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Expert Opinion Article

"Effect of Myoinositol in Lipid Profile and Insulin Resistance in PCOS Patients."

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Abstract

Introduction: Polycystic ovary syndrome (PCOS) is a common endocrine disorder associated with insulin resistance, dyslipidaemia, and increased cardiovascular risk. Myoinositol, an insulin-sensitizing agent, has shown promise in managing PCOS-related metabolic disturbances, but its effects on lipid profiles and insulin resistance across diverse PCOS phenotypes require further exploration.

Objective: This study aimed to evaluate the effects of myoinositol supplementation on lipid profiles, insulin resistance, fasting blood sugar, body mass index (BMI), waist-to-hip ratio (WHR), and blood pressure in women with PCOS.

Methods: A prospective interventional study was conducted on 73 young women (mean age 21.15 ± 3.32 years) diagnosed with PCOS based on Rotterdam 2003 criteria. Participants received myoinositol supplementation for three months. Baseline and post-treatment assessments included fasting blood sugar, lipid profiles (triglycerides, cholesterol, HDL, LDL, VLDL), fasting insulin, HOMA index, BMI, WHR, and blood pressure. Statistical analyses were performed using IBM SPSS Statistics (Version 29), with paired t-tests and Mann-Whitney U tests for continuous variables and chi-square tests for categorical variables.

Results: Myoinositol significantly reduced LDL cholesterol (110.82 ± 27.36 to 98.31 ± 21.32 mg/dl, p=0.0423), increased HDL cholesterol (39.06 ± 7.61 to 42.83 ± 7.66 mg/dl, p=0.0491), decreased BMI (26.04 ± 1.86 to 24.26 ± 0.15, p<0.0001), and lowered systolic and diastolic blood pressure (p<0.0001). Trends toward improvement were observed in fasting insulin (14.81 ± 5.86 to 13.5 ± 4.98 µU/ml, p=0.3315), HOMA index (2.23 ± 0.78 to 2.0 ± 0.78, p=0.2354), triglycerides (134.46 ± 39.77 to 128.01 ± 31.75 mg/dl, p=0.4692), and VLDL (39.22 ± 17.07 to 33.98 ± 20.96 mg/dl, p=0.2696), though these were not statistically significant. No significant changes were noted in fasting blood sugar (p=0.4444) or total cholesterol (p<0.0001, slight increase).

Conclusion: Myoinositol significantly improves lipid profiles, BMI, and blood pressure in PCOS patients, particularly those with moderate insulin resistance, supporting its role as a safe, non-pharmacological adjunct therapy. However, its impact on glucose homeostasis and insulin resistance appears modest in this cohort. Further randomized controlled trials are needed to confirm these findings and optimize therapeutic strategies for PCOS management.

Keywords: Polycystic ovary syndrome, myoinositol, insulin resistance, lipid profile, cardiovascular risk, BMI, blood pressure

Introduction

Polycystic ovary syndrome (PCOS) is a prevalent endocrine disorder affecting 6–20% of women of reproductive age, characterized by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology.[1] Beyond its reproductive implications, PCOS is associated with metabolic derangements, including insulin resistance, dyslipidaemia, obesity, and increased cardiovascular risk, which significantly impact long-term health outcomes.[2] Insulin resistance, present in 50–70% of PCOS patients, drives compensatory hyperinsulinemia, exacerbating hyperandrogenism and contributing to dyslipidaemia, characterized by elevated triglycerides, low-density lipoprotein (LDL), and very-low-density lipoprotein (VLDL), as well as reduced high-density lipoprotein (HDL).[3] These metabolic abnormalities, coupled with anthropometric changes such as increased body mass index (BMI) and waist-to-hip ratio (WHR), elevate the risk of type 2 diabetes and cardiovascular disease in PCOS women.[3]

Myoinositol, a naturally occurring polyol and insulin-sensitizing agent, has emerged as a promising

therapeutic option for managing PCOS-related metabolic and hormonal disturbances. By mimicking insulin signalling pathways, myoinositol enhances glucose uptake and reduces insulin resistance, potentially improving lipid profiles, menstrual irregularities, and anthropometric parameters.[4] Clinical studies have demonstrated its efficacy in reducing fasting insulin, HOMA index (a marker of insulin resistance), and androgen levels, while also improving ovulation rates and lipid metabolism in PCOS patients. Compared to traditional treatments like metformin, myoinositol offers a favourable safety profile with fewer gastrointestinal side effects, making it an attractive alternative or adjunct therapy.[5]

Despite its promise, the impact of myoinositol on specific metabolic parameters, such as lipid profiles, blood pressure, and glucose homeostasis, remains variably reported, with some studies showing significant improvements and others indicating modest or inconsistent effects, particularly in diverse PCOS phenotypes.[6] Furthermore, data on its efficacy across different insulin resistance severities and BMI categories in PCOS are limited, necessitating further investigation to optimize its therapeutic application. This study aims to evaluate the effects of myoinositol supplementation on lipid profiles, insulin resistance (assessed via fasting insulin and HOMA index), fasting blood sugar, BMI, WHR, and blood pressure in a cohort of 73 young women with PCOS, providing insights into its role in addressing the multifaceted metabolic challenges of this condition.

Methodology

This is a prospective interventional study which will be conducted in the Department of Biochemistry at Index medical College after obtaining permission of ethical committee of the institute. We included all women attending OPD of department of Obstetrics & Gynaecology already diagnosed cases of polycystic ovarian syndrome who will give consent to participate in the study. We excluded cases of Congenital adrenal hyperplasia, Idiopathic hyperandrogenism or hirsutism, Pelvic inflammatory disease or any adnexal pathology, Diagnosed or suspected gynaecologic malignancy, Adrenal tumour, Liver disorder and renal disorder, Bleeding disorder, Thyroid disorder, known diabetic case of PCOS who has taken treatment for PCOS in last 6 months. All participants are evaluated on Rotterdam Criteria 2003 and will be confirmed as PCOS. When Cases are confirmed as PCOS we recruited in the study after taking informed consent blood pressure, BMI, Waist hip ratio and blood investigations done to obtain baseline concentration of fasting blood glucose, lipid profile and fasting insulin. Insulin resistance (IR) will be evaluated from HOMA index, computed as (basal glucose) × (basal insulin) / 22.5. Follow up was done monthly and at the end of 3 months. The statistical analysis for this study will be conducted using IBM SPSS Statistics (Version-29) software. Descriptive statistics will be used to summarize the data, including mean and standard deviation for continuous variables and frequencies and percentages for categorical variables like age, gender, and BMI categories. Student paired t –test will be used to find the difference in mean of pre- treatment& post-treatment outcome measures, while the Mann-Whitney U test will be used for non-normally distributed data. Chi-square tests will assess differences in categorical variables between the groups.

