowest ratio (0.85±0.12). The F-test for the LH:FSH ratio gives a result of 5.38 with a p-value of 0.0142, indicating a significant difference. Prolactin levels are highest in Type C (29.91±8.83) ng/ml, followed by Type A (22.66±11.58) ng/ml and Type D (22.14±14.01) ng/ml, while Type B has the lowest level (12.75±3.58) ng/ml. The F-test result for Prolactin is 4.11, with a p-value of 0.0268, indicating a significant difference between the groups.

**Table 4:** Comparison of hormonal levels across PCOS phenotypes

| Parameters | Groups (mean±SD) | | | | F test | P value |
|---|---|---|---|---|---|---|
| | Type A | Type B | Type C | Type D | | |
| Anti-Müllerian hormone (AMH) | 3.68±1.85 | 2.35±0.58 | 3.89±2.09 | 3.59±3.31 | 3.05 | 0.0314* |
| Luteinizing hormone (LH) | 8.78±4.23 | 4.3±0.66 | 10.24±3.64 | 9.17±5.06 | 4.43 | 0.0184* |
| Follicle stimulating hormone (FSH) | 7.39±1.71 | 5.1±0.73 | 6.23±0.33 | 6.93±1.7 | 8.46 | 0.0036* |
| LH:FSH Ratio | 1.209±0.541 | 0.85±0.12 | 1.653±0.622 | 1.366±0.698 | 5.38 | 0.0142* |
| Prolactin | 22.66±11.58 | 12.75±3.58 | 29.91±8.83 | 22.14±14.01 | 4.11 | 0.0268* |

Note. * – significant difference between groups (p value ≤0.05), AMH = anti-Müllerian hormone, LH = luteinizing hormone, FSH = follicle stimulating hormone

**Metabolic parameters:** Table 5 presents a comparison of metabolic parameters – HOMA-IR, FSG, and fasting insulin across four groups (Type A, Type B, Type C, and Type D). The table includes F-test results and *p*-values, highlighting significant differences across all three parameters. HOMA-IR (Homeostasis Model Assessment of Insulin Resistance) mean values show the highest levels in Type A (3.67±2.89), followed by Type D (2.67±1.87) and Type C (2.63±1.28), while Type B has the lowest value (2.12±0.83). The F-test result for HOMA-IR is 3.845 with a *p*-value of 0.0472, which is below the 0.05 significance threshold, indicating a significant difference between the groups and suggesting that insulin resistance, as measured by HOMA-IR, is more pronounced in Type A than in the other groups. FSG mean levels are highest in Type C (93.38±4.87) mg/dl and Type D (83.97±22.18) mg/dl, while Type A (85.45±8.86) mg/dl and Type B (84.5±5.1) mg/dl have lower levels. The F-test for FSG results in a value of 3.935 with a *p*-value of 0.0392, indicating a significant difference between the groups.

**Table 5:** Comparison of metabolic parameters across PCOS phenotypes

| Parameter | Groups | | | | F test | P value |
|---|---|---|---|---|---|---|
| | Type A | Type B | Type C | Type D | | |
| HOMA-IR | 3.67±2.89 | 2.12±0.83 | 2.63±1.28 | 2.67±1.87 | 3.845 | 0.0472* |
| FSG | 85.45±8.86 | 84..5±5.1 | 93.38±4.87 | 83.97±22.18 | 3.935 | 0.0392* |
| Fasting insulin | 17.85±12.23 | 10.25±2.15 | 11.56±5.4 | 14.21±10.61 | 5.294 | 0.0257* |

Note. * – significant difference between groups (p value ≤0.05), AMH = anti-Müllerian hormone, LH = luteinizing hormone, FSH = follicle stimulating hormone

Fasting insulin mean levels are highest in Type A (17.85±12.23) µU/ml, followed by Type D (14.21±10.61) µU/ml and Type C (11.56±5.4) µU/ml, with Type B showing the lowest levels (10.25±2.15) µU/ml. The F-test for Fasting Insulin yields a result of 5.294 with a *p*-value of 0.0257, indicating a significant difference between the groups.

**Pearson Correlation analysis:** In Table 6, Pearson Correlation testing for the linear relationship between parameters in the study sample. There was a significant positive correlation between HOMA-IR and BMI (.256*, p = 0.05), and a nonsignificant negative correlation with Age and AMH. AMH shows no significant correlation with HOMA-IR, age, or BMI.

