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Original Research Article

Effect of myo-inositol on ovulation induction outcomes in women with polycystic ovary syndrome

Natasha T. Aleem[1]*, Mahbuba Akhter[2], Sadia Islam[1]

[1]Department of Obstetrics and Gynecology, Asgar Ali Medical Hospital, Dhaka, Bangladesh
[2]Department of Obstetrics and Gynecology, City Medical College, Gazipur, Bangladesh

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***Correspondence:**
Dr. Natasha T. Aleem,
E-mail: natasha_aleem@yahoo.com

ABSTRACT

Background: Polycystic ovarian syndrome (PCOS) is a common endocrine condition affecting reproductive-aged women that is distinguished by insulin resistance, hyperandrogenism, and anovulation. Myo-inositol, an insulin-sensitizing drug, has emerged as a promising complementary therapy for enhancing reproductive outcomes. The study aimed to assess whether myo-inositol supplementation affected ovulation induction outcomes in women with PCOS undergoing reproductive treatments.

Methods: This investigation was carried out at Department of Obstetrics and Gynecology, Asgar Ali Medical Hospital, Dhaka, Bangladesh from January 2023 to December 2025. A prospective comparison study included 100 women diagnosed with PCOS based on the Rotterdam criteria. Participants were divided into two groups: the control group (n=50) received standard ovulation induction, and the myo-inositol group (n=50) received 2 grams of myo-inositol twice daily. The primary outcomes were ovulation confirmation and endometrial thickness. The data were entered and analyzed with statistical package for the social sciences (SPSS) version 26.

Results: Baseline characteristics were comparable among groups. The myo-inositol group had significantly greater ovulation rates (74.0% versus 56.0%, p=0.04) and endometrial thickness (8.1±1.4 mm versus 7.4±1.3 mm, p=0.01) than the control group. Mid-luteal progesterone levels were higher in the myo-inositol group (10.6±4.1 ng/ml versus 8.9±3.7 ng/ml, p=0.03). Multivariable analysis revealed that myo-inositol supplementation is an independent predictor of ovulation (adjusted OR 2.30, 95% CI 1.01-5.24, p=0.047).

Conclusion: Myo-inositol supplementation dramatically improves ovulation rates and endometrial growth in women with PCOS who are undergoing ovulation induction, making it an excellent adjuvant therapy for improving reproductive outcomes in this population.

Keywords: Polycystic ovary syndrome, Myo-inositol, Ovulation induction, Infertility

INTRODUCTION

The female reproductive system, particularly a pair of ovaries, controls follicular growth and ovulation with the help of hormones and intraovarian signals. Interference with these mechanisms can result in anovulation and infertility. Polycystic ovary syndrome (PCOS) is the most prevalent endocrine disorder characterized by ovarian dysfunction, generally presenting with chronic anovulation and subfertility.[1,2] Approximately 6-13% of women of reproductive age are affected globally by PCOS, making it the most common cause of anovulatory infertility.[3] In Asia, estimates of the prevalence are between 9 and 18%, with South Asian women from the Indian subcontinent showing severe metabolic and reproductive manifestations.[4,5] Recent estimates from Bangladesh, though limited, suggest an increasing rate of diagnosis of PCOS among reproductive-aged women, with a reported prevalence of approximately 10-12% in some regional studies, particularly in urban and semi-urban areas.[6] The escalating burden of PCOS has important implications for infertility management in Bangladesh,

