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The Relationship between Anthropometric Indices and Lipid Profile in PCOS Women: A Cross-sectional

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ABSTRACT

Background & aim: Infertility presents a formidable challenge to global public health, with polycystic ovary syndrome (PCOS) emerging as a prominent contributor. PCOS is associated with persistent health complications, such as metabolic syndrome, type 2 diabetes, and cardiovascular diseases. Elevated body weight and central adiposity exacerbate the likelihood of anovulation and infertility in women. This study seeks to examine the relationship between anthropometric measurements and lipid profiles among infertile women with PCOS.

Methods: This cross-sectional study was conducted at infertility treatment center in Mashhad, Iran, between May 2023 and January 2024. The study enrolled 280 married women aged 18–45 years who met inclusion criteria, specifically those diagnosed with both PCOS and infertility. Participants were selected through consecutive sampling and their anthropometric measurements were assessed by trained personnel. Statistical analysis was performed using SPSS. Inferential analyses included Pearson correlation and ANOVA.

Results: A significant positive correlation was found between normal Body Mass Index (BMI) and triglyceride levels (TG) (P=0.030). Likewise, individuals with a BMI exceeding 30 displayed a positive correlation with elevated TG levels (P=0.035). Furthermore, a statistically significant positive correlation was observed between Waist-to-Height Ratio (WHtR) values exceeding 0.57 and elevated TG levels (P=0.003).

Conclusion: PCOS women, characterized by diverse features, exhibited distinct lipid profiles. The intricate relationship observed between lipid metabolism and various PCOS traits may shed light on the heightened susceptibility to long-term complications associated with elevated lipid levels. Regular monitoring of blood lipids and management of central obesity are imperative for women with PCOS.

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Introduction

Polycystic ovary syndrome (PCOS) is one of the most common endocrine and metabolic disorders affecting women of reproductive age . A prevalent contributor to infertility is polycystic ovary syndrome (PCOS) (1). Affecting around 13% of reproductive-age women, PCOS is defined by an excess of androgens, menstrual irregularities, and change in the morphology of ovaries (2). Notably, PCOS is linked to enduring health issues, including an elevated risk of metabolic syndrome, type-2 diabetes, cardiovascular disease, endometrial carcinoma, and obesity (3).

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Various theories surround the etiopathogenesis of PCOS, underscoring the influence of the endocrine environment on affected women. High levels of androgens are key contributors to central obesity, displaying a masculinized fat distribution commonly seen in PCOS. This excess weight and centralized fat accumulation increase the likelihood of anovulation and infertility among affected women (4). Notably, body composition, rather than body weight alone, emerges as a critical factor influencing the origins of obesity as well as its concomitant metabolic and hormonal disorders (3, 5).

Recent investigations emphasize the early emergence of anomalous cardiovascular risk profiles in individuals with PCOS. Women with PCOS frequently display irregularities in lipid metabolism, hypertension, and obesity, along with signs indicative of systemic inflammatory responses. The heightened prevalence of these risk factors aligns the symptoms of many PCOS-afflicted women with those characteristic of metabolic syndrome, a well-recognized risk factor for cardiovascular disorders (6). Notably, There is a clear connection between adiposity and the severity of PCOS symptoms, with a common android-type fat distribution associated with metabolic issues in those affected (7). In addition to cardiovascular risks, lipid disorders are implicated in infertility, as suggested by certain studies reporting reduced fertility potential among couples exhibiting abnormal lipids, evident in the fecundability Odds ratio (FOR) and elevated total cholesterol levels (8-9).

Anthropometry serves as a widely employed research tool for evaluating non-communicable disease risk factors within populations due to its cost-effectiveness and ease of community-level monitoring. Notably, these anthropometric parameters exhibit variability among different populations and lack uniformity. In specific populations, they demonstrate a heightened predictive value in the assessment of non-communicable disease risk factors, including diabetes, hypertension, and dyslipidemia (10). Numerous studies have illustrated diverse patterns of abnormal lipid profiles in women with PCOS, with variations noted across different populations (3, 11-12). Limited research has explored the relationship between lipid profiles and anthropometric indicators in women with PCOS. This research seeks to investigate the potential correlation between anthropometric indices and lipid profiles in infertile women with PCOS.

