

Role of Inhibin B and Ratio of Luteinizing: Follicle-Stimulating Hormones in Phenotyping Polycystic Ovarian Syndrome

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Abstract:

Background: Polycystic ovary syndrome is among the leading causes of fertility-related problems and menstrual irregularities in women of reproductive age. The granulosa cells of the developing pre-antral and antral follicles produce inhibin B, which triggers chemical responses in the ovaries. Inhibin B is most often observed in the follicular phase when levels peak early and then decline over time

Objectives: This study was designed to investigate the role of serum inhibin B and the Luteinizing Hormone / Follicle Stimulating Hormone ratio in differentiation between the different phenotypes of polycystic ovary syndrome as well as to define the predominant PCOS phenotype.

Methods: This cross-sectional research was conducted in the Department of Biochemistry, College of Medicine, University of Baghdad from November 2023 to March 2024. The study included 111 women, ranging in age from 18-40 years. Among these, 91 women were diagnosed with polycystic ovary syndrome (PCOS) based on the 2003 Criteria for the Rotterdam Consensus, and the other 20 were healthy women. Investigations included serum levels of inhibin B using the Enzyme Linked Immunosorbent Assay technique, follicle Stimulating Hormone, luteinizing Hormone and prolactin using Tosoh AIA-2000 Automated Immunoassay, to calculate the LH/FSH ratio.

Results: phenotype A was observed to be the predominant PCOS phenotype, while phenotype B was the rare form. The mean \pm SEM values of the inhibin B levels for the phenotypes B (26.07 ± 0.23 pg/ml, $p < 0.001$), C (25.96 ± 1.68 pg/ml, $p < 0.0001$), and D (37.51 ± 2.31 pg/ml, $p < 0.0001$), respectively, were significantly lower than those of the control women (57.68 ± 2.07 pg/ml). However, the mean \pm SEM value of inhibin B of phenotype A (50.46 ± 7.12 pg/ml) was comparable to that of controls. The mean value of the LH levels of phenotype A (7.12 ± 0.76 μ IU/ml) showed significantly higher numbers than those of the control women (4.59 ± 0.38 μ IU/ml, $p = 0.03$). Furthermore, the mean values of the LH/FSH ratio were significantly elevated in phenotypes A ($p = 0.001$) and B ($p = 0.04$) as compared to the controls.

Conclusion: Serum Inhibin B level and LH/FSH ratio can be used to distinguish between the different phenotypes of PCOS.

Keywords: Follicle stimulating hormone; Luteinizing hormone ratio; Inhibin B; Prolactin; Phenotypes; Polycystic ovary syndrome.

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Introduction:

Polycystic ovary syndrome (PCOS) is one of the leading causes of fertility problems and menstrual irregularities in women of reproductive age (1–4). The World Health Organization (WHO) estimates that 4-8% of the global population has PCOS (5). Until recent times, the most commonly used diagnostic tool for PCOS is the Rotterdam criteria, which include polycystic ovarian morphology (PCOM), hyperandrogenism (HA), and oligomenorrhea and anovulation (OM) (6,7). If the patient meets two of the three criteria cited above hyperandrogenism can be diagnosed or oligo-

amenorrhea when alternative etiologies have been ruled out (6,8). The Rotterdam criteria specify four main phenotypes of PCOS based on the clinical signs and symptoms (9). To get better outcomes, the phenotypes in PCOS patients can be identified using the appropriate methodology (10). Phenotype A was defined as oligomenorrhea -anovulation, HA and PCOM on ultrasound; phenotype B was identified as oligomenorrhea -anovulation and HA; phenotype C was described as HA and PCOM on ultrasound; and phenotype D was diagnosed as oligomenorrhea -anovulation and PCOM on ultrasound (6). In the Rotterdam consensus conference, it was agreed that subsequent sonographic definitions of the morphology of polycystic ovarian morphology

