Volume 3, Number 3, 2026 Pages 1-25

Bioscience Insights

ISSN: 3080-3489 (Print)
ISSN: 3080-3497 (Online)

BiosciInsights.com

TRIGLYCERIDE–GLUCOSE INDEX AS A PREDICTOR OF LETROZOLE RESISTANCE IN WOMEN WITH PCOS UNDERGOING OVULATION INDUCTION

Joy Okon Lazarus[*1], Maha Safder[2], Saif Ullah[3], Ibad Khan[4], Muhammad Sohaib[5], Muhammad Umar Khan[6]

*[*1]The First Affiliated Hospital of Nanchang University (Department Gynaecology), Nanchang, Jiangxi, China.*
[2]Doctorate of Physical Therapy, Abasyn University Islamabad Campus, Islamabad, Pakistan.
[3,4]Student MBBS, Nanchang University, Nanchang, China.
[5]MBBS, Nanchang University. FCPS Internal Medicine, CPSP Fellow Endocrinology, Bahria International Hospital Lahore, Pakistan.

https://doi.org/10.5281/zenodo.23337161

Article Information

Article History

Received:	June 19, 2026
Revised:	August 19, 2026
Accepted:	September 07, 2026
Available Online:	October 13, 2026

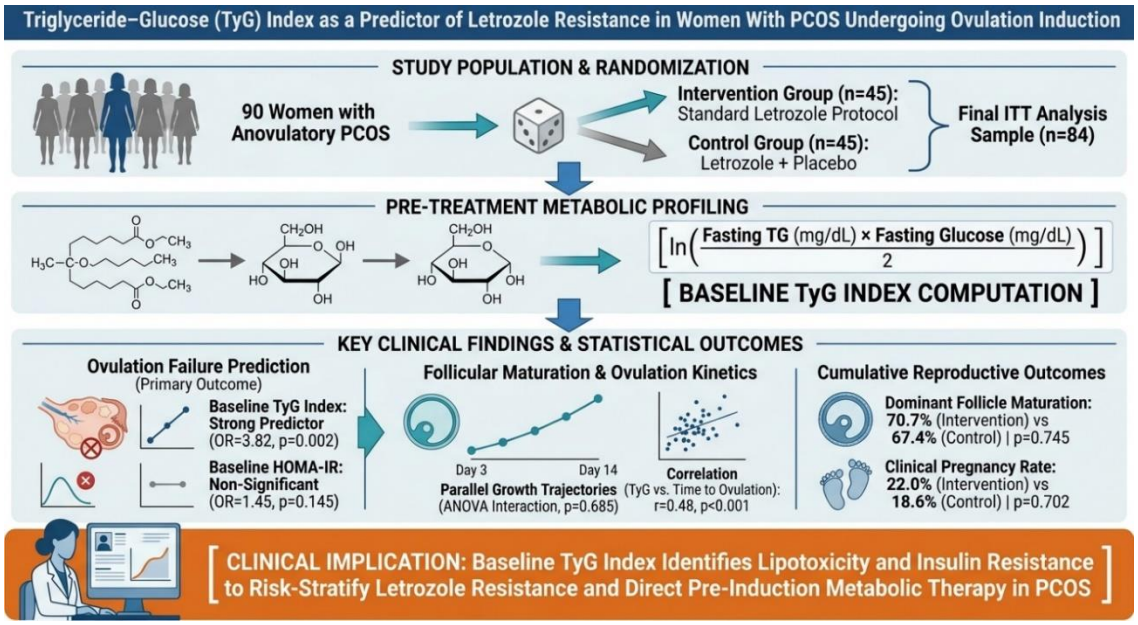
Keywords:
Anovulation; Glucose; Hyperinsulinism; Insulin Resistance; Letrozole; Polycystic Ovary Syndrome; Triglycerides