Materials and Methods

This cross-sectional study was conducted at one of the Infertility Treatment Center , Mashhad, Iran, between May 2023 and January 2024. The study included married women aged 18–45 who met specific inclusion criteria, namely those experiencing both polycystic ovary syndrome (PCOS) and infertility. PCOS diagnosis was confirmed by gynecologists by Rotterdam criteria. .The Rotterdam guidelines define PCOS as a heterogeneous disorder diagnosed by the presence of any two of three key characteristics: ovulatory dysfunction, evidence of hyperandrogenism, and polycystic ovarian morphology on ultrasonography, indicated by an increased follicle count (≥12 follicles of 2–9 mm) and/or enlarged ovarian volume (>10 mL) in at least one ovary (13).

Exclusion criteria encompassed participants with hypothyroidism, diabetes, congenital adrenal hyperplasia, hyperprolactinemia, and Cushing syndrome. total of 280 participants were enrolled in the study using consecutive sampling.

The present study was based on baseline data collected from infertile women with PCOS prior to their participation in a clinical trial. Therefore, no formal sample size calculation was performed for the current cross-sectional analysis. All eligible women with complete baseline data who attended the infertility clinic during the study period were consecutively recruited and included in the analysis.

The anthropometric indices assessed in this study included body weight, height, body mass index (BMI), waist circumference (WC), and waist-to-height ratio (WHtR).

Weight Measurement: Participants' weight was measured using a digital scale with minimal clothing and bare feet while standing upright, with the head in a neutral position and eyes facing forward.

Height Measurement: Height was measured without shoes using a stadiometer, with participants standing erect, heels against the wall, head straight, and eyes facing forward.

Waist Circumference Measurement: Waist circumference was measured in the standing position using a non-elastic tape measure at the narrowest point between the lower costal margin and the iliac crest. Measurements were obtained while participants wore light clothing. If clothing interfered with accurate measurement, participants were asked to remove the outer layer (14-15).

Body Mass Index: BMI was calculated as weight in kilograms divided by the square of height in meters (kg/m^2).(14)

Waist-to-Height Ratio: WHtR was calculated by dividing waist circumference (cm) by height (cm) (16).

All anthropometric measurements were performed by trained personnel using standardized protocols. The measuring instruments were calibrated regularly to ensure the accuracy and reliability of the measurements.

Biochemical parameters of the participants' lipid profile, including triglycerides, total cholesterol, LDL-C, and HDL-C, were assessed following a 14-hour fasting period. Venous sampling involved using a 5 ml syringe to extract 5 ml of blood from the brachial region. Measurements were made in the central laboratory using standard enzymatic methods and commercially available validated assay kits according to the manufacturer's instructions. Internal quality control procedures were routinely performed, and all analyses were conducted by trained laboratory personnel to ensure the accuracy and reliability of measurements.

Statistical analysis was performed using SPSS software version 28. Normality of data was assessed using the Shapiro–Wilk test, and homogeneity of variances was evaluated using Levene's test. Pearson correlation coefficient was used to examine relationships between continuous variables. One-way ANOVA was applied to compare mean differences across groups. Statistical significance was defined as $p < 0.05$.

Results

A total of 280 patients participated in this study. The participants had a mean age of 27.83 ± 5.55, and their mean BMI was 27 ± 4.94 kg/m^2. Detailed anthropometric indices and lipid profile findings are presented in Table 1.

Table 1. Descriptive statistics of anthropometric indices and lipid profiles in the total study population

Variables	Mean±SD	Minimum	Maximum
Age(years)	27.83±5.55	17	45
BMI (kg/m^2)	27.00±4.94	15.60	41.50
Weight (kg)	70.50±13.60	39.4	115.9
Height (cm)	161.64±5.58	147	182
WC (cm)	86.10±10.35	63	111
TC (mg/dl)	167.55±33.55	31	268
TG (mg/dl)	126.43±70.27	38	418
HDL (mg/dl)	43.51±10.34	12.0	72.0
LDL (mg/dl)	97.86±24.49	22.0	166.0
HC	102.45±8.39	84	123
WHIPR	0.79±0.06	0.24	0.67
WHtR	0.53±0.07	0.31	0.39

BMI = body mass index, WC= waist circumference, TC = total cholesterol, TG = triglycerides, HDL = high-density lipoprotein, LDL = low-density lipoprotein, HC= hip circumference, WHIPR= waist-hip ratio, WHtR= waist to height ratio

As outlined in Table 2, a positive correlation was evident between age and cholesterol ($P < 0.001$), age and triglycerides (TG) ($P = 0.032$), as well as age and low-density lipoprotein (LDL)

(P-value = 0.012). Similarly, a positive and statistically significant correlation was identified between Body Mass Index (BMI) and triglycerides (TG) (P-value < 0.001), and between BMI and LDL (P= 0.007). Additionally, positive and significant correlations were observed between Waist Circumference (WC) and triglycerides (TG) (P= 0.002), as well as between Waist–Height Ratio (WHtR) and triglycerides (TG) (P= 0.001).