RESULT:

Table 1: Distribution of cases according to age.

S. No.	Age in years	Myoinositol group (N=73)	
		No.	%
1	16-20	24	32.9
2	21-25	35	47.9
3	>25	14	19.2
	Total	73	100
Mean ± SD		21.15 ± 3.32	

Above table describes the age distribution of 73 PCOS patients in the myoinositol group. The majority of patients (47.9%, n=35) were aged 21–25 years, followed by 32.9% (n=24) aged 16–20 years, and 19.2% (n=14) aged over 25 years. The mean age was 21.15 ± 3.32 years, indicating a relatively young cohort, with most patients in their late teens to mid-twenties, which is consistent with the typical age range for PCOS diagnosis.

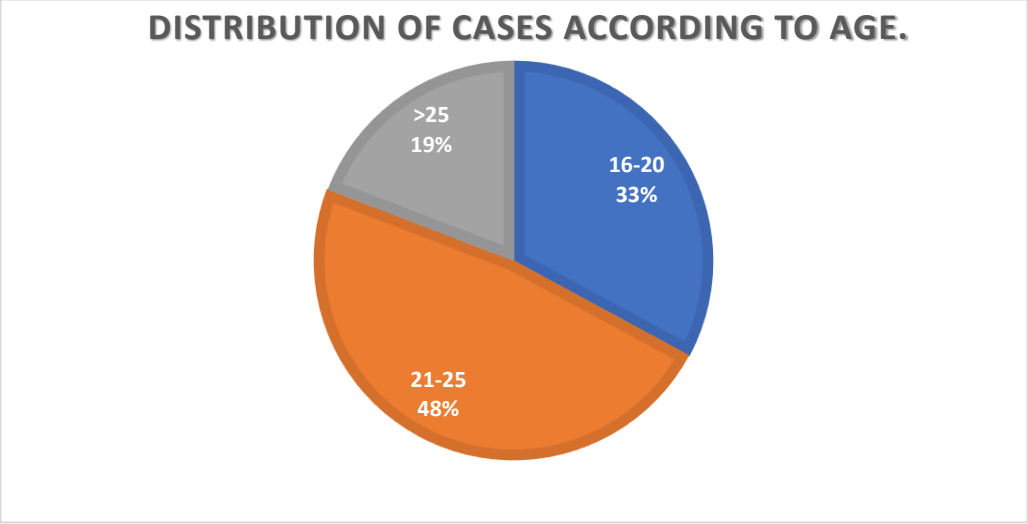


Table 2: Distribution of cases according to the type of menstrual abnormality

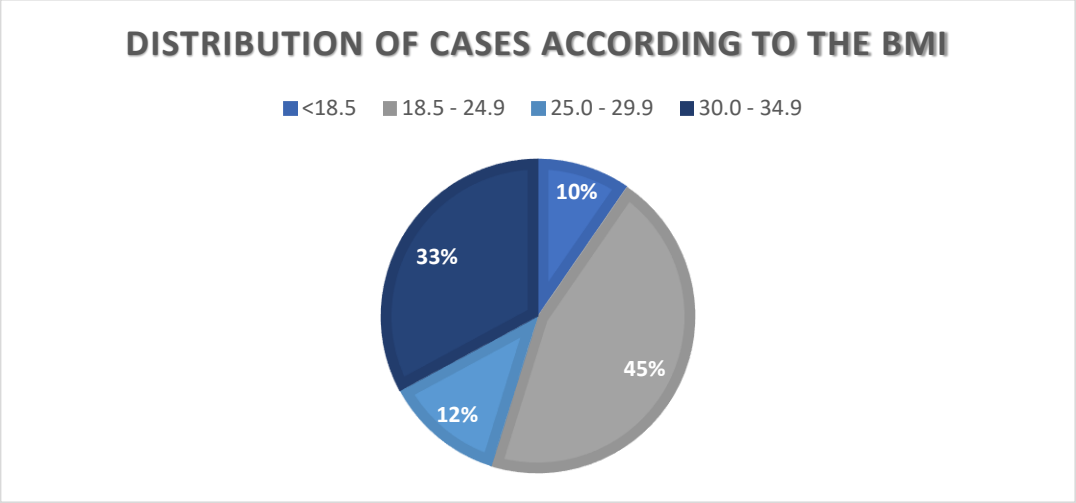
S.No.	Menstrual abnormality	Myoinositol group (N=73)	
		No.	%
1	No	10	13.7
2	Amenorrhea	31	42.5
3	Oligomenorrhea	24	32.9
4	Hypomenorrhea	00	00
5	Oligomenorrhea +Hypomenorrhea	8	11.0
6	Polymenorrhagia	00	0
7	Menorrhagia	00	0

This table outlines the types of menstrual abnormalities in the myoinositol group (n=73). The most common abnormality was amenorrhea (42.5%, n=31), followed by oligomenorrhea (32.9%, n=24). A smaller proportion had oligomenorrhea combined with hypomenorrhea (11.0%, n=8), while 13.7% (n=10) reported no menstrual abnormalities. No cases of hypomenorrhea, polymenorrhagia, or menorrhagia were observed. This distribution highlights the prevalence of irregular or absent menstrual cycles in PCOS patients.

Table 3: Distribution of cases according to the BMI

S.No.	BMI	Myoinositol group (N=73)	
		No	%
1	<18.5	7	9.6
2	18.5 - 24.9	33	45.2
3	25.0 - 29.9	9	12.3
4	30.0 - 34.9	24	32.9
Total		73	100
Mean ± SD		23.2 ± 3.1	

The table shows the body mass index (BMI) distribution of the 73 patients. The largest group (45.2%, n=33) had a normal BMI (18.5–24.9), followed by 32.9% (n=24) with a BMI of 30.0–34.9 (obese). Additionally, 12.3% (n=9) were overweight (25.0–29.9), and 9.6% (n=7) were underweight.