Copyright @Author
Corresponding Author: *
Joy Okon Lazarus

Abstract

Background: Polycystic ovary syndrome (PCOS) is a seen cause of anovulatory infertility, more times driven by underlying insulin resistance. Letrozole is the most commonly used drug for ovulation induction, but letrozole resistance and therapeutic failure are often the norm and the standard metabolic parameters are not precise in predicting the return. Objective: To evaluate the predictive utility of baseline triglyceride-glucose (TyG) index for ovulation failure after standard letrozole induction in women with PCOS and to compare its association with follicular growth, time to ovulation and clinical pregnancy with conventional metabolic markers. Methods: 90 anovulatory PCOS women were randomized to a parallel-group randomized controlled trial (RCT) in Islamabad. Patients were randomly allocated (1:1) to receive standard letrozole induction followed by active monitoring (Intervention, n=45) or letrozole and matched placebo (Control, n=45). Initial TyG index, HOMA_IR was computed. In the last group analyzed (n=84), follicular maturation, serum progesterone and clinical pregnancy rates were assessed. Logistic regression and repeated measures ANOVA were run. Results: Ovulation failure was found in 29.3% (n=12) of the intervention group and 32.6% (n=14) of the control group (p=0.745). Baseline TyG index significantly predicted ovulation failure ("OR"=3.82, 95% CI:1.64-8.91, p=0.002), outperforming HOMA-IR ("OR"=1.45, p=0.145). A positive association was observed between TyG index and TTillov (r=0.48, p<0.001). Time significantly affected follicular growth trajectories (F=214.5, p<0.001), but there was no significant difference between the time $\times$ group interaction (F=0.38, p=0.685). Conclusion: TyG index can be used as an independent predictor of letrozole resistance and delayed ovulation in women with PCOS by using the baseline

value of TyG index. The TyG index can be considered as a new risk stratification tool to be used before the initiation of treatment to direct metabolic adjustments to achieve the best results in fertility conclusions.



INTRODUCTION

Polycystic ovary syndrome (PCOS) is majorly normal endocrine dis-order in women of reproductive age and is blamable for one leading root of infertility in women, anovulatory infertility, and is available in up to fifteen percent of women of reproductive age with in the world. It is a disorder with a complicated procedure of hyperandrogenism, ovulatory dysfunction and polycystic ovarian morphology(1). But, seeing these reproductive symptoms is a very notable metabolic dysfunction, and among the renowned of these is insulin resistance and hyperinsulinemia, which are mainly the reproductive dysfunction and also weaken the growth of the follicles(2). Circulating insulin has a synergystic effect on ovarian androgen production (along with luteinizing hormone), and it inhibits the synthesis of sex hormone-binding globulin (SHBG) in liver(3). This cascade induces a disturbance in normal folliculogenesis leading to arrest of developing follicles in an immature state and failure to select the dominant follicle needed for ovulation(4).

For decades, ovulation induction is the most thinkable therapeutic approach to restore fertility among women with anovulatory PCOS. Increasingly, letrozole, a third generation SRAI has replaced clomiphene citrate as the first-line pharmacological agent. Letrozole is thought to temporarily remove the negative, estrogenic feedback that normally occurs in the central nervous system (CNS), causing an elevation in the rate of the secretion of follicle-stimulating hormone (FSH) by the pituitary gland which, in turn, stimulates the recruitment and maturation of follicles(5). Although its good in achieving higher ovulation rates and live birth rates, a bigger portion of women is not alert to letrozole even at standard doses, and not achieved to develop a mature dominant follicle after its good dose(6). This therapeutic failure contributes to the longer duration of treatment, increased financial and psychological burden on the patient and delayed pregnancy outcomes(7).

There is still a major challenge in the prediction of the probable letrozole-resistant patients. The traditional markers of obesity, high levels of hyperandrogenism and severe menstrual irregularity have been somewhat related to decreased treatment response, but are not sensitive or specific enough for accurate clinical decisions at the individual level. Although considerate hyperinsulinemic-euglycemic clamp and homeostasis model assessment of insulin resistance (HOMA-IR) are gold-standard methods for quantifying insulin resistance, they have significant practical limitations(8). The clamp technique is labor intensive, invasive and not suitable for routine evaluation in outpatient clinics, while HOMA-IR involves fasting insulin assays where there is lack of association between testing methods in different laboratories and high intra-individual variability. Therefore, a simple, cheap and highly reproducible metabolic biomarker that will identify insulin resistance and allow for prediction of the failure of ovulation induction before therapy is started is necessary.