considering that ovulatory dysfunction remains one of the major causes of clinic visits for subfertility in this region.[7] Beyond reproductive dysfunction, PCOS is strongly associated with insulin resistance, hyperinsulinemia, and compensatory hyperandrogenism, which collectively impair follicular development and ovulation.[8] Standard ovulation induction agents such as clomiphene citrate and letrozole are effective in many patients, but resistance to these agents, the risk of multiple pregnancy, and failure to address underlying metabolic abnormalities limit their long-term effectiveness.[9] Over the last two decades, therefore, therapeutic strategies aimed at targeting insulin sensitivity have gained increasing interest.[10] Myo-inositol, a natural insulin-sensitizing agent and an important mediator of intracellular insulin signaling, has emerged as a potentially promising adjunctive therapy or alternative therapy in PCOS management. Myo-inositol acts as a second messenger in FSH and insulin pathways, leading to enhanced ovarian responsiveness, decreased hyperandrogenism, and improved ovulation.[11] Several RCTs and meta-analyses have been conducted, which have shown that supplementation with myo-inositol significantly improves menstrual cyclicity, ovulatory rate, and metabolic parameters in women with PCOS, with good safety.[12-14] Compared to metformin, myo-inositol causes fewer gastrointestinal side effects and has better compliance among patients.[15] Despite increasing evidence of its efficacy, the results of these studies are still heterogeneous because of differing study designs, patient characteristics, treatment duration, and outcome measures.[14,16] Furthermore, most reported data come from Western or East Asian populations, while there is scant evidence regarding South Asian and Bangladeshi women who often reveal more pronounced insulin resistance and different phenotypic expressions of PCOS.[5,6] This highlights the need for region-specific clinical studies to assess the effectiveness of myo-inositol in ovulation induction within this population. In contrast to recent related studies, this study will focus on ovulation induction outcomes in a well-defined PCOS cohort, include clinically relevant ovulatory and hormonal endpoints, and contribute to an important evidence gap in the Bangladeshi context. Therefore, this study aims to evaluate the effect of myo-inositol on ovulation induction outcomes in women with polycystic ovary syndrome.

METHODS

This comparative study was prospectively carried out at Department of Obstetrics and Gynecology, Asgar Ali Medical Hospital, Dhaka, Bangladesh from January 2023 to December 2025. One hundred participants who were diagnosed with PCOS according to the Rotterdam criteria, with at least two out of three features comprising oligo/anovulation, clinical or biochemical hyperandrogenism, and polycystic ovarian morphology on ultrasound, were enrolled. Participants were divided into two groups of 50 participants each, including a control group (n=50) treated with a standard protocol of ovulation induction and a myo-inositol group (n=50) administered 2 grams of myo-inositol twice a day for a period of 12 weeks, combined with ovulation induction. Inclusion criteria included active women diagnosed with PCOS, primary or secondary infertility, patent fallopian tubes demonstrated by hysterosalpingography or laparoscopy, normal semen analysis of the partner, and written consent. Exclusion criteria included causes of infertility (tubal factor, severe male factor, endometriosis), prior ovarian surgery, uncontrolled thyroid disorders, hyperprolactinemia, Cushing syndrome, congenital adrenal hyperplasia, current pregnancy or breastfeeding, contraindications to ovulation induction agents, and refusal of informed consent. Evaluation at baseline was done with a detailed history, anthropometry (height, weight, BMI), clinical examination for hirsutism (modified Ferriman Gallwey score), presence or absence of acanthosis nigricans, and acne. Laboratory studies: hormonal profile (LH, FSH, TT, TSH, prolactin); blood sugar, insulin, HOMA-IR, and transvaginal ultrasound for ovarian volume, AFC, and endometrial thickness. Ovulation induction was carried out with standard ovulation induction protocols with serial follicular monitoring. Criteria for ovulation: a dominant follicle size >18 mm with a progesterone level >5 ng/ml in a mid-luteal phase serum sample. The primary outcomes were ovulation confirmation and endometrial thickness on trigger day. The secondary outcomes were biochemical, clinical, and ongoing pregnancies at 12 weeks. The data was analyzed with statistical package for the social sciences (SPSS) software version 26. The results were analyzed with a Chi-squared test for categorical data, an independent t-test for continuous data, and logistic regression for predictors of ovulation, with $p<0.05$ taken as significant.

RESULTS

Table 1 reveals a comparable mean age (26.4±4.2 years in the control group versus 25.9±4.5 years in the myo-inositol group), with the majority of participants in the 25-30 age group. The majority of participants (50% control, 48% myo-inositol) were overweight, with a mean BMI of 26.5 kg/m². The average duration of infertility in both groups was 3.8 years in the control group versus 3.6 years in the myo-inositol group.