Table 2. Correlations between anthropometric indices and lipid profiles

Variables	TC	TG	HDL	LDL
Age				
Pearson Correlation	.318	.194	-0.024	.234
Sig. (2-tailed)	0.000	0.032	0.802	0.012
BMI				
Pearson Correlation	0.051	.387	-0.197	.289
Sig. (2-tailed)	0.632	0.000	0.068	0.007
Weight				
Pearson Correlation	0.024	.292	-0.184	.224
Sig. (2-tailed)	0.818	0.004	0.086	0.036
Height				
Pearson Correlation	-0.077	-0.133	-0.051	-0.113
Sig. (2-tailed)	0.464	0.200	0.637	0.293
HC				
Pearson Correlation	-0.242	0.359	-.099	0.19
Sig. (2-tailed)	0.334	0.143	0.724	0.497
WC				
Pearson Correlation	0.196	.358	-0.212	0.193
Sig. (2-tailed)	0.090	0.002	0.073	0.105
WHtR				
Pearson Correlation	.247	.359	-0.177	0.208
Sig. (2-tailed)	0.031	0.001	0.138	0.080
WHIPR				
Pearson Correlation	0.253	0.401	-0.428	0.443
Sig. (2-tailed)	0.311	0.099	0.111	0.098

BMI = body mass index, HC= hip circumference, WC= waist circumference,
WHtR= waist to height ratio, WHIPR= waist-hip ratio

Among all participants, 114 people (40.71%) exhibited normal weight, 96 persons (34.28%) were classified as overweight, and 70 subjects (25%) fell into the obese category. According to Table 3, in women with PCOS, cholesterol and HDL levels did not vary significantly across different body mass index (BMI) categories (P = .322 and P = .485, respectively). However, triglyceride and LDL levels showed significant differences among the BMI categories (P = .016 and P = .010, respectively).

Among women with PCOS, 95 subjects (33.92%) were below 25 years old, 91 person (32.5%) fell within the age range of 26 to 30 years, and 94 (33.57%) were over 31 years old. In women with PCOS, mean cholesterol and LDL levels varied significantly across different age groups (p < .001 and P = .030, respectively). In contrast, triglyceride and HDL levels did not show significant differences between age groups (P = .440 and P = .938, respectively).

Based on the data presented in Table 5, it was determined that within the group of women with PCOS, 157 people (56.07%) had Waist–Height Ratio (WHtR) values below 0.05, while 60 persons (21.42%) fell within the range of 0.51 to 0.56, and 63 persons (22.5%) had WHtR values exceeding 0.57. In women with PCOS, mean cholesterol and LDL levels did not differ significantly across varying waist-to-height ratio (WHtR) levels (P = .062 and P = .098, respectively). However, significant differences were observed in mean triglyceride and HDL levels across different WHtR levels (P = .028 and P = .038, respectively).

Table 3. Mean Comparison of Lipid Profiles Across Various Weight Categories in Women with PCOS

Lipid profile	Normal <25	Over weight : 25-30	Obese ≥ 30	*P

chol	162.83 ± 31.54	164.45 ± 24.20	175.95 ± 43.76	0.322
TG	106.43 ± 51.79	118.49 ± 61.48	159.05 ± 84.35	0.016
HDL	46.02 ± 9.08	42.97 ± 10.58	44.53 ± 13.17	0.485
LDL	93.04 ± 20.88	92.40 ± 24.43	111.82 ± 23.58	0.010

BMI = body mass index, TC = total cholesterol, TG = triglycerides, HDL = high-density lipoprotein, LDL = low-density lipoprotein, *: ANOVA

Table 4. Mean Comparison of Lipid Profiles Across Different Age Groups in Women with PCOS

