ORIGINAL RESEARCH Article

Inositol has a beneficial effect in the management of polycystic ovary syndrome: Special data for Libyan patients

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Received: October 20, 2025, **Accepted:** January 10, 2026, **Published online:** January 12, 2026

HOW TO CITE THIS

AL-belazi et al. Inositol has a beneficial effect in the management of polycystic ovary syndrome: Special data for Libyan patients. Mediterr J Med Res. 2026; 3(1): 14-26. [Article number: 34]. https://doi.org/10.5281/zenodo.18203319

Keywords: Food supplement, hormonal imbalance, menstrual irregularities, metformin, PCO

Abstract: Polycystic ovary syndrome (PCOS) is a complex endocrine and metabolic disorder characterized by anovulation, infertility, obesity, insulin resistance, and polycystic ovaries. This study aims to overview of PCOS pathophysiology and therapy, focusing particularly on the therapeutic role of inositol, followed by an evaluation of a specific clinical intervention in Libyan patients. The study assessed the effects of inositol in combination with metformin on hormonal and metabolic parameters in Libyan patients with PCOS. This study was conducted at the Tripoli Infertility Treatment Hospital between August and October 2025. The study included 80 women diagnosed with infertility associated with PCOS according to the Rotterdam criteria, with 42 participants having complete data. Participants were divided into two groups who received treatment for three months: 21 patients received myo-inositol plus d-chiro-inositol, and 21 patients received inositol combined with metformin. The findings showed a reduction in LH, LH/FSH ratio, AMH, HOMA-IR, and HbA1c, along with increased estradiol. FSH, prolactin, and TSH remained unchanged. No differences were observed between myo-inositol and combination therapy. This study suggests that inositol is an effective supplement therapy for Libyan patients with PCOS, improving ovarian function and metabolic health. The benefits are likely mediated by enhanced insulin sensitivity, which helps restore gonadotropin balance and alleviate symptoms.

Introduction

Worldwide, polycystic ovary syndrome (PCOS) is the most common metabolic disorder among women of reproductive age, which is a prevalent condition that impacts up to 20.0%, depending on the diagnostic criteria applied [1]. This condition is characterized by irregular menstrual cycles, hyperandrogenism, and the presence of polycystic ovarian morphology [2]. While several factors contribute to its development, insulin resistance (IR) and elevated androgens are recognized as the primary causes of PCOS. It is viewed as a heterogeneous condition exhibiting various phenotypes (**Table 1**). The different phenotypes are believed to be partially influenced by genetic diversity, which may account for the observed variations in the condition [3, 4]. The diagnosis typically relies on the Rotterdam criteria, necessitating the presence of at least two of the following criteria: Oligo/anovulation, clinical or biochemical signs of hyperandrogenism, and polycystic ovaries as identified through ultrasound scan [5]. Treatment is usually personalized to the complaints and needs of the patient and involves targeting metabolic abnormalities through lifestyle changes, medication, and potentially surgery for the prevention and management of excess body weight and androgen suppression.

Table 1: Phenotypes of polycystic ovary syndrome based on Rotterdam criteria

Phenotype	Characteristics	Common name/severity
A	Hyperandrogenism, oligo/anovulation, polycystic ovarian morphology	Classic PCOS-most severe
B	Hyperandrogenism, oligo/anovulation	Classic non-polycystic
C	Hyperandrogenism, polycystic ovarian morphology	Ovulatory PCOS/mild Form
D	Oligo/anovulation, polycystic ovarian morphology	Non-hyperandrogenic PCOS

Phenotypes A and B are considered complex, associated with higher risks of metabolic disturbances such as IR, dyslipidemia, metabolic syndrome, and require prolonged treatment. In contrast, phenotypes C and D are less complex and may have a more favorable prognosis [6]. This syndrome is characterized by an imbalance between the hypothalamic pituitary ovarian (HPO) axis and peripheral insulin signaling [7]. The exact etiology remains multifactorial, involving genetic, hormonal, and environmental components. There is dysregulation of the HPO axis, leading to increased luteinizing hormone (LH**)** relative to follicle-stimulating hormone (FSH). The elevated LH stimulates ovarian theca cells to produce excess androgens, while the low FSH impairs follicular maturation. Thus, ovulation fails to occur, and multiple immature follicles accumulate in the ovaries, producing the polycystic appearance [8, 9]. Some women with PCOS exhibit IR, which causes hyperinsulinemia. Excess insulin enhances androgen synthesis in the ovary and suppresses hepatic production of sex hormone-binding globulin (SHBG), increasing the free circulating androgens. These elevated androgens contribute to the clinical features of hirsutism, acne, and menstrual irregularities, while IR increases the risk of type 2 diabetes mellitus (T2DM), dyslipidemia, and cardiovascular diseases [8].