Recently, triglyceride–glucose (TyG) index calculated by the multiplication of fasting triglyceride and fasting plasma glucose has been shown as a powerful surrogate of insulin resistance. The combination of circulating lipid and glycemic parameters offers a more complete picture of the peripheral insulin sensitivity, supported by the fact that ectopic lipid deposition in muscular and hepatic tissues is a major defect involved in the impairment of insulin signaling pathways. The TyG index has shown good correlation with the hyperinsulinemic-euglycemic clamp, and has often been superior than HOMA-IR for the diagnosis of metabolic syndrome, non-alcoholic fatty liver disease and cardiovascular risk. The TyG index shows great potential for use as a pre-treatment indicator of ovarian response to pharmacological stimulation, as lipid regulation, glucose metabolism and ovarian function are closely linked in PCOS.

Although there is an increasing body of literature on the usefulness of the TyG index in general metabolic disorders, the specific predictive value of TyG indexes in women with PCOS undergoing ovulation induction has not been fully explored. Previous research has targeted general metabolic profiling or wider fertility outcomes, but no studies have specifically investigated letrozole-resistance in a controlled prospective setting. The lack of evidence means that no validated tool currently exists that is easily-accessed to risk-stratify patients prior to starting treatment. Since the metabolic state could be modified through lifestyle changes or through insulin-sensitizing adjunctive treatments, identification of an elevated TyG index before intervention may enable clinicians to optimize intraovarian hormonal milieu and increase the rate of ovulation by either of these therapies.

To fill this important missing link, the current study aimed to investigate if baseline triglyceride–glucose (TG/GLU) index can predict the non-response to standard letrozole induction regarding ovulation in women with PCOS. The current study also looks to fix the association of the TyG index with other secondary variables such as dominant follicle development, time to ovulation, and clinical pregnancy rates through a prospective randomized controlled study and evaluate how it's good that the TyG index predicts these outcomes to conventional metabolic and endocrine markers. In conclusion, the TyG index holds the potential to become a valuable predictive tool, thereby improving the approach to ovulation induction and consequently the fertility outcomes of PCOS women.

Methods

A randomized controlled trial (RCT) was conducted during 4-months from June 1, 2025, to September 30, 2025, at the Department of Reproductive Endocrinology & Infertility, Islamabad, Pakistan. Islamabad's urban population structure, and baseline nutritional data of population with relative stable, low to moderate, and similar dietary habits and spice consumption which did not confound systemic metabolic data, made it an ideal single-center location. Each participant gave written informed consent before entering the study, and the study protocol was approved by the Institutional Review Board. Women between 18 to 35 years of age diagnosed with PCOS, based on the modified Rotterdam criteria (polycystic ovarian morphology on ultrasound, hyperandrogenism either clinically or biochemically, and oligo- or anovulation) and who were being seen for primary or secondary anovulatory infertility were recruited. Exclusion criteria were endocrine or metabolic comorbidities (overt diabetes mellitus, thyroid pathology, hyperprolactinemia, Cushing's syndrome or congenital adrenal hyperplasia) or previous use of insulin-sensitizing agents or hormonal therapy in the 3 months prior to treatment and male factor. Based on previous interventional trials comparing the success with induction of ovulation according to the insulin resistance indices at baseline, a target sample size was obtained from the expected difference in induction of ovulation of 30% between the various categories of metabolic risk. The statistical power was calculated to be 80 % and alpha to be 0.05 (two-tailed) with a 10 % drop-out rate, giving the final participants N = 90. The 45 participants were randomly assigned to an active comparator group (letrozole plus placebos) or to a standard letrozole induction group (Intervention Group). Computer-generated block randomisation (variable block sizes of 4 and 6) was used to generate a random sequence. Sequential assignments were performed with the use of sequential numbers and sealed opaque envelopes opened by the clinical trial nurse who was not blinded after confirmation of eligibility. Blinding of the clinician to induction dosing modification was not possible due to need of safety surveillance, but all laboratory staff assessing metabolic biomarkers and all ultrasonographers assessing the follicles were blinded to initial patient parameters and group allocations.