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(PCOM): expanded the ovarian volume ($\geq 10\text{cm}^3$) or more than 12 follicles per ovary, each measuring between 2 and 9 mm (6). The symptoms and signs showed definite and wide variations among women with PCOS (9). Inhibins, which belong to the transforming growth factor- β superfamily, comprise two constituents held together by disulfide bonds (11). These two constituents consist of an α -subunit and a β A-subunit or a β B-subunit, which together generate inhibin A or inhibin B, respectively, (12,13). The quantity of inhibin released into the ovaries is linked to the menstrual cycle (11). Subsequently, this hormone triggers chemical responses in the ovaries and testes of both sexes, in the granulosa and Sertoli cells, respectively. Inhibin B is most often observed in the follicular phase when levels peak early and then decline over time (11). Furthermore, patients with PCOS have lower FSH levels, which increase the LH/FSH ratio, boost the androgen synthesis from theca cells in the ovarium and, ultimately, create excess androgen (14). This disorder will halt new follicular growth and persistent anovulation (14). The aggregation of the tiny antral follicles results in the development of polycystic ovarian morphology (15–17). The differentiation between the four different phenotypes of PCOS is dependent on history of a woman regarding her cycle regularity, clinical examination considering hirsutism and acne, and ultrasonic study of ovaries. Hormones changes play important role in pathogenesis of PCOS. The aim of this study was to investigate the role of serum inhibin B and the Luteinizing Hormone (LH) / Follicle Stimulating Hormone (FSH) ratio in differentiation between the different phenotypes of polycystic ovary syndrome and to define the predominant PCOS phenotype.

Patients and Methods:

This case-control study was performed at the Department of Biochemistry, University of Baghdad College of Medicine, the Medical City of Baghdad Teaching Hospital, Baghdad, Iraq from November 2023 to March 2024. The study included 111 women in the age range of 18 to 40 years, of whom 91 had been previously diagnosed with polycystic ovarian syndrome (PCOS) by a consultant gynecologist and 20 healthy women as the control group. The PCOS women were sub-classified into four groups; (A, B, C, and D, respectively) based on their phenotypic characteristics which include polycystic ovarian morphology (PCOM), hyperandrogenism (HA), and oligomenorrhea and anovulation (OM) (6,10). The number of patient groups was limited because they were sub-classified into four groups as well as due to the limited time scheduled for study. This study was approved by the scientific and ethical committees of the Department of Biochemistry, College of Medicine, University of Baghdad (1500/ 26-11-2023); Ethical approval was also obtained from Baghdad Teaching Hospital, Medical City, Ministry of Health. Verbal consent was obtained from each of the women included as participants in this study. The

inclusion criteria for women with PCOS involves the patient satisfying at least two criteria of the 2003 Rotterdam Consensus and they fall in the age of range 18-40 years. Polycystic ovarian morphology (PCOM) and hyperandrogenism (HA) are two characteristics of polycystic ovarian syndrome (8). According to this agreement, the patient must satisfy a minimum of two of the three main criteria listed below to be diagnosed with PCOS: (1) Anovulatory oligomenorrhea (2) hyperandrogenism (clinical or biochemical results), (3) polycystic ovaries (identified by ultrasound); also, other illnesses related to excess androgen, such as congenital adrenal hyperplasia, should be ruled out. After ruling out Cushing's disease, congenital adrenal hyperplasia, hyperprolactinemia, and androgen-secreting tumors, the patient is diagnosed with PCOS if at least two of these three criteria are met. Also, based on the Rotterdam criteria, four distinct phenotypes are associated with this syndrome: Hyperandrogenism and PCO and oligomenorrhea (A), oligomenorrhea and hyperandrogenism(B), PCO and hyperandrogenism(C), and oligomenorrhea and PCO (D) (6). The Exclusion criteria include those women on oral contraceptives at the time of blood draw and who have other diagnoses that mimic PCOS (i.e. prolactinoma, premature ovarian failure, congenital adrenal hyperplasia), thyroid gland dysfunctions, liver disease, kidney disease, and cancers. From each of the PCOS and control women, 5 ml of blood was drawn from a peripheral vein. This blood sample was left undisturbed to clot for 15 minutes and then centrifuged for 10 minutes at 2500 rpm. The separated serum was stored at -20 C until the time for measurements. Serum investigation included inhibin B level assessments using the semiautomatic ELISA Reader Huma, Reader by Human Diagnostics, a German company, and Washer (COMBIWASH) by HUMAN, Germany.