Managing PCOS involves pharmacological and non-pharmacological therapies. Weight loss is a key strategy that can enhance fertility and improve ovarian structure [10, 11]. For women suffering symptomatic PCOS, hormonal contraceptives are used to address menstrual irregularities, hirsutism, and acne. Combined hormonal contraceptives are effective in lowering LH, reducing ovarian androgen production, and increasing of sex hormone-binding globulin (SHBG). In cases where hirsutism persists, six months of treatment with combined oral contraceptive pills or cosmetic interventions, antiandrogens are required [12]. Hirsutism and infertility are often the primary reasons prompting patients with PCOS to seek medical attention. IR manifests in a tissue-specific manner rather than as a generalized defect across all tissues. Skeletal muscle and adipose tissue become IR, leading to reduced glucose uptake and increased lipolysis. Insulin sensitivity is preserved in the ovaries, adrenal glands, and liver. Elevated insulin plays a role in the pathophysiology of the syndrome by indirectly stimulating androgen production in the ovaries and adrenal glands. Insulin works synergistically with LH to stimulate theca cells, enhancing androgen synthesis. This androgen excess disrupts normal follicular development, leading to follicular arrest and ultimately anovulation [2]. Obesity, particularly central obesity, is associated with metabolic features in PCOS. Visceral adipose tissue is metabolically active and releases pro-inflammatory cytokines and adipokines. The mediators that contribute to a state of chronic low-grade inflammation and interfere with normal insulin signaling, thereby exacerbating IR, are a central feature of PCOS. Compared to non-obese individuals with PCOS, those with obesity exhibit more pronounced metabolic dysfunction and severe clinical symptoms. Obesity worsens reproductive outcomes by amplifying hormonal dysregulation and contributing to menstrual irregularities and anovulation. Adolescents who gain weight rapidly are at a higher risk of developing PCOS later in life, highlighting the importance of lifestyle intervention. A modest weight loss of 10.0% has been shown to improve insulin sensitivity and decrease androgen-related symptoms [8]. Metformin is beneficial in improving metabolic outcomes in PCOS, in enhancing insulin sensitivity, glucose metabolism, and lipid profiles. The significant improvements in ovulation and menstrual regularity are observed in women with IR or metabolic dysfunction [13]. Elevated androgen levels can lead to hirsutism; this symptom affects 90.0% of women with PCOS. Individuals may be prone to acne and develop acanthosis nigricans, characterized by dark patches of skin around the groin, anus, and armpits [14]. PCOS increases the risk for metabolic issues such as T2DM and dyslipidemia [15].

Excessive ovarian androgen secretion plays a role in the pathophysiology of PCOS. Early androgen overproduction contributes to the development of IR, while disturbances in lipid metabolism within visceral adipocytes. Androgens impair pancreatic β-cell function, leading to hyperinsulinemia. These mechanisms establish a clear link between androgen excess, IR, and the risk of T2DM in women with PCOS. Gonadotropin releasing was noted to have much more secretion of LH with a normal abundance of FSH [16]. The increase in LH in PCOS women may be due to a pulsatile increase in the secretions of gonadotropin-releasing hormone (GRH). A change in GRH might be due to defects in the hypothalamus in PCOS patients [17]. However, there are increased secretions of androgens [18]. Thus, it is essential to emphasize the importance of accurate diagnosis, as the observed dysregulation in the GRH-LH-FSH axis may not represent a primary defect but rather a secondary consequence of underlying hyperandrogenism [2]. Hyperandrogenism, characterized by elevated androgens, plays a role in the development of abdominal obesity in PCOS [19]. While a negative association between plasma androgen and obesity has been reported [20], it has been suggested hyperandrogenic state in PCOS contributes to weight gain [21]. Excessive androgens can induce various cellular activities and endoplasmic reticulum stress in granulosa cells and oocytes, thus promoting the development of PCOS. The presence of hyperandrogenism and visceral adiposity makes weight loss challenging for women with PCOS, even when adhering to lifestyle interventions. This resistance to weight reduction exacerbates metabolic and reproductive complications, reinforcing the need for a therapeutic plan [22]. Infertility in PCOS is a multifactorial disorder involving enzymatic responses, chronic inflammation, IR, and oxidative stress. Some genes, including the CYP family, the androgen receptor gene, and SHBG are frequently disrupted in PCOS. Mutations in CYP19A impair estrogen synthesis and lead to elevated androgen levels. The anti-Müllerian Hormone (AMH) gene warrants special attention. It encodes a glycoprotein that regulates follicular development and steroidogenesis. Reduced AMH signaling fails to suppress CYP17, thereby enhancing androgen biosynthesis and compromising fertility. Elevated serum AMH in PCOS is directly associated with higher testosterone and LH, disrupted oocyte maturation, and poor embryo quality. Increased AMH concentrations in follicular fluid correlate with a higher proportion of immature oocytes and lower fertilization rates compared to other infertile women [23]. Combined oral contraceptives are considered the treatment for managing hyperandrogenism in PCOS. The estrogen component increases hepatic production of SHBG, thereby reducing free androgen levels. Meanwhile, the progestin component lowers LH secretion, which in turn reduces ovarian androgen synthesis. Combined oral contraceptives are associated with an increased risk of thromboembolic events and elevated production of pro-inflammatory cytokines [24, 25]. In cases where hirsutism or androgen-related symptoms persist, combined oral contraceptive pill therapy or cosmetic interventions, antiandrogenic agents may be considered. Spironolactone, cyproterone acetate, flutamide, and finasteride are less commonly used due to potential hepatotoxicity and teratogenic risks. These are useful in addressing symptoms with psychological impact [26, 27]. Metformin is used to treat IR, a central feature of PCOS. It improves metabolic and clinical outcomes by reducing circulating insulin and androgen levels, increasing SHBG, and enhancing glucose metabolism and lipid profiles. Metformin modulates adipocytokines contributing to improved endocrine function. However, its side effects are reported [24].

Nutraceuticals have gained attention as adjunctive therapies in the management of PCOS, particularly for their roles in modulating IR, oxidative stress, and hormonal imbalance [26]. They often favored for their favorable safety profiles and potential to complement pharmacological treatments [27, 28, 29, 30]. Vitamin D deficiency is prevalent in PCOS and was linked to impaired glucose metabolism and reproductive dysfunction [31, 32, 33, 34, 35]. Omega-3 fatty acids exert anti-inflammatory effects and improve lipid profiles. Folic acid plays a role in methylation processes and homocysteine regulation [36]. Its supplementation may support oocyte maturation and improve metabolic parameters mainly when combined with inositol [37]. Selenium contributes to the reduction of oxidative stress and may lower androgen and improve insulin sensitivity [38]. Supplements such as N-acetylcysteine (NAC), coenzyme Q10, berberine, melatonin, and zinc have also shown promise in clinical studies. NAC enhances glutathione synthesis and improves ovulation rates; CoQ10 supports