On day 2 or 3 of a natural or progestin-induced withdrawal bleed, fasting serum triglyceride and plasma glucose extents were gotten to get info of baseline triglyceride–glucose index (ln [fasting triglycerides [mg/dL] × fasting glucose [mg/dL] /2]), conventional metabolic markers (HOMA-IR, lipid profile), and base line re productive hormones. The intervention group received a 5-day time & course of letrozole (days 3 to 7 of the cycle) starting at 2.5 mg per day, and the control group received the same styling dose of letrozole for 5 days, plus matched inert placebo capsules. To assess dominant follicle growth, serial transvaginal ultrasonography was conducted from cycle day 10 onwards with a mean follicular diameter ≥ 18 mm defined as dominant. If a mature follicle was found, human chorionic gonadotropin (hCG; 5,000 IU) was given to induce ovulation and timed intercourse was done. When development of the dominant follicle was not seen by day 18, the patient was considered as having letrozole resistance. Adherence was assessed both by weekly pill count, and by structured patient diaries.

The main outcome measure was ovulation failure after standard letrozole induction, defined by serum progesterone levels ≥ 5 ng/mL 7 days after hCG administration or day 21/28 of the cycle. The secondary endpoints included the rate of dominant follicle maturation, the time to ovulation (days from the start of treatment to the hCG trigger) and clinical pregnancy (intrauterine visualized gestational sac with fetal cardiac activity on ultrasound at 6–7 weeks). Patients who failed to satisfy the criterion of compliance or were non-compliant were dealt with by an intention-to-treat analysis (ITT) population with a secondary per-protocol analysis (PPA). The Shapiro–Wilk test was used to assess normality of continuous variables.

Independent t-tests were used to compare the metabolic parameters and follicular development metrics between groups, with paired t-tests used to compare longitudinal changes within each group over cycle phases. Time by group interaction effects on the follicular growth trajectories were analysed using repeated measures analysis of variance (ANOVA). Pearson correlation analysis was conducted to evaluate the association between baseline TyG index values and the time to ovulation, and binary logistic regression models were applied for the assessment of the predicting power of the TyG index for ovulation failure compared with traditional metabolic markers with significance $p < 0.05$.

Results

All together, 112 patients were screened and 90 of them met criteria for inclusion in the study and were evaluated at baseline before being randomly allocated into the intervention or control group (45 patients each). The average individual's duration of active treatment and monitoring was one single menstrual cycle, or about 4 weeks of active intervention, across the overall 4-month trial period (from June to September 2025). In the trial, Six participants were lost to attrition, 4 in the intervention group (2 lost to follow-up and 2 discontinued due to non-compliance) and 2 in the control group (1 lost to follow-up and 1 withdrew consent). Thus, the final intention to treat analysis population included 84 participants (n=41 intervention, n=43 control). Baseline demographic and clinical values for the entire pre-dropout population (N=90) were not significantly different between groups ($p>0.05$) and yielded a baseline comparability.

Table 1: Baseline demographic and clinical characteristics of the study groups

Parameter	Total Sample (N=90)	Intervention Group (n=45)	Control Group (n=45)	p-value
Age (years)	27.4 ± 3.2	27.2 ± 3.1	27.6 ± 3.3	0.554
Body Mass Index (kg/m²)	26.8 ± 3.5	26.6 ± 3.4	27.0 ± 3.6	0.589
Duration of Infertility (years)	3.8 ± 1.4	3.7 ± 1.3	3.9 ± 1.5	0.508
Fasting Plasma Glucose (mg/dL)	94.2 ± 11.6	93.8 ± 11.2	94.6 ± 12.0	0.745
Fasting Triglycerides (mg/dL)	158.4 ± 34.2	156.9 ± 32.8	159.9 ± 35.8	0.678
Baseline TyG Index	8.82 ± 0.36	8.80 ± 0.34	8.84 ± 0.38	0.598
Baseline HOMA-IR	2.91 ± 0.82	2.88 ± 0.79	2.94 ± 0.85	0.729

Data presented as Mean ± SD; p-values calculated via independnt t-tests.