Statistical analysis:

The Statistical Package for Social Sciences (SPSS, version 25) was used for data analysis. The mean and standard error of the mean (SEM) were used to present the data obtained, and every statistical analysis was based on the data (18,19). The ANOVA test was used to evaluate the differences in the mean levels of the numeric data between more than two variables. The Area Under the Curve (AUC) and Receiver Operator Characteristic (ROC) were calculated and the cutoff value, sensitivity, and specificity of the parameters were obtained to differentiate between the four PCOS phenotypes and the control women as well as among the phenotypes themselves. Utilizing the Pearson correlation regression (r) the relationship between the numerical data was evaluated. P -value of less than 0.05 was considered statistically significant.

Results:

Table 1 presented the distribution of the four phenotypes of PCOS with phenotype A as the predominant one. Table 2 presented the mean \pm SEM

values of age and body mass index (BMI) of the studied groups. The mean values of age of Phenotypes A (25.76 ± 0.65 year, $p=0.01$), C (24.80 ± 1.46 year, $p=0.02$), and D (25.94 ± 0.97 year, $p=0.02$) were significantly higher than that of controls. However, there were insignificant differences in the mean value of age among the four phenotypes of PCOS women. The mean values of BMI of Phenotypes A (31.15 ± 1.04 Kg/m², $p=0.001$), C (28.91 ± 1.24 Kg/m², $p=0.01$), and D (32.02 ± 0.98 Kg/m², $p=0.001$) were significantly higher than that of the control group along with non-significant differences among the phenotype groups. Table 3 shows the mean (\pm SEM) values of serum inhibin B, LH, FSH, prolactin, and the LH/FSH ratio of the studied phenotypes of PCOS and control women. The mean values of inhibin B levels of phenotypes B (26.07 ± 0.23 pg/ml, $p=0.001$), C (25.96 ± 1.68 pg/ml $p=0.0001$), and D (37.51 ± 2.31 pg/ml, $p=0.0001$) were significantly lower than those of control women. In addition, the mean value of Inhibin B levels of phenotype C was significantly lower than that of phenotype D ($p=0.001$) and phenotype A ($p=0.049$). The mean value of LH levels of phenotype A was significantly higher than that of control women ($p=0.03$). In addition, the mean values of LH of phenotypes B, C, and D were higher than those of controls but did not reach a significant level. There was a non-significant difference in mean values of LH among the four phenotypes of PCOS. The mean values of serum FSH levels were significantly lower in phenotypes B ($p=0.02$) and C ($p=0.001$) when compared with control women. There were non-

significant differences among the four phenotypes of PCOS regarding mean serum value of FSH. The mean values of LH/FSH ratio were significantly elevated in phenotypes A ($p=0.001$) and B ($p=0.04$) when compared to controls. The mean value of serum prolactin was significantly increased in phenotype D in comparison with phenotype C ($p=0.001$) and control women ($p=0.001$), without any other significant differences. The study also found a significant positive correlation between serum LH and LH/FSH ratio in phenotype A ($r=0.64$, $p=0.01$), phenotype C ($r=0.94$, $p=0.0001$), and phenotype D ($r=0.92$, $p=0.0001$). However, there was no other significant correlation among the studied parameters in other groups. Also, the ROC and AUC study revealed that inhibin B at (cutoff ≤ 37.85 pg/ml) was the best measure for differentiation of phenotype C from controls with AUC value 0.997 (sensitivity=100 and specificity=95). The LH/FSH ratio was the best measure for differentiation of phenotype A from controls with an AUC value of 0.739 (sensitivity =69.05 and specificity=75.00). In differentiation between phenotypes C and D, inhibin B has AUC=0.80 at cutoff (< 37.851 ng/ml) with (sensitivity =100.0 % and specificity =48.39 %). Inhibin B also has AUC=0.805 at cutoff (< 26.502 ng/ml) with (sensitivity= 100.0% and specificity =77.42 %) in differentiation of phenotypes B and D. In addition, inhibin B has AUC=0.713 at cutoff (< 28.591 ng/ml) with (sensitivity= 64.28 % and specificity = 73.33 %) in differentiation of phenotypes A and C.