mitochondrial function and oocyte quality; berberine activates AMPK and mimics metformin-like effects; melatonin regulates circadian rhythm and follicular health; and zinc contributes to androgen regulation and immune modulation. Further randomized controlled trials are warranted to establish standardized dosing protocols and long-term safety profiles [37]. Inositol, the isoforms myo-inositol (MYO) and d-chiro-inositol (DCI), has emerged as a promising nutraceutical due to its ability to act as a second messenger in insulin and FSH signaling pathways. This mechanism allows inositol to effectively improve insulin sensitivity, reduce serum androgen, enhance oocyte quality, and restore ovulatory function and menstrual regularity [39]. Inositol plays a critical role in cellular signaling, insulin sensitivity, and ovarian function [40]. Among its nine stereoisomers, MYO and DCI are biologically active forms which are essential in metabolic and reproductive pathways implicated in PCOS [40, 41]. DCI was identified as a second messenger of insulin and has been detected in insulin-responsive tissues. Elevated DCI is found in organs responsible for glycogen storage. In states of IR, the activity of the epimerase enzyme is impaired in non-reproductive tissues, thereby reducing the conversion of MYO to DCI. These result in diminished DCI, decreased glycogen synthesis, elevated blood glucose, and compensatory hyperinsulinemia, eventually exacerbating IR [42]. The ovary maintains normal insulin sensitivity even in IR individuals-a phenomenon referred to as the ovarian paradox. Persistent hyperinsulinemia overstimulates the epimerase activity in ovarian cells, leading to production of DCI at the expense of MYO. The physiological MYO: DCI ratio (100: 1) shifts dramatically to 0.2: 1 [43]. This imbalance promotes increased androgen synthesis due to elevated DCI and compromises FSH signaling and oocyte quality due to MYO depletion. The selection of the 40: 1; MYO: DCI ratio is based on evidence demonstrating that this proportion closely replicates the natural plasma ratio and effectively restores ovarian physiology in PCOS. Randomized trials have shown that this ratio is superior to MYO alone or to formulations with higher DCI content in normalizing hormonal parameters, reducing hyperandrogenism, and reinstating ovulation [44]. Although DCI exhibits beneficial insulin-sensitizing activity in peripheral tissues, its excessive accumulation within the ovary-due to hyperinsulinemia-driven over conversion, can impair hyperandrogenism and disrupt granulosa cell function. This highlights the importance of balanced supplementation rather than high-dose DCI formulations [45]. MYO supplement improves the proportion of mature oocytes, enhances fertilization rates, and supports better embryo quality [46]. MYO supplement, particularly when combined with DCI at a physiological ratio, constitutes an effective non-hormonal therapeutic strategy for PCOS. This approach restores menstrual cyclicity and ovulation, increases progesterone and SHBG, and reduces LH, testosterone, and insulin [44]. MYO supplement improves insulin sensitivity, reduces hyperandrogenism, and helps restore ovulatory function in PCOS, thereby enhancing fertility potential [47]. The MYO-DCI combination is effective in overweight patients [9]. MYO is known to decrease LH, androgens, the LH/FSH ratio, testosterone, androstenedione, and IR. MYO helps re-establish regular ovulatory cycles in obese women with PCOS, which supports spontaneous conception through adequate luteal phase progesterone production. MYO improves ovarian sensitivity to gonadotropins, reducing the required doses of FSH and LH .Although various formulations may include additional ingredients such as folic acid, NAC, α-lipoic acid, and coenzyme Q10, inositol remains the central therapeutic agent responsible for the clinical benefits observe . Supporting compounds like α-lipoic acid and coenzyme Q10 may enhance treatment outcomes by reducing oxidative stress and improving mitochondrial function, so reinforcing inositol's effects on insulin response and ovarian function. Metformin has been used for managing IR in PCOS; its mechanism involves the enhancement of peripheral glucose uptake and reduction of hepatic gluconeogenesis [48, 49]. Inositol has the added advantage of improving ovarian function and oocyte quality. While metformin remains a valuable option, inositol presents a safer and patient-friendly alternative or adjunct in the metabolic and reproductive management of PCOS [50]. It does not interfere with hepatic, renal, or endocrine homeostasis, and its safety represents a major therapeutic advantage. This profile reinforces the role of inositol as a viable long-term strategy for managing PCOS [51, 52]. This study aims to evaluate the therapeutic effects of MYO in Libyan women with PCOS, and to compare these effects with the combination therapy of MYO and metformin.

Materials and methods

Study design: This observational study was conducted at the Tripoli Infertility Treatment Hospital, Tripoli, Libya, during 2025. The study included 80 women diagnosed with infertility associated with PCOS. 42 women had complete data sets and were included in the analysis. 21 patients received MYO plus DCI (combination with multivitamins), while the other 21 patients received inositol combined with metformin for three months.

Data collection: Data were collected using a structured data collection form designed and approved by the Department of Pharmacology and Clinical Pharmacy, University of Tripoli, Libya. The form consists of three sections**,** comprising of 15 items**,** including multiple-choice and open-ended questions. The first section includes demographic data and general data; Age, marital status, body mass index, duration of PCOS diagnosis in years, presence and type of primary or secondary infertility, regularity of the menstrual cycle, and hirsutism.

Treatment design: Type of medication used was MYO plus DCI with multivitamins or MYO combined with metformin, and laboratory data before and after treatment, including FSH, LH, LH/FSH ratio, AMH, prolactin, estradiol, TSH, HbA1c, and HOMA-IR were collected.

Post-treatment observations: The following are considered: whether the menstrual cycle became regular after treatment, body weight reduction since starting therapy, improvement in hirsutism, and whether pregnancy occurred following treatment.