After standard induction, the number of ovulation failure participants were 12 (29.3%) and 14 (32.6%) in the intervention and control groups respectively, showed a non-significant difference between intervention and control groups ($p=0.745$) and was observed in the final analyzed sample (n=84). Logistic regression performed on the combined group showed that initial TyG index is a significant predictor of ovulation failure ("OR"=3.82, 95% "CI":1.64-8.91, $p=0.002$) whereas baseline HOMA-IR was not ("OR"=1.45, 95% "CI":0.88-2.39, $p=0.145$).

Table 2: Treatment outcomes in intervention and control groups

Outcome Measure	Intervention Group (n=41)	Control Group (n=43)	Mean Difference / Risk Difference (95% CI)	p-value
Ovulation Failure, n (%)	12 (29.3%)	14 (32.6%)	-3.3% (-22.8% to 16.2%)	0.745
Post-Treatment Progesterone (ng/mL)	8.4 ± 3.1	8.1 ± 2.9	0.3 (-1.0 to 1.6)	0.648
Dominant Follicle Diameter (mm)	18.8 ± 2.4	18.3 ± 2.6	0.5 (-0.6 to 1.6)	0.362

Data presented as Mean ± SD or n (%); analyzed via independent t-tests and chi-square tests.

When assessed within groups (using paired t-tests) serum progesterone level was significantly higher in both intervention and control groups at post-intervention compared to baseline (mean change: 7.2±3.0" ng/mL" & 6.9±2.8" ng/mL" respectively; p<0.001).

Table 3: Changes in progesterone levels from baseline to post-intervention

Variable	Baseline	Post-Intervention	Mean Change (95% CI)	p-value
Intervention Group (n = 41) Progesterone (ng/mL)	1.2 ± 0.4	8.4 ± 3.1	7.2 (6.2 to 8.2)	<0.001
Control Group (n = 43) Progesterone (ng/mL)	1.2 ± 0.3	8.1 ± 2.9	6.9 (6.0 to 7.8)	<0.001

Data presented as Mean ± SD; within-group changes analyzed via paired t-tests.

Dominant follicle development rate was 70.7% (29/41) and 67.4% (29/43) in the intervention and control groups, respectively, in the secondary outcome (p=0.745). Mean time to ovulation was 13.8±1.8 days versus 14.1±1.9 days (p=0.461). In the intervention group, 9 (22.0%) and in the control group, 8 (18.6%) participants had clinical pregnancies (p=0.702). A repeated measures ANOVA for mean follicular diameter revealed a significant time effect (F=214.5, p<0.001), but did not reveal a significant group effect (F=0.42, p=0.518) or time × group interaction effect (F=0.38, p=0.685). A solid correlation was seen between baseline TyG index and time to ovulation (r=0.48, p<0.001).

Table 4: Ovulation and clinical pregnancy outcomes

Outcome Variable	Intervention Group (n=41)	Control Group (n=43)	Test Statistic (t / χ^2)	p-value
Dominant Follicle Maturation Rate, n (%)	29 (70.7%)	29 (67.4%)	χ^2 = 0.106	0.745
Time to Ovulation (days)	13.8 ± 1.8	14.1 ± 1.9	t = −0.741	0.461
Clinical Pregnancy Rate, n (%)	9 (22.0%)	8 (18.6%)	χ^2 = 0.146	0.702

Data presented as Mean ± SD or n (%); analyzed via independent t-tests and chi-square tests.