The clinical phenotypic characteristics are provided in Table 2. Oligo/amenorrhea was the most common menstrual irregularity. Clinical hirsutism (modified Ferriman-Gallwey score ≥8) was found in (48% versus 44%) of women in both groups, while acne (40% versus 36%) and acanthosis nigricans (34% versus 30%) had similar prevalence. 28% of the control group had a family history of diabetes, while 26% from the myo-inositol group had the same. Prior unsuccessful ovulation induction attempts occurred in (42% versus 40%) of patients, indicating complex ovulatory dysfunction.

Table 3 shows hormonal and metabolic factors. Mean LH levels (10.2 versus 9.8 IU/l) and FSH levels (5.7 versus 5.6

IU/l) were comparable, resulting in LH: FSH ratios of around 1.8, which is typical with PCOS. Total testosterone levels averaged 57 ng/dl across all groups. Fasting glucose (5.2 versus 5.1 mmol/l) and insulin levels (14.1 versus 13.7 µIU/ml) were within normal PCOS values. The HOMA-IR levels (3.3 versus 3.1) showed moderate insulin resistance. Thyroid function (TSH 2.3 versus 2.2 mIU/l) and prolactin levels (15.4 versus 14.8 ng/ml) ruled out other endocrinopathies. Table 4 reveals that the mean ovarian volumes (11.6 versus 11.2 ml) exceeded the diagnostic criterion of 10 ml, as per PCOS criteria. Antral follicle counts were 22.1 versus 21.6, indicating polycystic morphology. The baseline endometrial thickness was 44.8 versus 4.7 mm during the early follicular phase, indicating proliferative endometrium.

Table 5 reveals that 82% of the myo-inositol group achieved dominant follicles (≥18 mm) compared to 68% of the controls (p=0.10). Confirmed ovulation was considerably higher with myo-inositol (74% versus 56%, p=0.04), indicating an enhanced ovarian response. Endometrial thickness on trigger day improved significantly (8.1±1.4 mm versus 7.4±1.3 mm, p=0.01), which is critical for embryo implantation. Myo-inositol treatment resulted in considerably higher mid-luteal progesterone levels (10.6±4.1 ng/ml versus 8.9±3.7 ng/ml, p=0.03), indicating improved corpus luteum function. Monofollicular response rates (60% versus 46%) favored myo-inositol, but multifollicular responses were comparable, implying increased follicular quality without significant stimulation risk.

Table 1: Baseline socio-demographic and anthropometric characteristics (n=100).

Variable	Category	Control (n=50), N (%)/ mean±SD	Myo-inositol (n=50), N (%)/mean±SD
Age (years)	≤17	0 (0)	2 (4)
	18-24	17 (34)	14 (38)
	25-30	20 (40)	23 (46)
	>30	13 (26)	11 (22)
	Mean±SD	26.4±4.2	25.9±4.5
BMI (kg/m²)	Normal (18.5-24.9)	16 (32.0)	18 (36.0)
	Overweight (25.0-29.9)	25 (50.0)	24 (48.0)
	Obese (≥30)	9 (18.0)	8 (16.0)
	Mean±SD	26.8±3.6	26.4±3.8
Duration of infertility (years)	Mean±SD	3.8±2.1	3.6±2.0

Table 2: Baseline clinical profile and PCOS phenotype markers.

Variable	Control (n=50), N (%)/mean±SD	Myo-inositol (n=50), N (%)/mean±SD
Oligo/amenorrhea	38 (76)	40 (80)
Clinical hirsutism (mFG ≥8)	24 (48)	22 (44)
Acne	20 (40)	18 (36)
Acanthosis nigricans	17 (34)	15 (30)
Family history of diabetes	14 (28)	13 (26)
Prior failed OI attempt	21 (42)	20 (40)
Baseline systolic BP (mmHg)	114.6±10.8	113.2±11.1
Baseline diastolic BP (mmHg)	74.2±7.6	73.8±7.8

Table 3: Baseline biochemical and endocrine profile.