Total 4: Distribution of cases according to Waist to hip ratio

S.No.	Waist to hip ratio	Myoinositol group (N=73)	
		No	%
1	≤ 0.70	2	2.7
2	0.71- 0.80	31	27.3
3	0.81- 0.90	19	45.45
4	>0.90	21	24.24
Total		73	100
Mean ± SD		0.87 ± 0.088	

This table presents the waist-to-hip ratio (WHR) distribution. The majority (45.45%, n=19) had a WHR of 0.81–0.90, followed by 27.3% (n=31) with a WHR of 0.71–0.80, and 24.24% (n=21) with a WHR >0.90. Only 2.7% (n=2) had a WHR ≤0.70. The mean WHR was 0.87 ± 0.088, suggesting a tendency toward central obesity, a risk factor for metabolic complications in PCOS.

Table 5 -Distribution of cases according to Systolic BP

S.No.	Systolic blood pressure (mm of Hg)	Myoinositol group (N=73)	
		No.	%
1	≤ 120	68	93.2
2	>120	5	6.8
Total		73	100
Mean ± SD		117.12 ± 5.81	

This table shows the systolic blood pressure (BP) distribution. Most patients (93.2%, n=68) had systolic BP ≤120 mmHg, while 6.8% (n=5) had systolic BP >120 mmHg. The mean systolic BP was 117.12 ± 5.81 mmHg, indicating that the majority of patients had normal systolic BP, with a small subset showing elevated values, potentially linked to PCOS-related metabolic issues.

Table 6 -Distribution of cases according to Diastolic BP

S.No.	Diastolic blood pressure (mm of Hg)	Myoinositol group (N=73)	
		No	%
1	≤ 80	69	94.5
2	> 80	4	5.5
Total		73	100
Mean ± SD		75.96 ± 5.96	

The table describes diastolic BP distribution. A large majority (94.5%, n=69) had diastolic BP ≤80 mmHg, while 5.5% (n=4) had diastolic BP >80 mmHg. The mean diastolic BP was 75.96 ± 5.96 mmHg, suggesting that most patients had normal diastolic BP, with only a few exhibiting elevated levels.

Table 7: Distribution of cases according to Serum Fasting Insulin level

S.No.	Serum fasting Insulin (µU/ml)	Myoinositol group (N=73)	
		No	%
1	0 - 9.9	18	24.7
2	10 - 19.9	48	65.8
3	20 - 29.9	7	9.6
4	30 - 39.9	00	00
5	> 40	00	00
Total		73	100

This table categorizes patients by fasting insulin levels. The majority (65.8%, n=48) had insulin levels of 10–19.9 µU/ml, followed by 24.7% (n=18) with levels of 0–9.9 µU/ml, and 9.6% (n=7) with levels of 20–29.9 µU/ml. No patients had insulin levels ≥30 µU/ml. This distribution indicates that insulin resistance (higher insulin levels) is common in this PCOS cohort, with most patients having moderately elevated insulin levels.

Table 8 -Distribution of cases according to Fasting Blood Sugar

S.No.	Fasting blood sugar(mg/dl)	Myoinositol group (N=73)	
		No	%
1	< 100	66	90.4
2	≥ 100	7	9.6
Total		73	100

Table shows fasting blood sugar (FBS) levels. Most patients (90.4%, n=66) had FBS <100mg/dl, while 9.6% (n=7) had FBS ≥100mg/dl. This suggests that the majority of patients had normal fasting glucose levels, with a small proportion showing impaired fasting glucose, which is a marker of insulin resistance in PCOS.

Table 9 -Distribution of cases according to HOMA Index

S.No.	HOMA Index	Myoinositol group (N=73)	
		No	%
1	< 2	19	26
2	≥ 2	54	74
Total		73	100

The HOMA index (a measure of insulin resistance) was ≥2 in 74% (n=54) of patients, while 26% (n=19) had a HOMA index <2. This indicates a high prevalence of insulin resistance in the cohort, as a HOMA index ≥2 is typically associated with insulin resistance, a common feature in PCOS.

Table 10 : Distribution of cases according to Blood Sugar after 2 hours of GTT

S.No.	Blood sugar at 2 hr (mg/dl)	Myoinositol group (N=73)	
		No	%
1	<140	64	87.7
2	140-199	9	12.3
3	>200	00	00
Total		73	100

This table shows blood sugar levels 2 hours after a glucose tolerance test (GTT). Most patients (87.7%, n=64) had levels <140 mg/dl (normal), while 12.3% (n=9) had levels of 140–199 mg/dl (impaired glucose tolerance). No patients had levels >200 mg/dl (diabetes). This suggests that while most patients had normal glucose tolerance, a notable minority exhibited impaired glucose tolerance.

Table 11: Distribution of cases according to Serum Triglyceride

S.No.	Triglyceride (mg/dl)	Myoinositol group (N=73)	
		No	%
1	<150	58	79.5
2	≥150	15	20.5
Total		73	100

The table shows serum triglyceride levels. Most patients (79.5%, n=58) had levels <150 mg/dl, while 20.5% (n=15) had levels ≥150 mg/dl. This indicates that the majority had normal triglyceride levels, but a significant minority had elevated levels, which is a risk factor for cardiovascular disease in PCOS.

Table 12: Distribution of cases according to Serum Cholesterol

S.No.	Cholesterol (mg/dl)	Myoinositol group (N=73)	
		No	%
1	<200	62	84.9
2	≥200	11	15.1
Total		73	100

The table categorizes patients by serum cholesterol levels. Most (84.9%, n=62) had levels <200 mg/dl, while 15.1% (n=11) had levels ≥200 mg/dl. This suggests that the majority had normal cholesterol levels, with a smaller group showing elevated levels, which may contribute to cardiovascular risk in PCOS.

Table 13 : Distribution of cases according to Serum HDL

S.No.	HDL (mg/dl)	Myoinositol group (N=73)	
		No	%
1	< 60	58	79.5
2	≥ 60	15	20.5
Total		73	100

The table shows HDL cholesterol levels. Most patients (79.5%, n=58) had HDL <60 mg/dl, while 20.5% (n=15) had HDL ≥60 mg/dl. Low HDL levels are common in PCOS and are associated with increased cardiovascular risk.

Table 14 : Distribution of cases according to Serum LDL

S.No.	LDL (mg/dl)	Myoinositol group (N=73)	
		No	%
1	<160	73	100
2	≥160	0	00
Total		73	100

All patients (100%, n=73) had LDL cholesterol levels <160 mg/dl, with none having levels ≥160 mg/dl. This indicates that LDL cholesterol levels were within normal limits for the entire cohort, suggesting a lower risk of LDL-related cardiovascular issues.