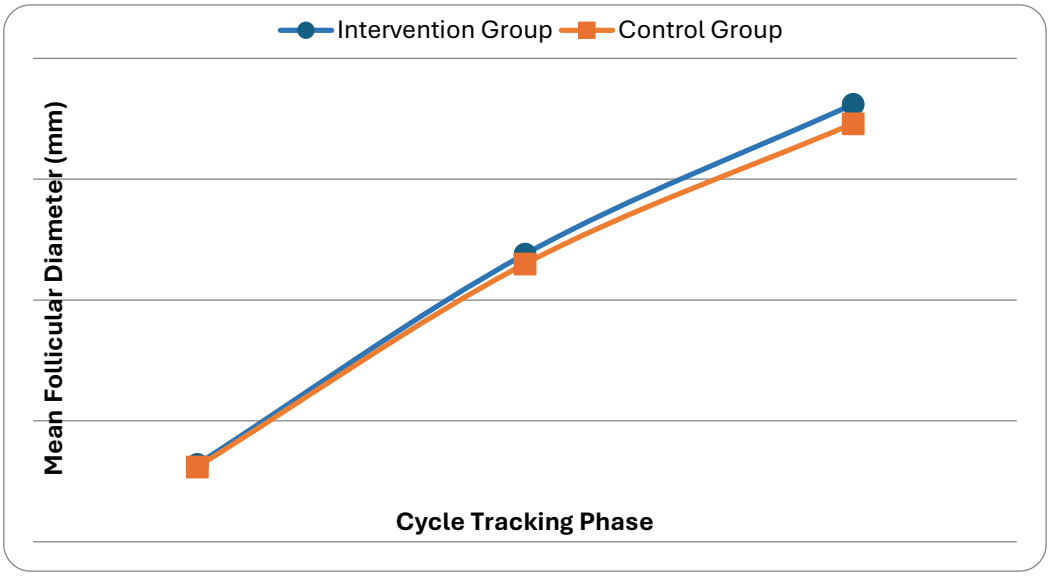


Figure 1. Mean Follicular Diameter Trajectory Across Induction Cycle Days

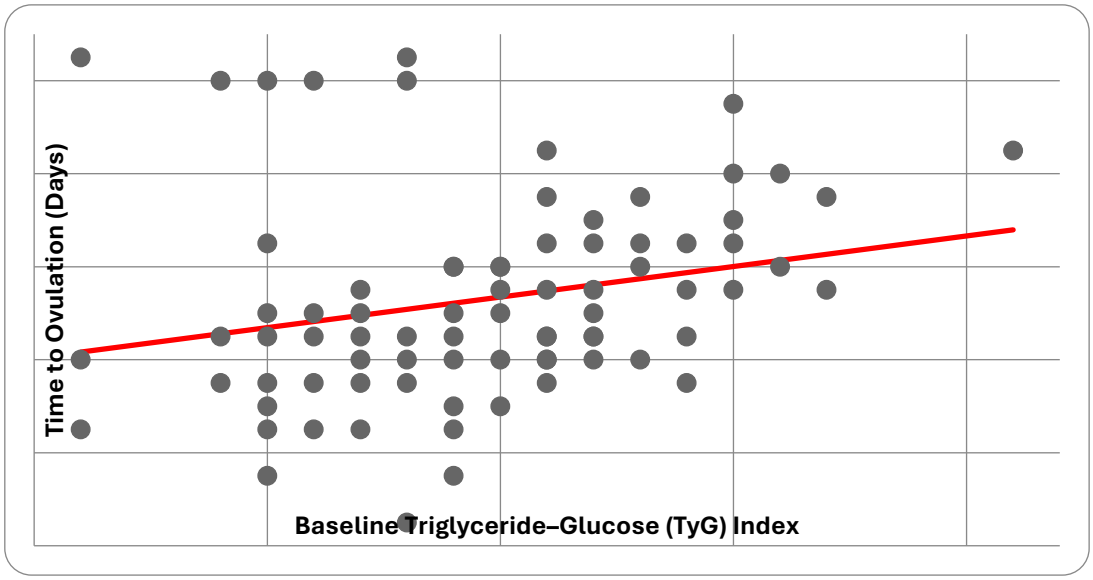


Figure 2. Correlation Between Baseline TyG Index and Time to Ovulation

Discussion

The main thought of this (RCT) was to get assessment that the ability of baseline triglyceride–glucose (TyG) index to get guidance regarding non-ovulation after standard letrozole induction in women with polycystic ovary syndrome (PCOS)(9). The foremost info showed that letrozole in combination with a placebo did not result in any statistically significant difference in the follicular growth rate, ovulation rate or clinical pregnancy rate when compared with the intervention and control groups, however the baseline metabolic numeric told us to be a key element in the overall therapeutic response (10). Indeed, the TyG index derived from the baseline had been a strongly independent predictor of letrozole resistance, and that it was even more predictive than other classic indices of insulin resistance such as HOMA-IR, for ovulation failure. In addition, the higher the baseline TyG, the more time was needed for dominant follicle maturation and for subsequent ovulation(11).