Ethical approval: All the patients provided verbal and written consent on their participation in the trial and their right to withdraw at any time, in accordance with the ethical guidelines outlined in the 1975 Declaration of Helsinki. In addition, After the study was approved by the Hospital Ethics Committee (C-210-2025), data were collected.

Statistical analysis: Data were collected retrospectively from patients' medical records. Records with complete and verified data were included in the analysis. Numerical data presented as median±standard deviation. A multi-stage statistical analysis was applied to compare the laboratory measurements. Comparison: pre-treatment vs. post-treatment: If the data was normally distribution (pre and post), a paired sample t-test was used. If the data were not normally distribution, the Wilcoxon rank test was used. Comparison between treatment groups (inositol vs. inositol plus metformin): If post-treatment data was normal, an independent sample t-test was used. If post-treatment data was not normal, the Mann-Whitney test was used.

Results

Table 2 shows included in this study, a group of 42 female participants with an equal participation of 50.0% used inositol, and an equal group of combination treatment of inositol plus metformin with 50.0%. The mean age of 28.0 years, with the median age of 27.0 years, suggesting the normal distribution of the participants (range 20-37 years). The standard deviation of 4.1 shows that the age distribution is clustered around the mean. The largest frequency was found in the group of 25-29 years with 47.6%. However, the lowest frequency was found in those 35 years and above with 9.5%.

Table 2: Age-related distribution of the Libyan women with PCOS

Age in years	Frequency	Percent
25-29	20	47.6
30-34	10	23.8
20-24	08	19.0
> 35	04	09.5
Total	**42**	**100.0**
Median ±SD	**27.0 ±4.1**	

In **Figure 1**, the median body mass index (BMI) of 37.6 (kg/m²) indicates that the patients are classified as class II obesity. The median of 37.6 is higher than the mean, suggesting the concentration of the data in the obesity range, with 24.8 minimum and a maximum of 46.0, with a range of 21.2. The standard deviation of 5.94 shows moderate variation. The highest rate was obesity class II with 33.3%. The lowest frequency was no one underweight.

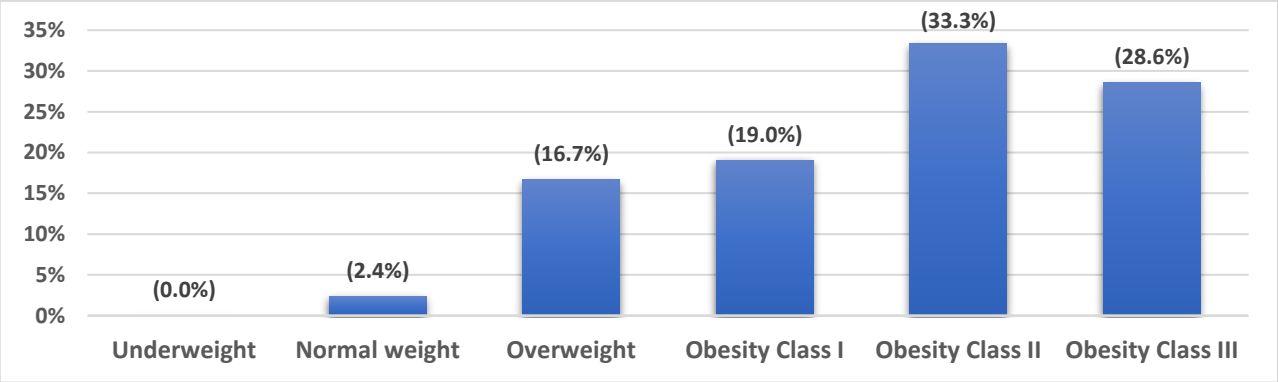

Figure 1: Distribution of body mass index (kg/m²) of the Libyan patients

Regarding menstrual cycle regularity, women were distributing as the highest frequency was irregular menstrual cycle with 76.2%, and 23.8% with regular cycle. The distribution of diagnosis duration is shown in **Figure 2**, with almost half of the patients are of two years duration and 5.0% with almost five years.

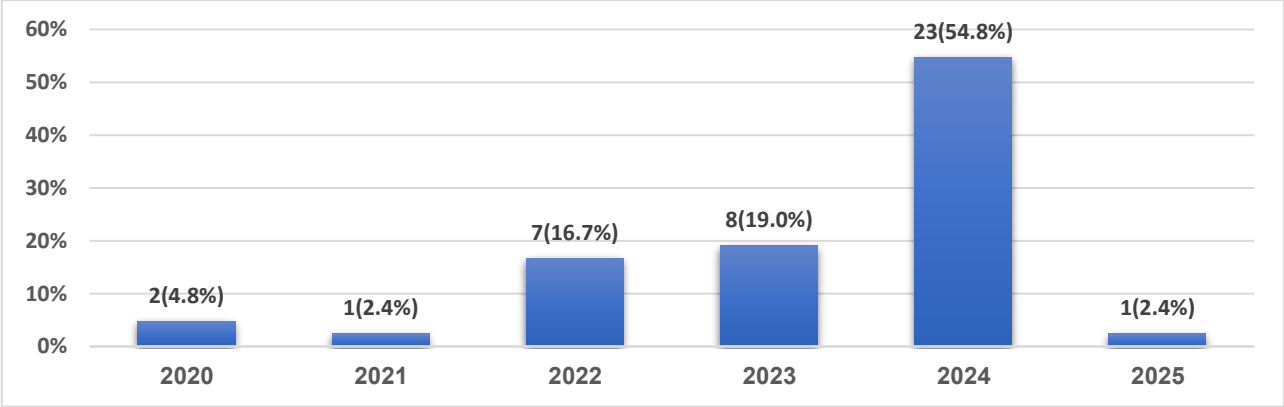

Figure 2: Duration of the diagnosis in patients with polycystic ovary syndrome

Regarding infertility, the highest frequency was primary, with 59.5%. The lowest frequency was secondary with 40.5%. The presence of hirsutism was present in 59.5% of the patients. However, the lowest was no hirsutism which was represented by 40.5%. In **Figure 3**, there is no significant difference in FSH levels between pre- and post-treatment by the Mann-Whitney *U*-test (p=0.256).