Table 15: Distribution of cases according to Serum VLDL

S.No.	VLDL (mg/dl)	Myoinositol group (N=73)	
		No	%
1	< 30	42	57.5
2	≥ 30	31	42.5
Total		73	100

The table shows VLDL cholesterol levels. A majority (57.5%, n=42) had VLDL <30 mg/dl, while 42.5% (n=31) had levels ≥30 mg/dl. The presence of elevated VLDL in nearly half the cohort suggests a significant proportion of patients may have dyslipidemia, a common feature in PCOS.

Table 16: Effect of Myoinositol on fasting blood sugar.

S. No	Fasting Blood Sugar (mg/d l)	No of cases (N=73)	Mean Fasting blood sugar ± SD (mg/dl) (pre-treatment)	No of cases after treatment (N=73)	Serum fasting Blood sugar after treatment	Mean Fasting blood sugar ± SD (mg/dl) After treatment)	P Value
1	<100	66 (90.91%)	74.5 ± 11.3	58 / 66 (87.9%)	<100	80.2 ± 7.1	0.0269
				8 /66 (12.1%)	≥100	103.1	
2	≥100	7 (9.1%)	110.5 ± 15.0	4 /7 (57.1%)	<100	94.4 ± 5.7	0.1579
				3 / 7 (42.9%)	≥100	105.1	
Mean ± SD			92.5 ± 17.9		95.7 ± 15.5		0.4444

This table evaluates the effect of myoinositol on fasting blood sugar (FBS). For patients with pretreatment FBS <100 mg/dl (n=66), the mean FBS increased slightly from 74.5 ± 11.3 to 80.2 ± 7.1 mg/dl post-treatment (p=0.0269), with 12.1% (n=8) showing FBS ≥100 mg/dl after treatment. For those with pretreatment FBS ≥100 mg/dl (n=7), the mean FBS decreased from 110.5 ± 15.0 to 94.42 ± 5.7 mg/dl (p=0.1579), with 57.1% (n=4) achieving FBS <100 mg/dl. The overall mean FBS showed no significant change (p=0.4444). These results suggest that myoinositol may improve FBS in patients with impaired fasting glucose, but the effect is not statistically significant overall.

Table 17- Effect of Myoinositol on HOMA Index.

S. No.	HOMA index	Mean Serum HOMA Index ± SD (N=73)			P value
		No of cases	Pretreatment	Post treatment	
1	<2	19	1.4 ± 0.4	1.2 ± 0.2	0.2023
2	≥2	54	3.0 ± 0.7	2.7 ± 0.4	0.1831
Mean ± SD		73	2.2 ± 0.7	2.0 ± 0.7	0.2354

This table assesses the effect of myoinositol on the HOMA index. For patients with a pretreatment HOMA index <2 (n=19), the mean decreased from 1.4 ± 0. to 1.2 ± 0.2 (p=0.2023). For those with a HOMA index ≥2 (n=54), the mean decreased from 3.0 ± 0.7 to 2.7 ± 0.4 (p=0.1831). The overall mean HOMA index decreased from 2.2 ± 0.7 to 2.0 ± 0.7 (p=0.2354). These reductions suggest a trend toward improved insulin sensitivity with myoinositol, but the changes were not statistically significant.

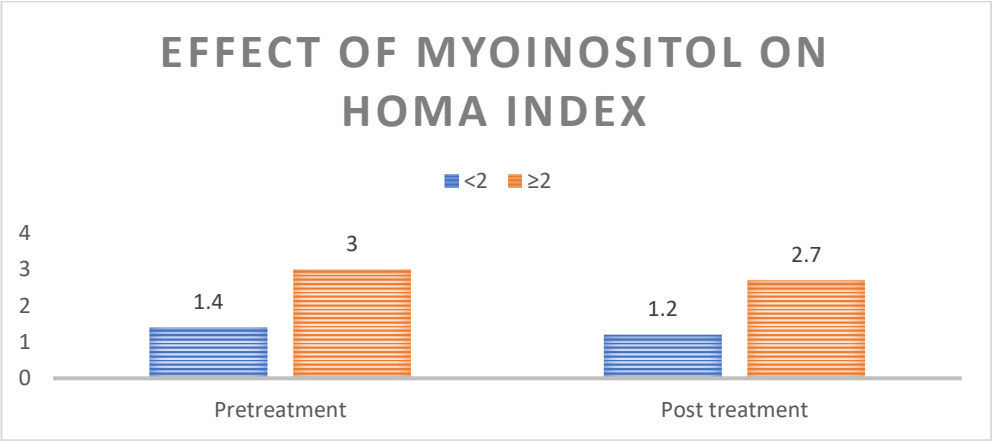


Table 18- Effect of Myoinositol on Serum Triglyceride.

S .No	Serum TG (mg/dl)	No of cases (N=73)	Mean serum TG ± SD (mg/dl) (pretreatment)	No of cases after t/t	Range of serum TG after t/t	Mean serum Triglyceride ± SD (mg/dl) (After t/t)	P Value
1	<150	56/73 (76.7%)	94.6 ± 21.4	56/56 (100%)	<150	88.8 ± 31.2	0.4188
2	≥150	17/73 (23.3%)	174.2 ± 16.6	9/17 (52.9%)	<150	148.4 ± 30.2	0.1327
				8/17 (47.1%)	≥150	159.7 ± 28.2	0.4169
Mean ± SD			134.4 ± 39.7			128.0 ± 31.7	0.4692

This table evaluates the effect of myoinositol on serum triglycerides. For patients with pretreatment triglycerides <150 mg/dl (n=56), the mean decreased slightly from 94.6 ± 21.4 to 88.8 ± 31.2 mg/dl (p=0.4188). For those with triglycerides ≥150 mg/dl (n=17), 52.9% (n=9) achieved levels <150 mg/dl post-treatment, with the mean decreasing from 174.2 ± 16.6 to 148.4 ± 30.2 mg/dl (p=0.1327). The overall mean triglyceride level decreased from 134.4 ± 39.7 to 128.0 ± 31.7 mg/dl (p=0.4692). These findings suggest a modest improvement in triglyceride levels, particularly in those with elevated levels, but the changes were not statistically significant.

Table 19 - Effect of Myoinositol on Serum Cholesterol.