Lipid profile	≤ 25	26-30	≥ 31	*P
chol	154.39 ± 19.03	**163.39** ± 37.63	182.88 ± 333.72	<0.001
TG	117.49 ± 84.79	124.93 ± 63.95	137.80 ± 62.49	0.440
HDL	43.99 ± 80.74	43.15 ± 12.25	43.38 ± 9.91	0.938
LDL	89.57 ± 17.42	98.97 ± 25.01	104.47 ± 28.08	0.030

BMI = body mass index, TC = total cholesterol, TG = triglycerides, HDL = high-density lipoprotein, LDL = low-density lipoprotein, *: ANOVA

Table 5. Mean Comparison of Lipid Profiles Across Different Categories of WHtR inWomen with PCOS

Lipid profile	≤ 0.5	0.51-0.56	≥ 57	*P
chol	158.27 ± 23.05	163.39 ± 25.97	175.19 ± 29.17	0.062
TG	92.50 ± 35.89	136.48 ± 71.31	131.19 ± 73.75	0.028
HDL	48.49 ± 10.52	40.48 ± 9.53	44.72 ± 11.31	0.038
LDL	90.18 ± 17.23	93.17 ± 26.10	104.04 ± 25.51	0.098

BMI = body mass index, TC = total cholesterol, TG = triglycerides, HDL = high-density lipoprotein, LDL = low-density lipoprotein, *: ANOVA

Discussion

This study revealed significant associations between lipid profiles and various anthropometric measures in women with PCOS. We found positive correlations between age and levels of cholesterol, triglycerides, and LDL, suggesting that older age is linked to higher lipid levels. Similarly, BMI showed strong positive correlations with triglycerides and LDL. Waist circumference (WC) and waist-to-height ratio (WHtR) were also positively correlated with triglycerides. Although cholesterol and HDL levels did not vary significantly across different BMI and age groups, triglycerides and LDL levels demonstrated notable differences. These findings highlight the influence of age, BMI, WC, and WHtR on lipid profiles, underscoring the need to consider these factors in the management of lipid abnormalities in PCOS.

Similar to our study, Sánchez et al.(2021) investigated the impact of women's age on metabolic parameters and lipid profiles. Their findings showed that age was associated with higher total cholesterol and LDL-c, irrespective of body composition and whether the women had PCOS. They also examined how PCOS influenced the relationship between aging and metabolic disorders (3) .The meta-analysis and systematic review of metabolic syndrome and its components in women with PCOS also highlighted several studies that supported the idea that the condition is associated with age (17). The high prevalence of metabolic syndrome in women under the age of thirty is concerning. The review also emphasizes the role of abdominal obesity in this population (18).

Dyslipidemia assumes a crucial role to the development of polycystic ovary syndrome (PCOS), marked by lipid irregularities such as elevated low-density lipoprotein and triglyceride levels, coupled with diminished high-density lipoprotein levels (19).

The prevalence of dyslipidemia in PCOS is substantial, encompassing diverse types and degrees of lipid abnormalities, affecting approximately 70% of PCOS patients, as recommended by the National Cholesterol Education Program (19-20). Ethnicity emerges as a noteworthy factor, introducing genetic and

behavioral influences contributing to the presence of dyslipidemia, as evidenced by variations observed among patients with the classic PCOS phenotype from different regions (20).

The multifactorial origins of dyslipidemia in PCOS involve insulin resistance playing a pivotal role. This insulin resistance is partially driven by the activation of lipolysis and changes in the expression of lipoprotein lipase and hepatic lipase (20).

The intricate interplay of hyperandrogenism and lipid metabolism in PCOS remains to be precisely defined, though clinical observations underscore the link between insulin resistance and dyslipidemia in women with PCOS (20-22).

Obesity, particularly the central distribution of adipose tissue, emerges as a contributing factor to dyslipidemia in PCOS. The increased waist-to-hip ratio and waist-to-height ratio, commonly used as proxies for central obesity, have gained attention as markers of early health risk. The boundary value of waist-to-height ratio, suggesting risk assessment, has been globally acknowledged, with findings supporting its efficacy in identifying health risks (20, 23-26).

Android obesity, characterized by an increased waist-to-hip ratio, is a significant risk factor for insulin resistance and other conditions that increase the likelihood of early cardiovascular disease. The study by Sumarac et al. confirmed the considerable effect of android obesity on insulin resistance in both women with PCOS and the control group, highlighting the heightened vulnerability of PCOS women to increased waist-to-hip ratio concerning insulin resistance development (27).