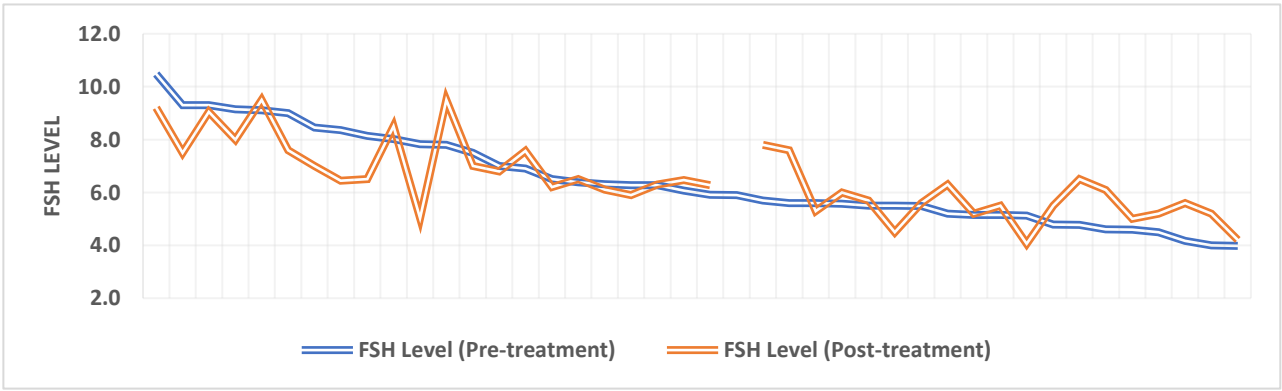

Figure 3: Follicle-stimulating hormone levels of the pre- and post-treatments

Regarding the LH level analysis, **Figure 4** shows that a highly significant difference exists in the LH levels between pre- and post-treatment ($p<0.001$), with no difference in comparison of LH between treatment groups.

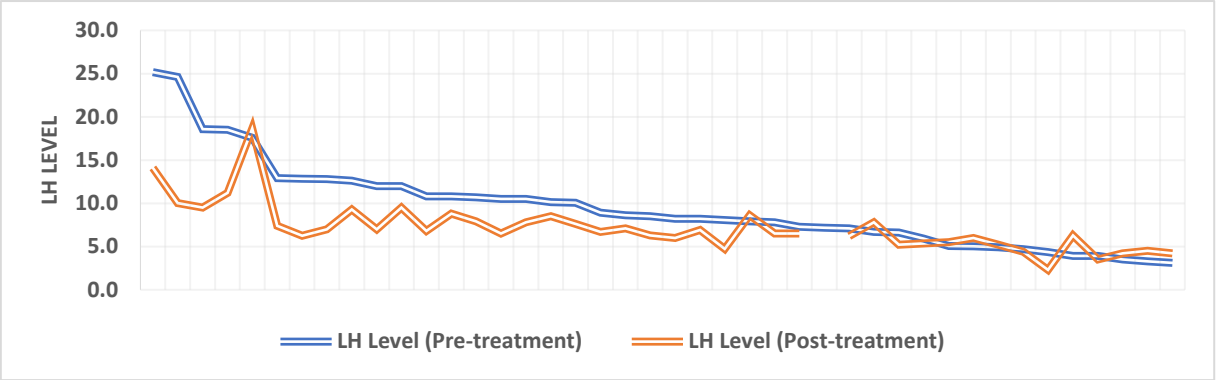

Figure 4: Luteinizing hormone levels of the pre- and post-treatments

With regard to the LH/FSH, **Figure 5** shows no significant difference in LH/FSH between pre- and post-treatment was found. However, at H1 highly significant difference exists in the LH/FSH ratio between pre- and post-treatment ($p<0.001$).

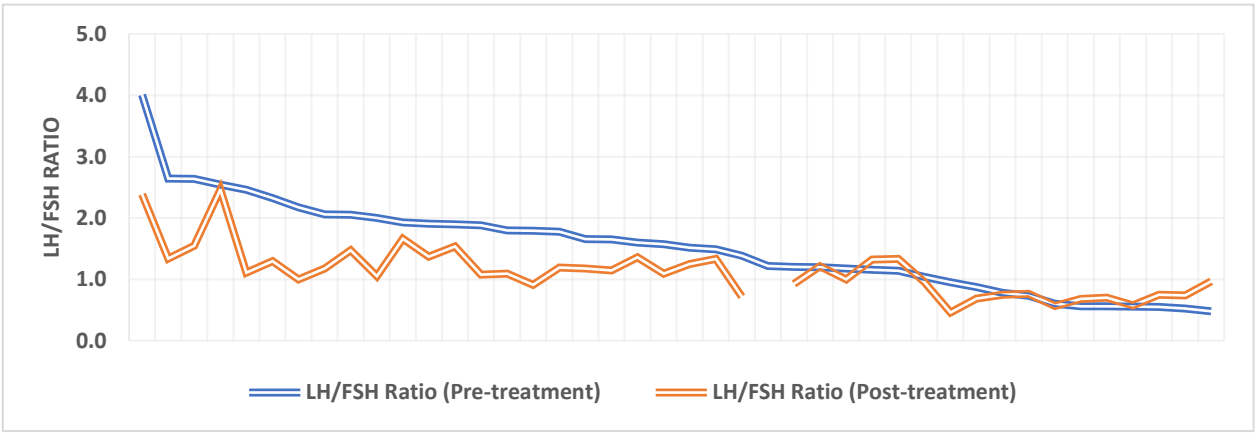

Figure 5: Luteinizing/Follicle-stimulating Hormones ratio of the pre- and post-treatments