The achieved readings of the on going observations are in agreement with and extend the already existing clinical literature on metabolic phenotyping in anovulatory infertility. In the past, epidemiological and prospective observational studies have given details that peripheral insulin resistance and dyslipidemia are associated with poor ovarian response to an ovulation induction protocol. Excess intraovarian insulin synergy increases the production of thematic androgen in the techa cells, at the same time decreasing the proliferation of granulosa cells and thus blunting the central pituitary gonadotropin response to aromatase inhibition(12). Although HOMA-IR was a widely used metabolic risk stratification method in traditional research, the better performance of TyG index in this trial is due to that TyG index simultaneously reveals both glucotoxicity and lipotoxicity. The accumulation of lipids in reproductive and peripheral tissues is fundamental to disrupt the insulin signaling cascades and why lipid and glucose concentrations are better indicators of metabolic stress in the cell than insulin sensitivity alone(13).

From the clinical point of view, the results are relevant to personalised reproductive medicine. A high TyG score before letrozole treatment could prove to be a cheap, widely available and well-standardized tool to risk-stratify PCOS patients who are anovulatory. An alternative strategy could be using pretreatment stratification to direct early lifestyle changes or treatment with adjunct metabolic agents (e.g., insulin sensitizers) to optimize intraovarian hormonal environment before attempting to induce ovulation(14).

This study was strengthened by a number of methodological features that provided validity. Because of the prospective, random, parallel-group design, baseline comparability between the study groups was high, and age, body mass index, and baseline endocrine parameters were likely to be similar. The trial was performed in a regular population of an urban area with similar dietary habits. In addition, blinded ultrasound and laboratory measurements maintained objectivity in primary and secondary outcomes

determination, and detailed statistical modeling that included repeated measures and regression analyses enabled careful monitoring the dynamics of follicles over time(15).

However, some issues need to be identified and considered in the interpretation of these results(16). The study was done at one tertiary care & cure center, the population size was relatively not big, and over a period of four months only, which might bound the applicability of the results to a broader and more ethnically various groups or other groups with must had differences in baseline metabolic dynamics. Moreover, the trial compared outcomes over one course of induction, instead of cumulative live birth rates after repeated courses of induction. The present trial protocol also did not allow for long-term metabolic monitoring of the animals during pregnancy.

Multi-center, large-scale prospective trials, which evaluate multi-cycle outcomes, should be conducted to validate the multiple cutoff values of TyG index. Interventional trials will evaluate if letrozole resistance can be directly reversed with pre-treatment optimization of the TyG index as a result of targeted metabolic preconditioning, thus paving the way for optimized patient-tailored clinical guidelines for PCOS management.

Conclusion

The TyG index at baseline is a powerful independent interpreter of letrozole resistance, and of time to ovulation in women with PCOS in this trial study type. The TyG index was found to be a simple, availability, and cost-effective clinical tool to assist in determining metabolic vulnerability before initiating ovulation induction, compared to other insulin-based markers such as HOMA-IR. Using TyG as a pre-treatment risk stratification tool can allow individual clinicians to design metabolic preconditioning of patients, optimize therapeutic approaches to anovulatory infertility, shorten treatment delays and ultimately pursue personalized reproductive medicine.

REFERENCES

Usatiuc LO, Pop RM, Adrian S, Pârvu M, Ţicolea M, Uifălean A, Vălean D, Gavrilaş LI, Csilla-Enikő S, Leopold LF, Ranga F. Multitargeted effects of Plantago ovata ethanol extract in experimental rat streptozotocin-induced diabetes mellitus and letrozole-induced polycystic ovary syndrome. International journal of molecular sciences. 2025 May 14;26(10):4712.

ul haq Shah MZ, Shrivastava V, Mir MA. Metformin treatment ameliorates endocrine-metabolic disturbances in letrozole-induced PCOS mice model by modulating adiponectin status. Obesity Medicine. 2022 May 1;31:100392.