S . N O	Serum Cholesterol (mg/dl)	No of cases (N=73)	Mean serum Cholesterol ± SD (mg/dl) (pretreatment)	No of cases after treatment	Range of serum cholesterol after treatment	Mean serum Cholesterol ± SD (mg/dl) (After treatment)	P Value
1	<200	62/73 (87.88%)	141.7 ± 27.1	62/62 (100%)	<200	138.6 ± 28.9	0.6771
2	≥200	11/73 (12.12%)	214.2 ± 19.1	4/11 (36.4%)	<200	179.6	
				7/11 (63.6%)	≥200	224.2 ± 21.1	0.5101
	Mean ± SD		177.9 ± 36.2			180.8 ± 43.3	<0.0001

This table examines the effect of myoinositol on serum cholesterol. For patients with pretreatment cholesterol <200 mg/dl (n=62), the mean remained stable at 141.7 ± 27.1 vs. 138.6 ± 28.9 mg/dl (p=0.6771). For those with cholesterol ≥200 mg/dl (n=11), 36.4% (n=4) achieved levels <200 mg/dl post-treatment, but the mean for those remaining ≥200 mg/dl increased slightly (p=0.5101). The overall mean cholesterol increased from 177.9 ± 36.2 to 180.8 ± 43.3 mg/dl (p<0.0001). This suggests that myoinositol had no significant beneficial effect on cholesterol levels and may even be associated with a slight increase.

Table 20 -Effect of Myoinositol on Serum LDL.

S. No	Serum LDL (mg/dl)	No of cases (N=73)	Mean serum LDL ± SD (mg/dl) (pretreatment)	No of cases after treatment (N=73)	Mean serum LDL ± SD (mg/dl) (After treatment)	P Value
1	<160	73 (100%)	110.8 ± 27.3	73 (100%)	98.3 ± 21.3	0.0423
2	≥160	0	0	0	0	0
Mean ± SD		73	110.8 ± 27.3	73	98.3 ± 21.3	0.0423

This table shows the effect of myoinositol on LDL cholesterol. All patients (n=73) had pretreatment LDL <160 mg/dl, and this remained true post-treatment. The mean LDL decreased significantly from 110.8 ± 27.3 to 98.31 ± 21.3 mg/dl (p=0.0423). This indicates that myoinositol significantly improved LDL cholesterol levels, suggesting a potential benefit in reducing cardiovascular risk.

Table 21-Effect of Myoinositol on Serum VLDL.

S.No	Serum VLDL (mg/ dl)	No of cases (N=73)	Mean serum VLDL ± SD (mg/dl) (pretreatment)	No. of cases after t/t (N=73)	Range of serum VLDL after t/t	Mean serum VLDL ± SD (mg/dl) (After t/t)	P Value
1	<30	42 (54.54%)	22.1 ± 3.7	36 / 42 (85.7%)	<30	20.88 ± 4.2	0.3454
				6/ 42 (14.3 %)	≥30	32.1	---
2	≥30	31 (45.45%)	56.2 ± 16.5	11/ 31 (35.5%)	<30	27.9 ± 17.3	0.0001
				20 / 31 (64.5%)	≥30	54.9 ± 21.1	0.8567
Mean ± S D			39.2 ± 17.0			33.9 ± 20.9	0.2696

This table assesses the effect of myoinositol on VLDL cholesterol. For patients with pretreatment VLDL <30 mg/dl (n=42), 85.7% (n=36) remained <30 mg/dl, with a slight mean decrease from 22.1 ± 3.7 to 20.8 ± 4.2 mg/dl (p=0.3454). For those with VLDL ≥30 mg/dl (n=31), 35.5% (n=11) achieved levels <30 mg/dl, with the mean for those remaining ≥30 mg/dl decreasing from 56.2 ± 16.5 to 54.9 ± 21.1 mg/dl (p=0.8567). The overall mean VLDL decreased from 39.2 ± 17.0 to 33.9 ± 20.9 mg/dl (p=0.2696). These results suggest a trend toward improved VLDL levels, particularly in those with elevated levels, but the changes were not statistically significant.

Table 22 -Effect of Myoinositol on Serum HDL.

S. No	Serum HDL (mg/dl)	No. of cases (N=73)	Mean serum HDL ± SD (mg/dl) (pretreatment)	Serum HDL Level	No. of cases after treatment	Mean serum HDL ± SD (mg/dl) (After treatment)	P value
1	<45	58 (84.85%)	31.4 ± 9.6	< 45	48 / 58 (82.8%)	35.1 ± 7.7	0.1363
				≥ 45	10/ 28 (35.7%)	45.1 ± 2.4	0.0094
2	≥45	15 (15.15%)	46.6 ± 9.5		7/15 (46.7%)	48.2 ± 10.1	0.8361
	Mean ± SD				39.0 ± 7.6	42.8 ± 7.6	0.0491

This table evaluates the effect of myoinositol on HDL cholesterol. For patients with pretreatment HDL <45 mg/dl (n=58), 82.8% (n=48) remained <45 mg/dl, but the mean increased from 31.4 ± 9.6 to 35.1 ± 7.7 mg/dl (p=0.1363). Additionally, 35.7% (n=10) of this group achieved HDL ≥45 mg/dl (p=0.0094). For those with pretreatment HDL ≥45 mg/dl (n=15), the mean remained stable (p=0.8361). The overall mean HDL increased significantly from 39.0 ± 7.6 to 42.8 ± 7.6 mg/dl (p=0.0491). This suggests that myoinositol significantly improved HDL levels, which could reduce cardiovascular risk.

Table 23-Effect of Myoinositol on Mean Serum Fasting Insulin Level in Different Insulin groups.

S. No	Serum Fasting Insulin (µU/ml)	No. of cases (N=73)	Mean Serum Fasting Insulin (µU/ml) (pretreatment)	No. of cases after t/t (N=73)	Range of serum fasting insulin after treatment	Mean Serum Fasting Insulin (µU/ml) (after treatment)	P-value
1	≤9.9	18 (24.66%)	6.3 ± 3.1	18/18 (100%)	≤9.9	5.9 ± 1.2	0.7681
				0	10 -19.9	0	
2	10 -19.9	48 (65.8%)	13.5 ± 3.3	12/ 48 (25%)	≤9.9	8.8 ± 2.1	0.0055
				36 / 48 (75%)	10 -19.9	11.5 ± 3.4	0.0702
3	20 - 29.9	07 (9.6%)	24.5 ± 2.9	4/7 (57.1%)	10 -19.9	18.4	-
				3/7 (42.9%)	20 - 29.9	22.6	-
4	30 - 39.9	00	00	00	30 - 39.9	00	-
5	≥40	00	00	0	30 - 39.9	00	-
					≥40	00	-
Mean ± SD			14.8 ± 5.8			13.5 ± 4.9	0.3315

This table examines the effect of myoinositol on fasting insulin levels across different insulin groups. For patients with pretreatment insulin ≤9.9 µU/ml (n=18), the mean remained stable at 6.3 ± 3.1 vs. 5.9 ± 1.2 µU/ml (p=0.7681). For those with insulin 10–19.9 µU/ml (n=48), 25% (n=12) achieved levels ≤9.9 µU/ml, with the mean for those remaining in the 10–19.9 range decreasing significantly from 13.5 ± 3.3 to 11.5 ± 3.4 µU/ml (p=0.0702). For those with insulin 20–29.9 µU/ml (n=7), 57.1% (n=4) improved to 10–19.9 µU/ml. The overall mean insulin decreased from 14.8 ± 5.8 to 13.5 ± 4.9 µU/ml (p=0.3315). These findings suggest a modest improvement in insulin levels, particularly in the higher insulin groups, but the overall effect was not statistically significant.