**Table 6:** Pearson correlation between age, BMI, HOMA-IR, and anti-Müllerian hormone

| Correlation (n=132) | Test | AGE | BMI | HOMA-IR | AMH |
|---|---|---|---|---|---|
| AGE | Pearson Correlation | 1 | 0.21* | -0.029 | -0.015 |
| | Sig. (2-tailed) | | 0.042 | 0.78 | 0.888 |
| BMI | Pearson Correlation | 0.21* | 1 | 0.256* | -0.033 |
| | Sig. (2-tailed) | 0.042 | | 0.013 | 0.752 |
| HOMA-IR | Pearson Correlation | -0.029 | 0.256* | 1 | -0.028 |
| | Sig. (2-tailed) | 0.78 | 0.013 | | 0.792 |
| Anti-Müllerian hormone | Pearson Correlation | -0.015 | -0.033 | -0.028 | 1 |
| | Sig. (2-tailed) | 0.888 | 0.752 | 0.792 | |

Note. * – correlation is significant at the 0.05 level (2-tailed). AMH = anti-Müllerian hormone, LH = luteinizing hormone, FSH = follicle-stimulating hormone

Table 7 shows a positive linear Pearson correlation between AMH and the number of ovarian follicles, with a significant p-value at the 0.01 level. No significant correlation was found between HOMA-IR and the number of ovarian follicles.

**DISCUSSION**

The phenotypic classification of PCOS patients according to the NIH 2012 consensus panel allows assessment of patients based on their clinical and metabolic condition. Accordingly, subfertile PCOS women in our study were classified phenotypically into four groups. Type A, the full-blown PCOS syndrome, represented the largest group (57.57%) in our study. Groups B (6.06%) and C (6.06%), representing the non-PCOS and ovulatory types, respectively, constituted only a small proportion. Meanwhile, Type D (30.31%), the non-hyperandrogenic type, represented the second major group in our study.

**Table 7:** Pearson correlation among AMH, HOMA-IR, right and left follicle number

| Correlation (n=132) | | HOMA-IR | AMH | Right ovary number | Left ovary number |
|---|---|---|---|---|---|
| HOMA-IR | Pearson Correlation | 1 | -0.028 | -0.076 | -0.075 |
| | Sig. (2-tailed) | | 0.792 | 0.464 | 0.474 |
| AntiMüllerian hormone | Pearson Correlation | -0.028 | 1 | 0.417** | 0.433** |
| | Sig. (2-tailed) | 0.792 | | 0.0001 | 0.0001 |
| Right ovary number | Pearson Correlation | -0.076 | 0.417** | 1 | 0.826** |
| | Sig. (2-tailed) | 0.417 | 0.0001 | | 0.0001 |
| Left ovary number | Pearson Correlation | -0.075 | 0.433** | 0.826** | 1 |
| | Sig. (2-tailed) | 0.474 | 0.0001 | 0.0001 | |

Note. * – correlation is significant at the 0.05 level (2-tailed). ** – correlation is significant at the 0.01 level (2-tailed)

Similar results were reported in a study conducted in Basra, Iraq, by Falh et al. (2024), which showed a higher percentage of Type A (46%), followed by Type D (34%), Type C (16%), and Type B (3.2%) [21]. Comparable findings were also reported in a study from Brazil by Tavares et al. (2019), where Type A (54.1%) and Type D (19.8%) constituted the major groups, while Type B (11.7%) and Type C (14.4%) were less common [22].

Type A was the most frequent presentation in many studies. In contrast, Type C was observed at low frequencies in several studies [23–26]. The prevalence

of Type D, however, was highly variable across studies. It was the least prevalent in a study by Ladrón de Guevara, which evaluated 220 Chilean and 206 Argentinian women, where Type D accounted for 1% and 10% of PCOS cases, respectively [27].

These variations in phenotype distribution among different populations may be attributed to ethnic differences [22, 23, 27]. Furthermore, the differing distribution of PCOS phenotypes across studies may be explained by variations in study populations (such as subfertile women, as in our study, obese individuals, or general PCOS populations), as well as differences in genetic backgrounds, lifestyle, and dietary factors [23].

The mean age of PCOS sub-fertile women in our study was between 25.89 and 26.78 years. The young age of the sample is attributed to the fact that PCOS diagnosis declines with age due to a reduction in the number of ovarian follicles. There was no significant difference in the mean age between PCOS phenotype groups in our study, consistent with many previous studies [24].

Complete understanding of PCOS pathogenesis seems out of reach but some clearly defined aspect is that PCOS is closely associated with obesity which mediates its effect through insulin resistance and its role in development of PCOS manifestation and phenotypes [28], this is also evident in our study, the 4 studied groups of PCOS phenotypes have increased BMI >25kg/m$^2$ and tend to be obese (cut-off value >25kg/m$^2$) [24], with no significant difference between these groups with highest value in Type A (29.72±5.28) and lowest in Type B (25.78±1.28), the effect of BMI may have additive effect on PCOS manifestation which is agreed by a study depends on mendelian randomization revealing that increasing BMI seems to be causal for the PCOS but being a PCOS does not appear to increase BMI [29], also increased BMI has additive effect on PCOS by increasing free androgen index through reduction in sex hormone binding globulin rather than increasing serum androgen levels which had an additive effect on manifestation of PCOS [30].