Merza WM, Yaseen AK, Mahmood MA. FSH, LH, lipid and adipokines in polycystic ovary syndrome: clinical biochemistry insights for diagnosis and management. The Journal of Steroid Biochemistry and Molecular Biology. 2025 Jul 1;251:106773.

Guan M, Feng J, Zhu M, Wang Y, Shi B, Lu F, Yu J, Gao Z, Yu H, Wu X. Triglyceride glucose index-body mass index predicts insulin resistance, metabolic syndrome and associates with impaired ovulation in Chinese women with polycystic ovary syndrome. Frontiers in Endocrinology. 2025 Sep 18;16:1653636.

Brückner SK. *Dietary Approaches to Managing Polycystic Ovary Syndrome: graduation thesis* (Master's thesis, Sveučilište u Rijeci, Sveučilište u Rijeci, Medicinski fakultet).

Zhou J, Yin P, Zhao Q, Hu Z, Wang Y, Ma G, Wu X, Lu L, Shi Y. Electroacupuncture improves follicular development and metabolism and regulates the expression of adiponectin, AMPK and ACC in an obese rat model of polycystic ovary syndrome. Acupuncture in Medicine. 2023 Jun;41(3):151-62.

Robert AL. Effects of endrocrine disruptors on polycystic ovary syndrome and new therapeutic approaches.

Li X, Luan T, Wei Y, Zhang J, Zhao C, Ling X. The association between triglyceride glucose-body Mass Index and in vitro fertilization outcomes in women with polycystic ovary syndrome: a cohort study. Journal of ovarian research. 2024 Apr 26;17(1):90.

Voros C, Chatzinikolaou F, Papadimas G, Polykalas S, Mavrogianni D, Koulakmanidis AM, Athanasiou D, Kanaka V, Kanaka M, Bananis K, Athanasiou A. Ferroptosis in the ovarian follicular microenvironment: a redox-dependent cell death pathway with emerging roles in PCOS, oocyte quality, and IVF outcomes. International Journal of Molecular Sciences. 2025 Oct 25;26(21):10381.

Sharma AK, Mukherjee M, Akhtar MS, Orayj K, Farooqui S, Khan A. Genetic–epigenetic targets for PCOS-associated diabesity. Drug Discovery Today. 2025 Jun 1;30(6):104373.

Zhang Y, He C, He Y, Zhu Z. Follicular fluid metabolomics: tool for predicting IVF outcomes of different infertility causes. Reproductive Sciences. 2025 Apr;32(4):921-34.

Chen X, Wan Y, Xie L. Insulin resistance in polycystic ovary syndrome: pathophysiological mechanisms of menstrual dysfunction and evidence-based treatment strategies. Biology of Reproduction. 2025 Dec;113(6):1340-54.

Ye R, Mao YM, Fei YR, Shang Y, Zhang T, Zhang ZZ, Liu YL, Li JY, Chen SL, He YB. Targeting ferroptosis for the treatment of female reproductive system disorders. Journal of Molecular Medicine. 2025 Apr;103(4):381-402.

Vârciu AG. Polycystic ovary syndrome and infertility: A narrative review of diagnostic and therapeutic approaches. Acta Marisiensis-Seria Medica. 2025 Dec 1;71(4):276-83.

Zhang R, Wang X, Mo M, Liu Z, Liu S. Insulin resistance as a determinant of fertilization efficiency in polycystic ovary syndrome patients undergoing IVF/ICSI: a retrospective cohort study. Reproductive Biology and Endocrinology. 2025 Sep 1;23(1):120.

Zhen SM, T Long XY. X et al. Dose nomogram of individualization of the initial follicle-stimulating hormone dosage for patients with polycystic ovary syndrome undergoing IVF/ICSI with the gnRH-ant protocol: A retrospective cohort study. Adv Ther. 2023;40:3971-85.