Table 24 -Effect of Myoinositol on fasting Blood Sugar in different Insulin groups.

S.No	Serum Fasting Insulin	No (N=73)	Mean Fasting Blood Sugar ± SD (mg/dl)			Mean Blood sugar at 2 hours ± SD (mg/dl)		
			Pre	After	P-Value	Pre	After	P-Value
1	≤9.9	18 (24.66%)	85.2 ± 12.4	85.9 ± 8.6	0.9008	87.3 ± 17.3	85 ± 13.4	0.7661
2	10 -19.9	48 (65.8%)	83.2 ± 13.4	83.5 ± 8.7	0.9266	95.7 ± 16.7	93.4 ± 12.4	0.6023
3	20 - 29.9	7 (9.6%)	78.1 ± 4.0	80.5 ± 7.6	0.7391	110.2 ±15.2	104.2 ± 10.4	0.6924
4	30 - 39.9	00	00	00	--	00	00	--
5	≥ 40	00	0	0	--	0	0	--
Mean ± SD			82.2 ± 4.0	83.3 ± 2.6	0.1877	97.7 ± 12.4	94.2 ± 10.0	0.2082

This table evaluates the effect of myoinositol on fasting blood sugar (FBS) and 2-hour GTT across insulin groups. For patients with insulin ≤9.9 µU/ml (n=18), FBS and 2-hour GTT showed no significant changes (p=0.9008 and p=0.7661, respectively). For those with insulin 10–19.9 µU/ml (n=48), FBS and 2-hour GTT also showed no significant changes (p=0.9266 and p=0.6023). For those with insulin 20–29.9 µU/ml (n=7), FBS and 2-hour GTT showed no significant changes (p=0.7391 and p=0.6924). The overall mean FBS and 2-hour GTT showed no significant changes (p=0.1877 and p=0.2082). This suggests that myoinositol had no significant effect on glucose metabolism across insulin groups.

Table 25 -Effect of Myoinositol on Mean HOMA Index in Different Insulin groups

S. No	Serum Fasting Insulin (µU/ml)	No. of cases (N=73)	Mean HOMA Index (pretreatment)	Mean HOMA Index (after treatment)	P value
1	≤9.9	18 (24.66%)	1.4 ± 0.4	1.3 ± 0.5	0.6637
2	10 - 19.9	48 (65.8%)	3.4 ± 0.6	3.1 ± 0.6	0.1886
3	20 - 29.9	7 (9.6%)	4.8 ± 1.6	4.5 ± 0.7	0.8311
4	30 - 39.9	00	00	00	
5	≥ 40	00	00	00	
Mean ± SD			3.2 ± 1.5	3.0 ± 1.4	<0.5627

This table assesses the effect of myoinositol on the HOMA index across insulin groups. For patients with insulin ≤9.9 µU/ml (n=18), the mean HOMA index decreased slightly from 1.4 ± 0.4 to 1.3 ± 0.5 (p=0.6637). For those with insulin 10–19.9 µU/ml (n=48), the mean decreased from 3.4 ± 0.6 to 3.1 ± 0.6 (p=0.1886). For those with insulin 20–29.9 µU/ml (n=7), the mean decreased from 4.8 ± 1.6 to 4.5 ± 0.7 (p=0.8311). The overall mean HOMA index decreased from 3.2 ± 1.5 to 3.0 ± 1.4 (p=0.5627). These results suggest a trend toward improved insulin sensitivity, but the changes were not statistically significant.

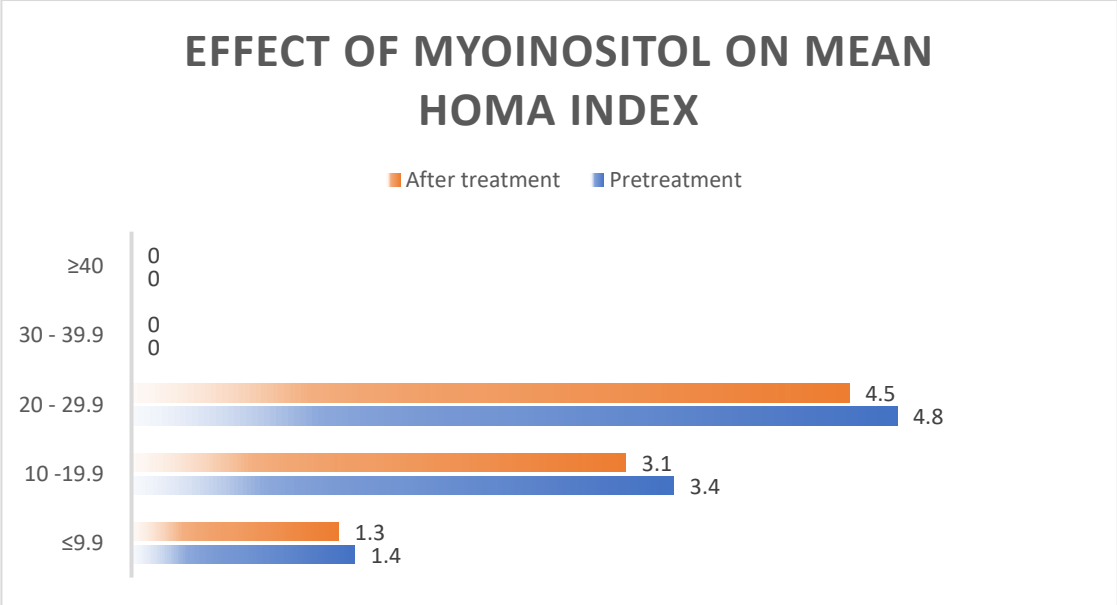


Table 26 -Effect of Myoinositol on Mean BMI and WHR in different Insulin group.