The elevated waist-to-hip ratio in PCOS women proves more sensitive to dyslipidemia, attributable to centrally located adipocytes exerting detrimental effects on blood lipids. Additionally, adipose tissue in central distribution secretes adipokines, contributing to inflammation in PCOS. The prevalence of circulating free fatty acids, particularly common in obese PCOS patients, further underscores the intricate relationship between dyslipidemia and obesity in PCOS, exacerbating endocrine, metabolic, and reproductive disturbances, including infertility (19, 28).

Our study identifies a significant association between elevated triglycerides and the occurrence of ectopic fat obesity, aligning with previous research findings (29-32).

Both excess weight and the central accumulation of fat are associated with a higher risk of anovulation and infertility in PCOS women (3). Body composition, rather than just body weight, is linked to the origins of obesity, as well as to metabolic and hormonal disorders (5, 33).

In prior investigations, researchers have demonstrated that triglycerides (TG) serve as a primary form for storing and transporting fatty acids within cells and the plasma (29). Bril F and colleagues recently established a connection between triglycerides and ectopic fat, highlighting the interactive roles of BMI in the association between TG and ectopic fat obesity (29, 34, 35).

The intricate interplay between anthropometric measures and lipid parameters is evident across various studies, with the waist-to-height ratio showing the most significant correlation with cholesterol among PCOS patients (11, 36). Some researchers have delved into anthropometric measurements, discovering the presence of android obesity concerning BMI and waist-to-hip ratio (37-38). Nevertheless, the full understanding of the potential interactions between endocrine disorders, metabolic abnormalities, and lipid profiles in women with PCOS remains an underexplored area. Further exploration of the independent and interconnected effects of obesity on circulating lipid profiles is imperative for unraveling the underlying mechanisms in PCOS (39).

Central obesity plays a pivotal role in the development and perpetuation of polycystic ovary syndrome (PCOS), influencing the extent of cardiovascular and metabolic risk profiles. Elevated fasting insulin levels accompany central obesity, with its prevalence being influenced by ethnic origin or cultural habits, particularly in PCOS women of South Asian descent (23). Infertile women with PCOS represent a subgroup exhibiting more pronounced manifestations of the syndrome (18, 40).

Our research underscores dyslipidemia profiles marked due to increased triglyceride levels in women with PCOS and higher BMI. The growing trend in triglyceride levels with BMI

suggests obesity-induced alterations in fatty acid metabolism, leading to ectopic fat obesity. Hypertrophic adipocytes in obesity release higher levels of free fatty acids into the bloodstream, promoting FFA uptake and lipid production in the live (41-42). This surge of FFAs to the liver culminates in hepatic triglyceride accumulation, coupled with macrophage infiltration and a shift to a pro-inflammatory phenotype, contributing to hypertriglyceridemia (39, 43-44).

The association between dyslipidemia and central fat in obese PCOS patients underscores the adverse effects of adipocyte location on plasma lipids. Intra-abdominal visceral fat accumulation is implicated in lipid metabolism disorders, further linking to insulin resistance (45). Moreover, obese PCOS patients exhibit a significantly lower suppression of FFAs than healthy controls, indicating increased recycling fatty acid lipolysis (46). Heightened lipolysis in turn compromises insulin action in adipose tissue. High expression of nicotinamide adenine nucleotide phosphate oxidase, stimulated by FFAs, and subsequent reactive oxygen species generation may promote insulin resistance in obese women with PCOS (19).

Our findings align with prior research indicating that imbalances in glucose and insulin homeostasis in young PCOS patients are influenced by BMI, reinforcing the connection between insulin resistance and obesity. Enhanced lipolysis in obese PCOS patients, coupled with increased nicotinamide adenine nucleotide phosphate oxidase expression, contributes to insulin resistance, shedding light on the intricate interactions influencing lipid profiles in PCOS. Different forms of dyslipidemia in PCOS are ascribed to the influence of hyperandrogenism, insulin resistance, and lifestyle factors commonly observed in these individuals. Discrepancies in dyslipidemia among PCOS women of diverse ethnic and geographical backgrounds are likely shaped by genetic and environmental factors, encompassing diet and activity levels (7, 48).