Parameter	Control (n=50), mean±SD	Myo-inositol (n=50), mean±SD
LH (IU/l)	10.2±4.1	9.8±4.0
FSH (IU/)	5.6±1.4	5.7±1.5
LH: FSH ratio	1.86±0.78	1.74±0.73
Total testosterone (ng/dl)	58.5±18.6	56.2±17.9
Fasting glucose (mmol/l)	5.2±0.7	5.1±0.7
Fasting insulin (µIU/ml)	14.1±5.6	13.7±5.4
HOMA-IR	3.3±1.5	3.1±1.4
TSH (mIU/l)	2.3±1.0	2.2±0.9
Prolactin (ng/ml)	15.4±6.3	14.8±6.0

Table 4: Baseline pelvic ultrasound characteristics.

Variable	Control (n=50), mean±SD	Myo-inositol (n=50), mean±SD
Mean ovarian volume (ml)	11.6±3.9	11.2±3.7
Antral follicle count (AFC)	22.1±6.8	21.6±6.6
Baseline endometrial thickness (mm)	4.7±1.1	4.8±1.2

Table 5: Ovulation induction cycle response and key outcomes (primary outcomes).

Outcome	Control (n=50) N (%)/mean±SD	Myo-inositol (n=50) N (%)/mean±SD	P value
Dominant follicle achieved (≥18 mm)	34 (68.0)	41 (82.0)	0.10
Day of dominant follicle (days)	13.1±2.4	12.6±2.2	0.29
Endometrial thickness on trigger/day 12 (mm)	7.4±1.3	8.1±1.4	0.01
Ovulation confirmed	28 (56.0)	37 (74.0)	0.04
Mid-luteal progesterone (ng/ml)	8.9±3.7	10.6±4.1	0.03
Monofollicular response	23 (46.0)	30 (60.0)	0.16
Multifollicular response (≥2 follicles ≥16 mm)	8 (16.0)	10 (20.0)	0.60

Figure 1 depicts that myo-inositol supplementation was a significant positive predictor (adjusted OR 2.30, 95% CI 1.01-5.24, p=0.047), resulting in a more than doubling of ovulation odds after correcting for confounders. Insulin resistance, evaluated by HOMA-IR, had a significant negative correlation (adjusted OR 0.81 per unit increase, 95% CI 0.67-0.98, p=0.030), demonstrating insulin resistance as an independent barrier to ovulation. BMI showed a negative correlation (adjusted OR 0.92 per kg/m², p=0.053), reaching statistical significance. Age, prior ovulation induction failures, and oligo/amenorrhea could not independently predict ovulation outcomes, indicating that myo-inositol advantages are widespread among PCOS subtypes.

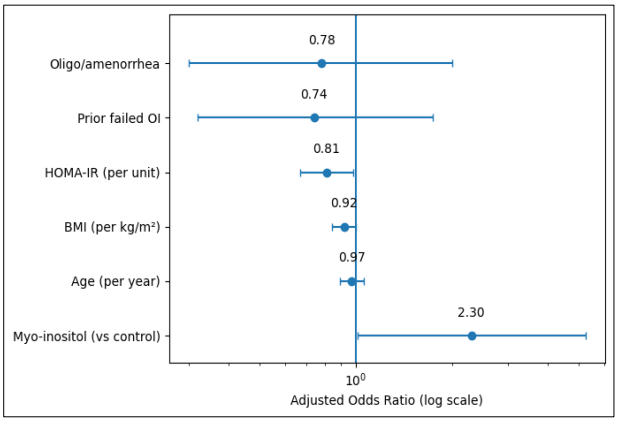

Figure 1: Forest plot of adjusted odds ratios for predictors of ovulation outcomes in women with PCOS.