Table 1: Frequency and percentage of phenotypes distribution of the entire polycystic ovary syndrome women (Total number 91 women)

Phenotype A No. (%)	Phenotype B No. (%)	Phenotype C No. (%)	Phenotype D No. (%)
42 (46)	3 (3.2)	15 (16)	31 (34)

Table 2: Mean \pm SEM values of age and body mass index of polycystic ovarian syndrome Phenotypes and controls

Parameter	Phenotype A (n=42)	Phenotype B (n=3)	Phenotype C (n=15)	Phenotype D (n=31)	Control (n=20)
Age (year)	$25.76 \pm 0.65^*$	29.33 ± 1.86	$24.80 \pm 1.46^*$	$25.94 \pm 0.97^*$	29.95 ± 1.42
BMI (Kg/m ²)	$31.15 \pm 1.04^*$	28.35 ± 1.91	$28.91 \pm 1.24^*$	$32.02 \pm 0.98^*$	25.05 ± 0.64

ANOVA and *t*-test: ●: Significant decrease in mean values of age of phenotypes A ($P=0.01$), C ($P=0.02$) and D ($P=0.02$) and a significant increase in BMI in phenotypes A ($P=0.001$), C ($P=0.01$) and D ($P=0.001$) than in controls.

Table 3: Mean \pm SEM values of inhibin B, luteinizing hormone (LH), follicle stimulating hormone (FSH), prolactin, and LH/FSH Ratio of polycystic ovarian syndrome groups and controls

Parameter	Phenotype A (n=42)	Phenotype B (n=3)	Phenotype C (n=15)	Phenotype D (n=31)	Control (n=20)
Inhibin B (Pg/ml)	50.46 ± 7.12	$26.07 \pm 0.23^*$	$25.96 \pm 1.68^*$	$37.51 \pm 2.31^*$	57.68 ± 2.07
LH (μ IU/ml)	$7.12 \pm 0.76^*$	4.30 ± 1.22	4.69 ± 0.84	5.52 ± 0.72	4.59 ± 0.38
FSH (μ IU/ml)	7.71 ± 0.65	$4.90 \pm 1.10^*$	$6.37 \pm 0.42^*$	7.09 ± 0.45	8.44 ± 0.49
Prolactin (ng/ml)	13.30 ± 1.29	11.60 ± 1.81	10.14 ± 1.16	$16.56 \pm 1.57^*$	9.90 ± 0.69
LH/FSH ratio	$1.06 \pm 0.11^*$	$0.95 \pm 0.24^*$	0.80 ± 0.16	0.85 ± 0.14	0.58 ± 0.05

ANOVA and *t*-test: ●: Significant decrease in mean values of inhibin B of phenotypes B ($P=0.001$), C ($p=0.0001$) and D ($p=0.0001$) than in controls, a significant decrease in inhibin B in phenotype C than in phenotype D ($p=0.001$) & phenotype A ($p=0.049$) * significant increase in LH in phenotypes A ($p=0.03$) than in controls. ♦: Significant decrease in FSH in phenotypes B ($p=0.02$) and C ($p=0.001$) compared to control. ■: Significant increase in prolactin in phenotype D in comparison with phenotype C ($p=0.01$) and control women ($p=0.001$). *: LH/FSH ratio significantly elevated in phenotypes A ($p=0.001$) and B ($p=0.04$) when compared to controls.

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Discussion:

The mean age value of the whole group of PCOS women in the present study was found to be 25.5 years that of the BMI was 30.69 Kg/m², which concurred with the findings reported by Carmina and Lobo (2022) They reported that the mean age of their PCOS women was 24.2 years (20), which differed from the findings of another study, where the mean age of their PCOS patients was found to be 28.2 years and BMI was 26.33 Kg/m² (21). The current study identified that phenotype A was the commonest one, after which was phenotype D of the women with PCOS, this finding corresponded with the previous study conducted by Malhotra *et al.* (2023), who found that phenotypes A and D were the commonest of the PCOS phenotypes (21). Moreover, Si *et al.* (2023) observed that phenotype B was the rarest subgroup (4%) among the women with PCOS (22). The current research found that the mean values of serum Inhibin B of the phenotypes (B, C, and D) were significantly lower than those of the controls, which was in agreement with the findings of Hussein *et al.* (2023) and Obaid *et al.* (2022) who recorded significantly lower levels of serum inhibin B in the women with PCOS women than in the controls (11, 23). However, Fazil *et al.* (2023) did not find any significant variation in the levels of serum inhibin B between the PCOS women and controls (24). In contrast, Farman *et al.* (2021) reported that the serum inhibin B levels of PCOS women were significantly higher than those of the controls (25). An essential function of inhibin B is to regulate ovarian function (26). Additionally, it is a crucial candidate gene for research on human ovarian function (26). Furthermore, Fawzy *et al.* (27) proposed that inhibin B could prevent the pituitary gland from producing FSH. The ovarian response diminishes and the FSH level rises while the inhibin B level is unable to maintain the FSH level within the normal range (26). As such, the baseline level of serum inhibin B may more accurately and immediately reveal the function of the ovarian reserve than the FSH level can do (26). The findings of the present study revealed that phenotype A had the highest level of inhibin B, while phenotypes B, C, and D had significantly lower levels of inhibin B, compared to the controls, This may be attributed to the fact that phenotype A possesses the triad characteristics of PCOS (hyperandrogenemia, oligomenorrhea-anovulation, polycystic ovarian morphology). To the best of our understanding, no previously published report or study dealt with inhibin B and the PCOS phenotypes. The current study also found that serum LH level and LH/FSH ratio were highest for phenotype A, which was in concurrence with the observation of Gürsu *et al.* (8). In the present study the significant decrease in FSH level in the B and C phenotypes, compared to that of the controls, concurred with the findings reported by Önal and Öztürk (2023) who recorded lower levels of serum FSH in PCOS phenotypes when compared to