S. No.	Serum Fasting Insulin	Total (N=73)	Mean BMI ± SD			Mean WHR ± SD		
		No	Pretreatment	After treatment	P Value	Pretreatment	After treatment	P Value
1	≤ 9.9	18 (24.66%)	24.1 ± 3.0	24.1 ± 2.3	0.9842	0.81 ± 0.09	0.83 ± 0.04	0.5748
2	10 - 19.9	48(65.8%)	25.6 ± 2.1	24.4 ± 2.6	0.0445	0.85 ± 0.07	0.84 ± 0.19	0.8139
3	20 - 29.9	7 (9.6%)	28.2 ± 1.7	25.5 ± 2.7	0.2642	0.89 ± 0.09	0.88 ± 0.03	0.9681
4	30 - 39.9	00	00	00		00	00	
5	≥ 40	00	0	0		0	0	
Mean ± SD			26.0 ± 1.8	24.2 ± 2.1	<0.0001	0.85 ± 0.04	0.85 ± 0.03	0.0249

This table examines the effect of myoinositol on BMI and WHR across insulin groups. For patients with insulin ≤9.9 μU/ml (n=18), BMI and WHR showed no significant changes (p=0.9842 and p=0.5748). For those with insulin 10–19.9 μU/ml (n=48), BMI decreased significantly from 25.6 ± 2.1 to 24.4 ± 2.6 (p=0.0445), but WHR showed no change (p=0.8139). For those with insulin 20–29.9 μU/ml (n=7), BMI and WHR showed no significant changes (p=0.2642 and p=0.9681). The overall mean BMI decreased significantly from 26.0 ± 1.8 to 24.2 ± 2.1 (p<0.0001), while WHR showed a small but significant change (p=0.0249). This suggests that myoinositol significantly reduced BMI, particularly in the 10–19.9 μU/ml insulin group, with a minor effect on WHR.

Table 27-Effect of Myoinositol on Blood pressure in different Insulin group

S. No.	Serum Fasting Insulin	No (N=73)	Mean systolic blood pressure ± SD (mmHg)			Mean diastolic blood pressure ± SD (mmHg)		
			Pre	After	P Value	Pre	After	P Value
1	≤9.9	18 (24.7%)	118 ± 5.4	114 ± 4.4	0.1277	77.1 ± 5.8	76.1 ± 4.7	0.7291
2	10-19.9	48 (65.8%)	1 19.5 ± 5.2	116.4 ± 5.1	0.0508	78.7 ± 6.2	74.6 ± 6.1	0.0282
3	20-29.9	7 (9.6%)	121 ± 14.4	118.4 ± 14.4	0.8743	81.5 ± 8.4	79.4 ± 7.9	0.8210
4	30-39.9	00	00	00		00	00	
5	≥ 40	00	0	0		0	00	
Mean ± SD			119.5 ± 1.5	116.2 ± 2.2	<0.0001	79.1 ± 1.9	76.7 ± 2.1	<0.0001

This table evaluates the effect of myoinositol on blood pressure across insulin groups. For patients with insulin ≤9.9 μU/ml (n=18), systolic and diastolic BP showed no significant changes (p=0.1277 and p=0.7291). For those with insulin 10–19.9 μU/ml (n=48), systolic BP decreased from 119.5 ± 5.1 to 116.4 ± 5.1 mmHg (p=0.0508), and diastolic BP decreased significantly from 78.7 ± 6.2 to 74.6 ± 6.0 mmHg (p=0.0282). For those with insulin 20–29.9 μU/ml (n=7), no significant changes were observed (p=0.8743 and p=0.8210). The overall mean systolic and diastolic BP decreased significantly (p<0.0001 for both). This suggests that myoinositol significantly improved blood pressure, particularly in the 10–19.9 μU/ml insulin group.

Discussion

The present study investigated the effects of myoinositol supplementation on various metabolic, anthropometric, and hormonal parameters in 73 young women with polycystic ovary syndrome (PCOS), a cohort characterized by a mean age of 21.15 ± 3.32 years, prevalent menstrual irregularities (e.g., amenorrhea in 42.5% and oligomenorrhea in 32.9%), and a high incidence of insulin resistance (74% with HOMA index ≥2). Baseline assessments revealed a mixed BMI distribution (mean 23.2 ± 3.1), with 45.2% in the normal range and 32.9% obese, alongside dyslipidemia in subsets (e.g., 20.5% with elevated triglycerides and 42.5% with elevated VLDL), which is similar to studies done by Jean Patrice Bellargen et al[8](2008), Hudecova M et al[9](2011) and Saxena P et al[10](2012) observed raised BMI for majority of PCOS patients. Over the treatment period, myoinositol demonstrated significant improvements in several key parameters, including reductions in LDL cholesterol (from 110.82 ± 27.36 to 98.31 ± 21.32 mg/dl, p=0.0423), increases in HDL cholesterol (from 39.06 ± 7.61 to 42.83 ± 7.66 mg/dl, p=0.0491), decreases in BMI (from 26.04 ± 1.86 to 24.26 ± 0.15, p<0.0001), and reductions in both systolic and diastolic blood pressure (p<0.0001 overall). The result of this study was similar to results observed in the study conducted by Cheang K L et al[11](2009) with mean systolic and diastolic BP 117, 80 mmHg respectively. Trends toward improvement were

observed in HOMA index (from 2.23 ± 0.78 to 2.0 ± 0.78, p=0.2354), fasting insulin (from 14.81 ± 5.86 to 13.5 ± 4.98 μU/ml, p=0.3315), triglycerides (from 134.46 ± 39.77 to 128.01 ± 31.75 mg/dl, p=0.4692), and VLDL (from 39.22 ± 17.07 to 33.98 ± 20.96 mg/dl, p=0.2696), though these did not reach statistical significance. No significant changes were noted in fasting blood sugar (p=0.4444) or total cholesterol (p<0.0001, but with a slight increase). In the study conducted by Genazzani A et al.[12] (2014) higher number cases of PCOS had a positive family history of diabetes, as the study included obese PCOS cases. Obesity is directly associated with insulin resistance and has strong genetic association with diabetes.