DISCUSSION

This prospective study demonstrates that the supplementation of myo-inositol indeed increases ovulation rates and enhances endometrial receptivity in PCOS patients who undergo induction of ovulation. There was an absolute 18% improvement in ovulation rates in the study, increasing from 56% to 74%, which is significant at p=0.04. There was also a significant improvement found in endometrial thickness from 7.4 mm to 8.1 mm in our study. Our study also substantiates the theory of using inositol in PCOS management by means of insulin-sensitizing action and improvement in oocyte quality.[17,18] The mechanism by which myo-inositol has a beneficial effect on the body is complex. First, myo-inositol, as part of the phosphatidylinositol signaling system, is involved in insulin signal transduction, thereby enhancing insulin sensitivity and alleviating compensatory hyper-insulinemia, a hallmark of PCOS.[19] Papaleo et al also suggested that myo-inositol supplementation significantly enhanced insulin sensitivity as well as hormonal status in PCOS patients, comparable to the improved metabolic profile we also noted.[20] Moreover, inositol phosphoglycans act as second messengers for insulin action, which also facilitates glucose uptake and decreases androgen biosynthesis.[21] Similarly, the notable increase in mid-luteal progesterone seen in our study (10.6 ng/ml versus 8.9 ng/ml, p=0.03) would indicate an improvement in follicular maturation and corpus luteum function, which is a mechanism also purportedly backed by the work of Unfer et al, where myo-inositol has been shown to optimize FSH signaling.[22] Of particular note are the endometrial benefits seen. The statistically significant improvement in endometrial thickness seen (0.7 mm difference, p=0.01) holds clinical significance; endometrial thickness less than 7 mm is associated with poor implantation rates, while between 8-14 mm optimizes pregnancy outcomes.[23] This might suggest myo-inositol improves endometrial receptivity through up-regulation of estrogen receptor expression and improving uterine perfusion. Zheng et al's meta-analysis has previously shown that inositol supplementation improved pregnancy rates in a clinical setting in women with PCOS, and partly attributed this to endometrial development.[24] Myo-inositol supplementation was an independent predictor of successful ovulation, as assessed by the multivariable

logistic regression analysis, adjusted for age, BMI, insulin resistance, previous treatment failures, and menstrual irregularity (adjusted OR=2.30, p=0.047). This analysis strengthens causal inference by adjusting for confounding variables. Of note, HOMA-IR was an independent negative predictor of ovulation (adjusted OR=0.81, p=0.030), which further strengthens the patho-physiological link between insulin resistance and anovulation. Given the insulin-sensitizing action of myo-inositol, as shown by Greff et al in their study of the metabolic and reproductive effects of inositol, this may be considered a mediator of the ovulation-promoting action of myo-inositol.[25] Agarwal et al showed in PCOS women that myo-inositol supplementation improved ovarian function, which was reflected in improved ovulation, and it reduced hyperandrogenism, thus supporting our findings for ovulation improvement.[26] The results from the systematic review carried out by Pacchirotti et al accord a positive role of myo-inositol in improving ovulation rates along with metabolic parameters in the PCOS population and supported our primary findings.[27] Myo-inositol represents a cost-effective, well-tolerated adjuvant treatment that can be easily integrated into traditional ovulation induction regimens. Compared to traditional pharmacological insulin-sensitizing drugs like metformin, which are significantly associated with gastrointestinal side effects, myo-inositol shows optimal tolerability and safety profiles.[28,29] Moreover, the 12-week supplementation regimen was found to be adequate to induce considerable effects, though the optimal duration and dosage regimen need to be clarified. Moreover, the dosage regimen of 4 g/dl, given twice a day at 2 g each time, corresponds to standard dosages found to be effective in PCOS clinical trials.

Limitations

The study's small sample size may have limited statistical power for detecting differences in secondary pregnancy outcomes, though primary ovulation endpoints achieved significance. Long-term pregnancy outcomes and live birth rates require extended follow-up beyond the 12-week gestational timepoint assessed.

CONCLUSION

Supplementation with 4 grams of myo-inositol significantly enhances ovulation rates and endometrial growth in women with PCOS undergoing ovulation induction. Therapy possessed an independent predictive value for the likelihood of successful ovulation following control for metabolic and phenotypic confounders. Higher mid-luteal progesterone levels and promising pregnancy trends further reinforce myo-inositol's role as an effective, safe, and well-tolerated adjuvant in fertility management among women with PCOS. These results support the inclusion of myo-inositol supplementation as part of standard ovulation induction protocols for the optimization of reproductive outcomes in PCOS populations.

Recommendations

Future studies should investigate dose-response relationships, optimal supplementation duration, and comparative efficacy against other insulin sensitizers. Large-scale randomized controlled trials powered for live birth outcomes would definitively establish myo-inositol's position in evidence-based PCOS fertility treatment algorithms.

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