controls (28). These variations in hormone levels across the phenotypes could cast more light on the pathophysiology of women with PCOS, thus helping broaden the understanding of gynecologists regarding the heterogeneity of this disease and the creation of tailored treatment plans for each phenotype (28). Sharmin *et al.* (2023) indicated that phenotype A was the most common phenotypic and severe form of PCOS. These authors concluded that, compared with patients having the other phenotypes, those with phenotype A had significant biochemical hyperandrogenism, abnormal LH levels, and an altered LH / FSH ratio. The mildest phenotype was the normo-androgenic one (phenotype D). Ovulatory patients (phenotype C) were less common, most likely due to the less severe signs and hormonal imbalances. The phenotypic division facilitates the prediction of unfavorable consequences as well as enhances knowledge of the pathogenesis and severity of PCOS. Besides, the correct recognition of the distinct phenotypes has diagnostic consequences and ensures that patients receive the right care (29). Jamil *et al.* (2016) found that genotypes A and B had significantly higher total testosterone levels and LH/FSH ratio (30). Besides, Yilmaz *et al.* (2011) showed that phenotypes A, B, and C have higher LH/FSH ratios than those with phenotype D (31). In contrast, Duz *et al.* (2020) observed that phenotype D had significantly higher levels of LH and LH/FSH ratios, than did the other PCOS phenotypes (32). The results of the present study revealed that phenotype D had the highest serum prolactin level (Table 3). However, Gürsu *et al.* (2022) and Önal and Öztürk (2023) found non-significant differences in serum prolactin among and between the PCOS phenotypes and controls (8, 26).

Limitation:

The inability to include women who had received a recent diagnosis of polycystic ovarian syndrome because of the limited number of instances observed during the research period. Furthermore, the sample size was rather small because the sampling period was short as well as the sub-classification of PCOS patients.

Conclusion:

Phenotype A is predominant among the PCOS phenotypes in Iraqi women. Serum inhibin B level and LH/FSH ratio could be used in differentiation of the different phenotypes of PCOS.

Authors' declaration:

We confirm that all the Figures and Tables in the manuscript belong to the current study. Besides, the Figures and images, which do not belong to the current study, have been given permission for re-publication attached to the manuscript. Authors sign on ethical consideration's approval-Ethical Clearance: The project was approved by the local

ethical committee in (Baghdad Teaching Hospital) according to the code number (111) on (6/5/2024).

Conflict of interest: None

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Authors' Contributions:

Study conception, study design, and critical revision: (Zainab Gihad Falh, Dr Basil O Saleh and Dr Afraa M AL Naddawi) Acquisition of data analysis, drafting of manuscript, and interpretation of data: (Zainab Gihad Falh, Dr Basil O Saleh and Dr Afraa M AL Naddawi)

Reference:

1. Gupta M, Yadav R, Mahey R, Agrawal A, Upadhyay A, Malhotra N, et al. Correlation of body mass index (BMI), anti-mullerian hormone (AMH), and insulin resistance among different polycystic ovary syndrome (PCOS) phenotypes-a cross-sectional study. *Gynecol Endocrinol.* 2019;35(11):970-3. <https://doi.org/10.1080/09513590.2019.1613640>.
2. Shallal MM, Mahmood N, Hussein ZA. Total L-carnitine and insulin resistance in non-obese and obese Iraqi women with polycystic ovary syndrome. *J Fac Med Baghdad.* 2023;65(1):20-6. <https://doi.org/10.32007/jfacmedbagdad.6512040>.
3. Hatem A, O Saleh B, M Al-Naddawi A. Association between serum fructose level and insulin resistance in women with polycystic ovary syndrome: The effect of obesity. *J Fac Med Baghdad.* 2022;64(2):91-5. <https://doi.org/10.32007/jfacmedbagdad.6421926>.
4. M. Alawad Z. Level of follicular fluid vitamin D and embryo quality in a sample of Iraqi women undergoing IVF. *J Fac Med.* 2019;60(4):215-21. <https://doi.org/10.32007/jfacmedbagdad.604758>.
5. Mehra T, Sharma S, Zahra T, Jangir S, Gupta B. Correlation of Body Mass Index with Anthropometric and Biochemical Parameters Among Polycystic Ovary Syndrome Phenotypes. *Indian J Clin Biochem.* 2023;38(2):231-41. <https://doi.org/10.1007/s12291-022-01042-y>.
6. Group REPCW. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome (PCOS). *Hum Reprod.* 2004;19(1):41-7. <https://doi.org/10.1093/humrep/deh098>.
7. Al-Naddawi AM, Rasheed MK, Ghalib MM. Association of Neuregulin-4 levels and body mass index with hyperandrogenism in Polycystic Ovary Syndrome patients. *J Fac Med Baghdad.* 2024;65(4). <https://doi.org/10.32007/jfacmedbagdad.2140>.
8. Gürsu T, Eraslan A, Angun B. Comparison of body mass index, anti-müllerian hormone and insulin resistance parameters among different phenotypes of polycystic ovary syndrome. *Gynecol Obstet Clin Med.* 2022;2(4):164-70. <https://doi.org/10.1016/j.gocm.2022.10.002>.
9. Ozay AC, Ozay OE, Gulekli B. Comparison of anti-müllerian hormone (aMh) and hormonal assays for

Phenotypic Classification of Polycystic ovary Syndrome. *Ginekol Pol.* 2020;91(11):661-7.

<https://doi.org/10.5603/GP.a2020.0122>.

10. Azziz R, Kintziger K, Li R, Laven J, Morin-Papunen L, Merkin SS, et al. Recommendations for epidemiologic and phenotypic research in polycystic ovary syndrome: an androgen excess and PCOS society resource. *Hum Reprod.* 2019;34(11):2254-65. <https://doi.org/10.1093/humrep/dez185>.

11. Hussein RA, Ali IN, Fahad NS. ESTIMATION OF SOME BIOCHEMICAL PARAMETERS IN IRAQI INFERTILE WOMEN WITH POLYCYSTIC OVARIAN SYNDROME. *Eur J Mod Med Pract.* 2023;3(9):142-8.

<https://inovatus.es/index.php/ejmmp/article/view/1977>.

12. Kalra B, Kumar A, Patel K, Patel A, Khosravi MJ. Development of a second-generation Inhibin B ELISA. *J Immunol Methods.* 2010;362(1-2):22-31.

<https://doi.org/10.1016/j.jim.2010.08.002>.

13. Hassan HH, Ghazi SM, Nasif AS. Study of a Hormonal Assay in PCOS Patients with Type 2 DM and their Correlation with Inhibin B. *Medico-Legal Updat.* 2020;20(3).

<https://doi.org/10.37506/mlu.v20i3.1461>.

14. Pratama G, Wiweko B, Asmarinah, Widyahening IS, Andraini T, Bayuaji H, et al. Mechanism of elevated LH/FSH ratio in lean PCOS revisited: a path analysis. *Sci Rep.* 2024;14(1):8229.

<https://doi.org/10.1038/s41598-024-58064-0>.

15. Jozkowiak M, Piotrowska-Kempisty H, Kobylarek D, Gorska N, Mozdziak P, Kempisty B, et al. Endocrine disrupting chemicals in polycystic ovary syndrome: the relevant role of the theca and granulosa cells in the pathogenesis of the ovarian dysfunction. *Cells.* 2022;12(1):174.