The same findings were found with the prolactin levels and AMH level between pre- and post-treatment (**Figure 6**). Regarding hemoglobin A1c (HbA1c) analysis at H0, there is no difference in HbA1c levels between pre- and post-treatment. However, at H1, a highly significant difference exists in HbA1c levels between pre- and post-treatment (**Figure 7**). With regard to the TSH analysis, at H0, there is no difference in TSH levels between pre- and post-treatment. At H1, a difference exists in TSH levels between pre- and post-treatment (**Figure 8)**. However, estradiol level analysis at H0, no difference in estradiol levels between pre- and post-treatment. At H1, a difference exists in estradiol levels between pre- and post-treatment ($p<0.001$). Thus, no difference between medication groups in post-treatment estradiol levels.

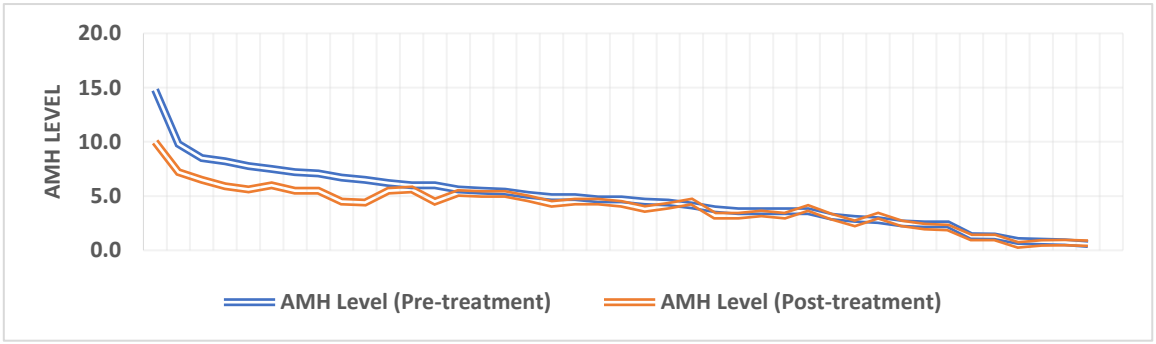

Figure 6: Anti-Müllerian hormone levels of the pre- and post-treatments

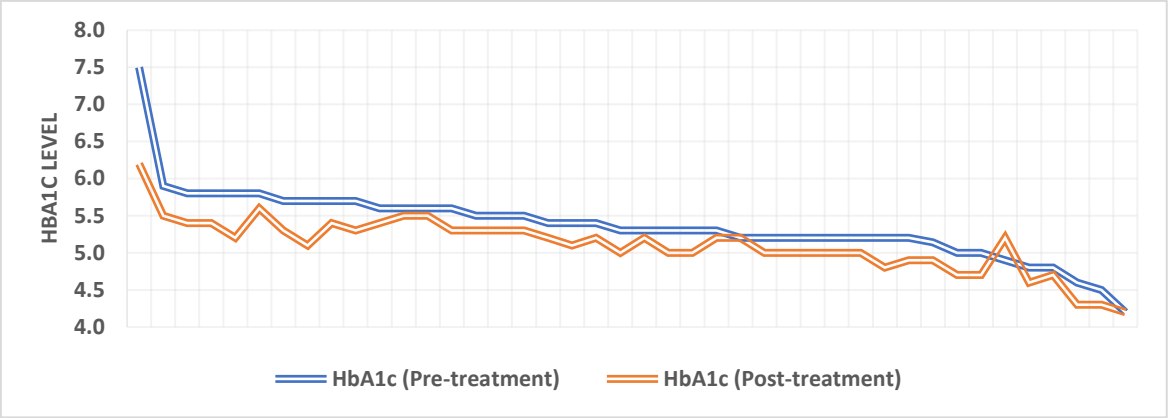

Figure 7: Hemoglobin A1c levels of the pre- and post-treatments

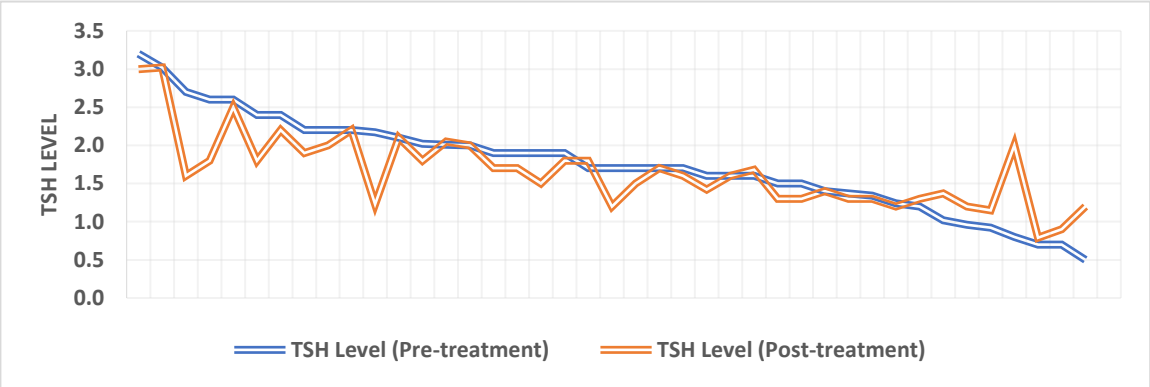

Figure 8: TSH levels of the pre- and post-treatments

Figure 9 shows that homeostatic model assessment of insulin resistance (HOMA-IR) at H0, there is no significant difference in HOMA-IR levels between pre- and post-treatment. However, at H1, a significant difference exists in HOMA-IR levels between pre- and post-treatment ($p<0.001$).