In our study, AMH showed significant differences across PCOS phenotypes. Type A (3.68±1.85) ng/ml and Type D (3.59±3.31) ng/ml have the highest AMH level. In contrast, Type C (3.89±2.09) ng/ml was a minor group with lower BMI, which may affect AMH levels due to the inverse relationship between AMH and BMI. Type B was the lowest level (2.35±0.58) ng/ml and also a minor group in the study. These results were similar to those of Falh et al. (2024), who reported that Type A and Type D had the highest AMH values [21]. AMH level was well known to correlate with PCOS clinical, hormonal, and metabolic state. Still, there were no well-established cut-off values for the diagnosis of PCOS. A study by Malhotra et al. (2023) found that the majority of high AMH cases belong to PCOS types A and D, with a cut-off value of > 6.06 ng/mL selected for the diagnosis of Type A and for detecting those with worse clinical, endocrinological, and metabolic parameters [31].

Another study by Gursu et al. (2022) found that Type A had higher AMH and BMI values and identified a lower AMH cut-off of 3.105 ng/mL, with 90.8% sensitivity and 90% specificity. It was accepted that a higher AMH level was associated with greater clinical, endocrine, and metabolic severity of the disorder [32].

In our study, all PCOS phenotypes showed IR, as evidenced by HOMA-IR values >2, with a significant difference between groups. Type A has the highest HOMA-IR (3.67±2.89), while Type B has the lowest value (2.12±0.83). This result of high IR in all studied PCOS phenotype groups could be explained since our study group was obese (BMI >25); it was reported that obese PCOS women had a 2.74-fold increased value of HOMA IR [22]. It was also well- established that obesity worsens PCOS metabolic state [33], and although our PCOS study groups have no significant difference in BMI. However, there is still a significant difference in HOMA-IR between these groups, mainly due to the variable severity of PCOS manifestations across the 4 phenotypes, resulting in different IR values [24]. Our study group has higher fasting insulin in Type C, while fasting insulin was higher in Type A, with a significant difference between the groups.

Since the 4 groups have IR assessed by HOMA-IR values >2, which is responsible for many PCOS-linked problems, such as obesity, anovulation, subfertility, and type 2 diabetes, all PCOS phenotypes should be screened for IR.

Our study shows that IR was highest in Type A similar to result from Sobti et al. (2017), in New delhi [34], while other Iranian study by Rahmatnezhad et al. (2023), claim that type B exhibit highest IR [23], this variation in the result of IR among different phenotypes in different studies could be due to ethnic variation or due to different genetic inheritance, or different life style and dietary habits. Also the variation in the mean age of the study sample may elicit different clinical and metabolic environment in PCOS phenotypic groups, in our study was its lowest value in Type B (25.75±3.24 years) and highest in Type D (26.78±7.03 years) but in a study in Latin America (an area with tendency of high IR) most of women in their study were less than 25 years which made their evaluation of Insulin resistance was difficult to detect at these young age [22].

Our study identifies a significant positive linear correlation between AMH values and follicle number in both ovaries and a non-significant, negative correlation

with HOMA-IR, Age, and BMI. These were consistent with a study by Butt et al. (2022) [35]. AMH is closely related to PCOS endocrinopathy and can be selected as a predictor and marker for its severity and diagnosis. It can be used instead of PCO morphology when accurate visualization of PCO is difficult, to provide early diagnosis, especially in virgin and obese patients [31, 32, 35].

In our study, there was a significant positive correlation between HOMA-IR and BMI (p=0.05) and a negative but significant correlation with AMH. HOMA-IR has been linked to BMI in many studies, including that by Hussein et al. (2023) [36]. Metabolic complications in PCOS, and also HOMA-IR, have been linked to ovarian sensitivity during infertility treatment [37]. So, HOMA-IR assessment is mandatory in PCOS patients, regardless of AMH levels or BMI, as IR is a prominent feature that should be addressed across all PCOS phenotypes.

**CONCLUSION**

The majority of PCOS patients in our study were in Type A and Type D phenotypes. All PCOS phenotypes exhibit high AMH and HOMA-IR values, with significant variation; the highest values are observed in Type A. AMH shows a positive, significant correlation with ovarian follicle number. In contrast, HOMA-IR shows a positive, significant correlation with BMI.

## ETHICAL CONSIDERATIONS

The study was approved by the Medical Research Ethical Committee at the University of Mosul (Approval Letter Number UOM/COM/MREC/23-24/FEB7, dated 14.02.2024).

## AUTHOR CONTRIBUTIONS

The author designed the study, performed the experiments, collected the data, statistically analyzed the data, and wrote the results section and the manuscript.

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None.

## CONFLICT OF INTEREST

The authors have no conflict of interest to declare.

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## ARTIFICIAL INTELLIGENCE DISCLOSURE

No AI was used to generate the written text.

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