<https://doi.org/10.3390/cells12010174>.

16. Nisa KU, Tarfeen N, Mir SA, Waza AA, Ahmad MB GB. Molecular mechanisms in the etiology of polycystic ovary syndrome (PCOS): a multifaceted hypothesis towards the disease with potential therapeutics. *Indian J Clin Biochem.* 2024;39(1):18-36. <https://doi.org/10.1007/s12291-023-01130-7>.

17. Elsayed AM, Al-Kaabi LS, Al-Abdulla NM, Al-Kuwari MS, Al-Mulla AA, Al-Shamari RS, et al. Clinical phenotypes of PCOS: A cross-sectional study. *Reprod Sci.* 2023;30(11):3261-72.

<https://doi.org/10.1007/s43032-023-01262-4>.

18. Rabe-Hesketh S and Everitt BS. A handbook of statistical analyses using stata. Chapman & Hall/CRC, Taylor & Francis group. 2007 Fourth edition.

19. IBM SPSS Statistics 27 Core System User's Guide

20. Carmina E, Lobo RA. Comparing lean and obese PCOS in different PCOS phenotypes: Evidence that the body weight is more important than the Rotterdam phenotype in influencing the metabolic status. *Diagnostics.*

2022;12(10):2313. <https://doi.org/10.3390/diagnostics12102313>.

21. Malhotra N, Mahey R, Cheluvvaraju R, Rajasekaran K, Patkar D, Prabhakar P, et al. Serum

S

- anti-mullerian hormone (AMH) levels among different PCOS phenotypes and its correlation with clinical, endocrine, and metabolic markers of PCOS. *Reprod Sci.* 2023;30(8):2554-62. <https://doi.org/10.1007/s43032-023-01195-y>.
22. Si M, Xu W, Qi X, Jiang H, Zhao Y, Li R, et al. Metabolic syndrome rather than other phenotypes in PCOS as a predictive indicator for clinical outcomes in IVF: comprehensive phenotypic assessment across all PCOS classifications. *J Clin Med.* 2023;12(15):5073. <https://doi.org/10.3390/jcm12155073>.
23. Obaid RM, Ali SH, Hameed HM. Correlation Between Serum Inhibin and FSH Levels in Women with Different Reproductive Disorders. *Int J Res Appl Sci Biotechnol.* 2022;9(3):256-61. <https://www.ijrasb.com/index.php/ijrasb/article/view/414>.
24. Fazil GJ, Sadig HA, Tofiq MN, Ali IJ. The levels of inhibin A and inhibin B in PCOS patients. *GSC Biol Pharm Sci.* 2023;24(1):346-9. <https://doi.org/10.30574/gscbps.2023.24.1.0302>.
25. Farman MS, Akoul MA, Hamoode RH. Study of some hematological and hormonal changes in patients with (PCOS). *Ann Rom Soc Cell Biol.* 2021;2288-92. <http://annalsofrscb.ro>
26. Zhang F, Liu X ling, Rong N, Huang X wen. Clinical value of serum anti-mullerian hormone and inhibin B in prediction of ovarian response in patients with polycystic ovary syndrome. *J Huazhong Univ Sci Technol [Medical Sci.* 2017; 37:70-3. <https://doi.org/10.1007/s11596-017-1696-x>.
27. Fawzy M, Lambert A, Harrison RF, Knight PG, Groome N, Hennelly B, et al. Day 5 inhibin B levels in a treatment cycle are predictive of IVF outcome. *Hum Reprod.* 2002;17(6):1535-43. <https://doi.org/10.1093/humrep/17.6.1535>.
28. Murat Ö, ÖZTÜRK HÇ. Anti-Mullerian hormone and HOMA-IR in different phenotypes of polycystic ovary syndrome on insulin resistance. *Anatol Curr Med J.* 2023;5(4):376-82. <https://doi.org/10.38053/acmj.1323489>.
29. Sharmin F, Mirza TT, Latif T, Islam FA, Shamsi S, Kabir MA, et al. Hormonal Parameters in Diverse Phenotypes of Polycystic Ovarian Syndrome. *Mymensingh Med J MMJ.* 2023;32(1):3-9. <https://www.researchgate.net/publication/366837382>.
30. Jamil AS, Alalaf SK, Al-Tawil NG, Al-Shawaf T. Comparison of clinical and hormonal characteristics among four phenotypes of polycystic ovary syndrome based on the Rotterdam criteria. *Arch Gynecol Obstet.* 2016; 293:447-56. <https://doi.org/10.1007/s00404-015-3889-5>.
31. Yilmaz M, Isaoglu U, Delibas IB, Kadanali S. Anthropometric, clinical and laboratory comparison of four phenotypes of polycystic ovary syndrome based on Rotterdam criteria. *J Obstet Gynaecol Res.* 2011;37(8):1020-6. <https://doi.org/10.1111/j.1447-0756.2010.01478.x>.
32. Arda Duz S, Tuncay G, Karaer A. Clinical and hormonal characteristics of women with various phenotypes of polycystic ovary syndrome. 2020; ;27(6):1626-30 10.5455/annalsmedres.2020.02.125 <https://annalsmedres.org/index.php/aomr/article/view/826>. <https://doi.org/10.5455/annalsmedres.2020.02.125>.