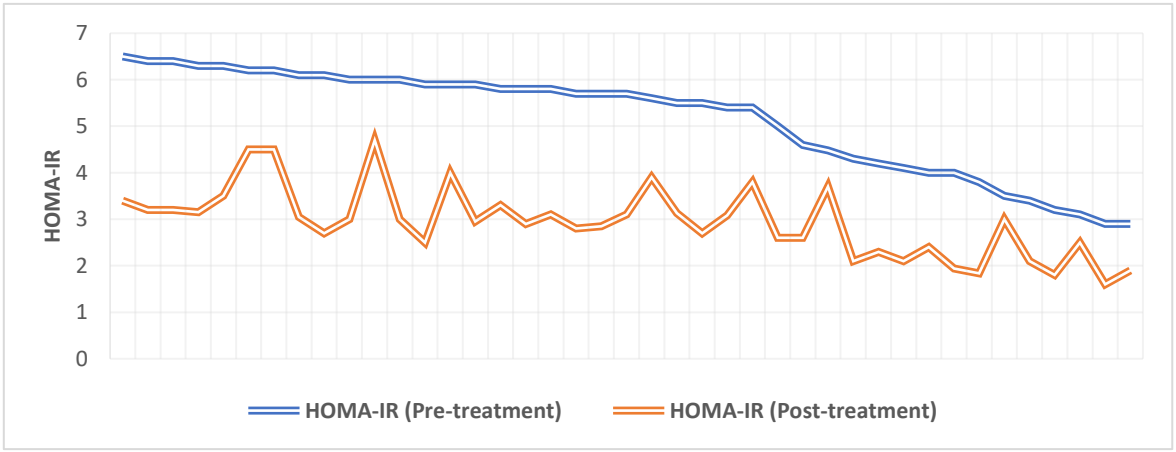

Figure 9: Homeostatic model assessment of insulin resistance level of the pre- and post-treatments

Discussion

In this study of 42 women diagnosed with PCOS according to the Rotterdam criteria, the mean age was 28.0 years with near half of participants between 25-29 years. Most of the participants were diagnosed in 2024, reflecting an actively managed patient population. A high prevalence of metabolic and reproductive disturbances was observed: Overweight and obesity were common, with the majority classified as obesity class II or class III. Menstrual irregularity and hirsutism were highly observed, highlighting the prominence

of hyperandrogenic features. Primary infertility accounted for more than half of the patients. These clinical characteristics provide important context for interpreting treatment outcomes. The combination of high BMI, IR, and hyperandrogenism underpins reproductive and metabolic dysfunction in PCOS. Irregular cycles reflect LH hypersecretion and impaired gonadotropin balance, while excess androgen activity manifests as hirsutism and contributes to follicular arrest. IR, common in overweight and obese participants, further exacerbates these disturbances by promoting hyperinsulinemia, which stimulates ovarian androgen production and disrupts normal ovarian function. The present study also evaluated the therapeutic effects of MYO alone or in combination with metformin, on hormonal and metabolic parameters in women with PCOS. The findings demonstrated improvements in several key parameters associated with PCOS pathophysiology, particularly LH, LH/FSH ratio, AMH, estradiol, HbA1c, and HOMA-IR. However, no differences were observed between the two medication groups, suggesting that MYO monotherapy may provide comparable benefits to the combined regimen for the studied outcomes. FSH did not show post-treatment changes, which aligns with multiple studies reporting that inositol has minimal direct impact on FSH [53]. In contrast, LH decreased after treatment. This decline is consistent with the well-established effect of m-inositol on improving ovarian sensitivity to insulin, reducing ovarian androgen production, and subsequently lowering LH hypersecretion. Previously, a reduction in LH following MYO supplementation was reported [54]. Treatment with MYO alone or in combination with metformin resulted in a decrease in the LH/FSH ratio indicating an improvement in the HPO axis regulation. However, with no difference between the two treatment groups, suggesting that MYO monotherapy may be sufficient to achieve hormonal modulation. It is reported that MYO supplementation can lead to modest improvements in gonadotropin balance, although the magnitude of change may vary depending on population characteristics, treatment duration, and baseline hormonal status [55, 56]. The reduction in the LH/FSH ratio in the current study could be influenced indirectly by improvements in metabolic parameters, as the data showed decreases in HOMA-IR and HbA1c. This supports the mechanism whereby MYO improves insulin sensitivity, which in turn may normalize LH secretion and ovarian responsiveness. The findings highlight that while hormonal changes may be moderate, MYO exerts a clinically meaningful effect on endocrine and metabolic aspects of PCOS. The current finding revealed no differences pre- and post-treatment, nor between treatment groups, aligning with the understanding that MYO's effects are pronounced on insulin-sensitive and gonadotropin-regulated pathways rather than on pituitary hormones. Estradiol levels increased in post-treatment in the MYO and MYO plus metformin groups with no difference between the two groups. This may suggest that MYO alone is sufficient to enhance ovarian estrogen production. Improved insulin signaling enhances FSH receptor sensitivity at the ovarian follicle, promoting follicular maturation and increasing estradiol secretion. These are consistent with recent data showing that myo-inositol supports ovarian responsiveness and estradiol synthesis, even without combination therapy [56, 57].