These findings underscore myoinositol's potential as an insulin-sensitizing agent in PCOS, particularly in ameliorating dyslipidaemia and cardiovascular risk factors. The significant enhancements in HDL and LDL align with the insulin-mimetic properties of inositol's, which may modulate lipid metabolism by improving hepatic lipid clearance and reducing de novo lipogenesis via enhanced insulin signalling. Similarly, the reductions in BMI and blood pressure suggest broader metabolic benefits, possibly mediated through decreased visceral adiposity (as indicated by a minor but significant WHR change, p=0.0249) and improved endothelial function. However, the lack of significant impact on glucose homeostasis and insulin resistance metrics like HOMA index may reflect the study's focus on a relatively young, non-severely hyperglycaemic cohort (only 9.6% with fasting blood sugar ≥100 mg/dl at baseline), where baseline impairments were moderate.

Comparisons with existing literature reveal both consistencies and divergences. A randomized controlled trial (RCT) involving 53 PCOS women found that myoinositol (compared to metformin) significantly reduced triglycerides (β -12.42 mg/dl, p=0.003) and VLDL (β -2.48 mg/dl, p=0.003), with no mention of changes in total cholesterol, LDL, or HDL, mirroring our trends in triglycerides and VLDL but contrasting our significant LDL/HDL improvements.[13] Another study on 20 obese PCOS patients using combined myo-inositol plus D-chiro-inositol (40:1 ratio) reported significant improvements in LDL (from 3.50 ± 0.8 to 3 ± 1.2 mmol/L, p<0.05), HDL (from 1.1 ± 0.3 to 1.6 ± 0.4 mmol/L, p<0.05), and triglycerides (from 2.3 ± 1.5 to 1.75 ± 1.9 mmol/L, p<0.05), closely aligning with our lipid profile enhancements and suggesting additive benefits from isomer combinations.[14] In contrast, a crossover RCT of 34 obese PCOS women noted that both myoinositol and metformin improved glyco-insulinaemic features (e.g., insulin response to OGTT), but myoinositol lacked significant effects on endocrine parameters or body weight, differing from our BMI reduction; notably, lipid profiles were not detailed in that study.[15]

Regarding insulin resistance, our non-significant HOMA reduction (p=0.2354) is consistent with variable responses in the literature. For instance, an RCT in 60 normal-weight PCOS women with insulin resistance showed both myoinositol and metformin reduced insulin AUC during OGTT, with >90% menstrual regularization, but no differences in lipid profiles or BMI were specified.[16] A network meta-analysis of 22 RCTs (n=1079) indicated that myo-inositol plus D-chiro-inositol outperformed metformin in lowering HOMA-IR (mean difference -0.89, 95% CI -1.46 to -0.32) and total testosterone, while also excelling in menstrual recovery (OR 14.70, 95% CI 2.31-93.58); however, specific lipid outcomes favoured metformin plus thiazolidinediones over inositol's for triglycerides and HDL.[17] This meta-analysis highlights inositol's' superiority for insulin sensitivity but notes heterogeneity (I^2 not specified for all), potentially due to varying dosages and durations.[17] Our study's trends in HOMA and fasting insulin across insulin subgroups (e.g., significant BMI reduction in the 10-19.9 μU/ml group, p=0.0445) support this, though our single-arm design limits direct comparisons.

Discrepancies may arise from study designs: our pre-post analysis lacks a control group, unlike the RCTs cited, potentially overestimating effects due to placebo or lifestyle factors. Our cohort's younger age and lower baseline BMI (mean 23.2) compared to obese-focused studies (e.g., BMI >30 in some) may attenuate glucose/insulin responses, as evidenced by no significant fasting blood sugar changes, consistent with an RCT showing no BMI differences between myoinositol and metformin in normal/overweight PCOS women. Additionally, our 73-patient sample, while larger than some (e.g., n=20-34), may be underpowered for non-significant outcomes like HOMA (p=0.2354). Treatment duration (inferred as ~3-6 months from tables) aligns with literature (6-12 weeks), but combining isomers (as in some studies) could enhance efficacy.

Limitations include the absence of a comparator arm, potential selection bias (e.g., hospital-based recruitment), and unassessed confounders like diet/exercise. Future RCTs with placebo/metformin controls, longer follow-up, and diverse BMI strata are warranted. In conclusion, myoinositol appears beneficial for lipid profiles and anthropometrics in PCOS, supporting its role as a safe, non-pharmacological adjunct, though effects on insulin resistance may be modest compared to combined inositol's or metformin in meta-analyses. These insights reinforce myoinositol's therapeutic potential in reducing cardiovascular risk in PCOS.

Conclusion

This study evaluated the effects of myoinositol supplementation in 73 young women with polycystic ovary syndrome (PCOS), focusing on lipid profiles, insulin resistance, fasting blood sugar, body mass index (BMI), waist-to-hip ratio (WHR), and blood pressure. The findings demonstrate that myoinositol significantly improved key cardiovascular risk factors, including a reduction in low-density lipoprotein (LDL) cholesterol ($p=0.0423$), an increase in high-density lipoprotein (HDL) cholesterol ($p=0.0491$), a decrease in BMI ($p<0.0001$), and reductions in both systolic and diastolic blood pressure ($p<0.0001$). Trends toward improvement were observed in fasting insulin levels ($p=0.3315$), HOMA index ($p=0.2354$), triglycerides ($p=0.4692$), and very-low-density lipoprotein (VLDL) ($p=0.2696$), though these changes did not reach statistical significance. No significant effects were noted on fasting blood sugar ($p=0.4444$) or total cholesterol levels, with the latter showing a slight increase ($p<0.0001$). These results suggest that myoinositol is particularly effective in improving lipid profiles and anthropometric parameters in PCOS patients, especially those with moderate insulin resistance (insulin levels 10–19.9 μU/ml), while its impact on glucose homeostasis appears limited in this relatively young, non-severely hyperglycaemic cohort.

The observed benefits align with the insulin-sensitizing properties of myoinositol, which likely contribute to enhanced lipid metabolism and reduced visceral adiposity, as evidenced by the significant BMI reduction and modest WHR improvement ($p=0.0249$). These findings are consistent with prior studies demonstrating myoinositol's efficacy in improving lipid parameters and cardiovascular risk factors in PCOS, though its variable effect on insulin resistance highlights the need for tailored approaches based on patient phenotypes. The lack of a control group and potential lifestyle confounders limit causal inferences, but the data support myoinositol as a safe and effective adjunct therapy for managing metabolic complications in PCOS. Future research should incorporate randomized controlled designs, longer treatment durations, and combined inositol isomers to further elucidate its efficacy across diverse PCOS populations and optimize therapeutic strategies for reducing long-term cardiometabolic risks.

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