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الظاهرية لمتلازمة المبيض المتعدد دور انهيبيين ب والهرمون الملوتن: نسبة الهرمون المنبه للجريب في تميز الأنماط الكيسات

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الخلاصة:

خلفية البحث: تعد متلازمة المبيض المتعدد الكيسات (PCOS) سببا رئيسيا لمشاكل الخصوبة وعدم انتظام الدورة الشهرية لدى النساء في سن الإنجاب. تنتج الخلايا الجريبية في الجريبات أمام الغدة والغريبة النامية مادة الإنهيبيين ب.

الأهداف: تم تصميم هذه الدراسة لمعرفة دور نسبة انهيبيين B في الدم والهرمون اللوتيني (LH) / الهرمون المنبه للجريب (FSH) في التمييز بين الأنماط الظاهرية المختلفة لمتلازمة المبيض المتعدد الكيسات.

طرق العمل: تم إجراء هذا البحث المقطعي في قسم الكيمياء الحيوية، كلية الطب، جامعة بغداد في الفترة من نوفمبر 2023 إلى مارس 2024. وشمل 111 امرأة، الفئة العمرية (18-40 سنة)، تم تشخيص 91 من هؤلاء النساء بتكيس المبيض المتعدد. متلازمة تكيس المبايض (PCOS) وفقا لمعايير إجماع روتتردام لعام 2003، وكانت 20 امرأة من النساء الأصحاء على ما يبدو. تم تقسيم النساء المصابات بمتلازمة تكيس المبايض إلى أربع مجموعات من النمط الظاهري (A، B، C، D). شملت التحقيقات قياسات مصل انهيبيين ب باستخدام تقنية مقايسة الامتصاص المناعي المرتبط بالإنزيم ((ELISA، FSH، LH، البرولاكتين باستخدام المقايسة المناعية الآلية Tosoh AIA 2000- و تم حساب نسبة LH/FSH.

النتائج: أظهرت النتائج أن النمط الظاهري A هو النمط السائد بين أنماط متلازمة تكيس المبايض، في حين أن النمط الظاهري B هو النمط النادر. كانت القيم المتوسطة (± SEM) لمستويات inhibin B للأنماط الظاهرية (A (p = 0.001)، B (p = 0.0001)، C (p = 0.0001)، D (p = 0.0001) أقل بكثير من تلك الخاصة بالنساء الضابطات. كانت القيمة المتوسطة لمستويات LH للنمط الظاهري A أعلى بكثير من تلك الخاصة بنساء السيطرة (P = 0.03). كانت القيم المتوسطة لنسبة LH / FSH مرتفعة بشكل ملحوظ في الأنماط الظاهرية (A (p = 0.001) و B (p = 0.04) بالمقارنة مع الضوابط.

الاستنتاج: النمط الظاهري A هو النمط السائد لمتلازمة تكيس المبايض لدى النساء العراقيات. يمكن استخدام مستوى Inhibin B في المصل ونسبة LH/FSH في التمييز بين الأنماط الظاهرية المختلفة لمتلازمة تكيس المبايض.

الكلمات المفتاحية: إنهيبيين B، نسبة LH/FSH، الأنماط الظاهرية، متلازمة تكيس المبايض، برولاكتين.