The current findings showed a decrease in AMH levels post-treatment in both groups, indicating improved follicular maturation and a reduction in the pool of small, arrested follicles. MYO likely mediates this effect by enhancing intra-ovarian insulin signaling and FSH responsiveness, allowing follicles to progress through normal maturation rather than remaining arrested. This mechanism indirectly lowers AMH production from granulosa cells of small antral follicles. Indeed, no difference was observed between monotherapy and combination therapy, suggesting that MYO alone can exert this effect. These results align with modern evidence supporting its role in regulating ovarian function and follicular dynamics in PCOS [45]. Similarly, supplementation with MYO combined with folic acid has been shown to reduce AMH levels in women with PCOS. This reduction in AMH may indicate improved ovarian function and could contribute to enhanced fertility outcomes. These underscore the therapeutic potential of MYO in managing reproductive dysfunction associated with PCOS [58]. HOMA-IR values decreased in the MYO monotherapy and MYO plus metformin groups with no difference between the two groups. This indicates that MYO alone effectively improves insulin sensitivity in PCOS. MYO acts as a second messenger for insulin, enhancing intracellular signaling pathways in muscle and adipose tissue, improving glucose uptake, and reducing circulating insulin. Lower insulin levels

subsequently decrease ovarian theca cell androgen production, indirectly contributing to normalization of LH secretion and LH/FSH ratio. These findings align with multiple studies which report that MYO consistently improves HOMA-IR in PCOS patients [55, 56]. Data showed a reduction in HbA1c post-treatment in both groups, reflecting improved long-term glycemic control. The underlying mechanism is closely related to MYO insulin-sensitizing action by improving post-receptor insulin signaling, glucose uptake increases and systemic glycemia stabilizes, leading to reduced HbA1c levels. These metabolic effects are clinically relevant, as better glycemic control reduces hyperinsulinemia-driven ovarian dysfunction and supports reproductive outcomes in PCOS. This result is consistent with publications from studies demonstrating HbA1c improvement with MYO supplementation [9, 54]. It should be mentioned that healthcare professionals such as pharmacists and physicians can thus provide comprehensive care to patients with PCOS and the intake of food supplements in certain clinical diseases [59-62].

Conclusion: Inositol, whether administered alone or in combination with metformin, produces improvements in hormonal and metabolic parameters in PCOS. The findings include a reduction in LH levels, LH/FSH ratio, AMH, HOMA-IR, and HbA1c, along with an increase in estradiol**,** while FSH, prolactin, and TSH remained unchanged. No differences were observed between myo-inositol monotherapy and combination therapy, suggesting that inositol alone may be sufficient for clinical benefit in similar populations. Inositol functions effectively as a supplement therapy, supporting ovarian function, regulating the menstrual cycle, improving ovulation, and alleviating PCOS-related symptoms.

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Acknowledgements: The authors would like to thank all the participants for their contributed to this study.
Authors' contribution: BJA & LMA conceived, designed the study. LMA collected data. BJA & LMA contributed to data analysis and performed analysis. FMS & BJA drafted and reviewed the manuscript important intellectual context. All authors approved the final version of the manuscript and agreed to be accountable for its contents.
Conflict of interest: The authors declare the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.
Ethical issues: The authors completely observed ethical issues, including plagiarism, informed consent, data fabrication or falsification, and double publication or submission.
Data availability statement: The raw data that support the findings of this article are available from the corresponding author upon reasonable request.
Author declarations: The authors confirm that they have followed all relevant ethical guidelines and obtained any necessary IRB and/or ethics committee approvals.
Generative AI disclosure: No Generative AI was used in the preparation of this manuscript.

للإينوزيتول تأثير مفيد في علاج متلازمة تكيس المبايض: بيانات خاصة بالمريضات الليبيات

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الملخص: متلازمة تكيس المبايض (PCOS) اضطراب معقد في الغدد الصماء والتمثيل الغذائي، يتميز بانقطاع الإباضة، والعقم، والسمنة، ومقاومة الأنسولين، وتكيس المبايض. تهدف هذه الدراسة إلى تقديم نظرة عامة على الفيزيولوجيا المرضية لمتلازمة تكيس المبايض وعلاجها، مع التركيز بشكل خاص على الدور العلاجي للإينوزيتول، يليه تقييم لتدخل سريري محدد لدى مريضات ليبيات. قيّمت الدراسة تأثيرات الإينوزيتول مع الميتفورمين على المؤشرات الهرمونية والتمثيل الغذائي لدى مريضات ليبيات مصابات بمتلازمة تكيس المبايض. أُجريت هذه الدراسة في مستشفى طرابلس لعلاج العقم بين أغسطس وأكتوبر 2025. شملت الدراسة 80 امرأة تم تشخيص إصابتهن بالعقم المرتبط بمتلازمة تكيس المبايض وفقًا لمعايير روتردام، مع توفر بيانات كاملة لدى 42 مشاركة. قُسّمت المشاركات إلى مجموعتين تلقتا العلاج لمدة ثلاثة أشهر: 21 مريضة تلقين ميو-إينوزيتول بالإضافة إلى د-كيرو-إينوزيتول، و21 مريضة تلقين الإينوزيتول مع الميتفورمين. أظهرت النتائج انخفاضًا في مستوى الهرمون اللوتيني (LH)، ونسبة LH/FSH، وهرمون AMH، ومؤشر مقاومة الأنسولين (HOMA-IR)، ومستوى الهيموجلوبين السكري (HbA1c)، بالإضافة إلى ارتفاع مستوى الإستراديول. بينما لم يطرأ أي تغيير على مستويات هرمون FSH، والبرولاكتين، وهرمون TSH. ولم تُلاحظ أي فروق بين العلاج بالميو-إينوزيتول والعلاج المركب. تشير هذه الدراسة إلى أن الإينوزيتول يُعدّ علاجًا تكميليًا فعالًا للمريضات الليبيات المصابات بمتلازمة تكيس المبايض، إذ يُحسّن وظائف المبيض والصحة الأيضية. ومن المرجح أن هذه الفوائد تعود إلى تحسين حساسية الأنسولين، مما يُساعد على استعادة توازن الهرمونات التناسلية وتخفيف الأعراض.