The meta-analysis and systematic review of metabolic syndrome in PCOS women underscore the correlation between the condition and age, with a noteworthy the occurrence of metabolic syndrome in women under the age of thirty. Abdominal obesity emerges as a significant component of metabolic syndrome in PCOS women (46,39). The diverse types of dyslipidemia observed in PCOS women are attributed to the interplay of hyperandrogenism, insulin resistance, and lifestyle factors, with variations across ethnic and geographical backgrounds likely influenced by genetic and environmental factors, including diet and activity level (9, 47-48).

This study has some strengths and limitations. It represents the pioneering analysis of anthropometric indices and lipid profiles in infertile PCOS patients. Notably, it boasts a substantial and appropriate sample size. Further, a multidisciplinary team comprising nutritionists, gynecologists, and radiologists collaborated in this research, ensuring comprehensive and reliable results.

One limitation of this study was the absence of Very Low-Density Lipoprotein (VLDL) data in the laboratory findings. Incorporating VLDL measurements in future research endeavors would enhance the comprehensiveness of the investigation. Furthermore, employing study designs with greater strength, such as Randomized Controlled Trials (RCTs) and cohort studies, rather than relying solely on cross-sectional approaches, would bolster the robustness of the investigation.

Conclusion

Women with PCOS, displaying diverse characteristics, exhibited distinct lipid profiles. The observed relationship between anthropometric indices and lipid parameters suggests that central obesity may be associated with adverse lipid profiles in this population. Regular monitoring of blood lipid levels and management of central obesity may help identify women at greater risk of long-term complications associated with dyslipidemia. Further studies are needed to identify subgroups of women with PCOS who may benefit from lipid-lowering interventions and to evaluate the effectiveness of such strategies.

Declerations

Acknowledgments

None.

Conflicts of interest

The authors declared no conflicts of interest.

Ethical considerations

The present study is a secondary cross-sectional analysis based on baseline data collected from participants prior to the initiation of the intervention. Before enrollment in the original study, all participants received detailed information about the study procedures and provided written informed consent.

Code of Ethics

Approval for the original clinical trial was obtained from the Ethics Committee of Mashhad University of Medical Sciences (Approval ID: 4020097; IR.MUMS.REC.1402.191). The trial was also registered in the Iranian Registry of Clinical Trials (IRCT20230712058752N1).

Use of Artificial Intelligence (AI)

The authors used artificial intelligence (AI)-assisted tools solely for language editing and improving the readability of the manuscript. All scientific content, study design, data analysis, interpretation of results, and final manuscript revisions were performed and verified by the authors, who take full responsibility for the content of this article.

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Authors' contribution

NKh and MN conceived and designed the study and supervised the research process. FR and FM were responsible for data collection, data analysis, and manuscript preparation. All authors contributed to the interpretation of the data, critically revised the manuscript, and approved the final version for publication.

References

1. Kicińska AM, Maksym RB, Zabielska-Kaczorowska MA, Stachowska A, Babińska A. Immunological and Metabolic Causes of Infertility in Polycystic Ovary Syndrome. Biomedicines. 2023; 11(6).
2. Haddad-Filho H, Tosatti JAG, Vale FM, Gomes KB, Reis FM. Updates in diagnosing polycystic ovary syndrome-related infertility. Expert Review of Molecular Diagnostics. 2023; 23(2): 123-132.
3. Sánchez-Ferrer ML, Arense-Gonzalo JJ, Prieto-Sánchez MT, Gómez-Carrascosa I, Hernández-Peñalver AI, Mendiola J, et al. Anthropometric characteristics of polycystic ovary syndrome and their associations with insulin resistance and lipid profile. Applied Sciences. 2021; 11(12): 5395.
4. Zaadstra BM, Seidell JC, Van Noord P, te Velde ER, Habbema J, Vrieswijk B, et al. Fat and female fecundity: prospective study of effect of body fat distribution on conception rates. BMJ: British Medical Journal. 1993; 306(6876): 484.
5. Luke B. Adverse effects of female obesity and interaction with race on reproductive potential. Fertility and Sterility. 2017; 107(4): 868-877.
6. Scicchitano P, Dentamaro I, Carbonara R, Bulzis G, Dachille A, Caputo P, et al. Cardiovascular risk in women with PCOS. International Journal of Endocrinology and Metabolism. 2012; 10(4): 611.
7. Kiranmayee D, Kavya K, Himabindu Y, Sriharibabu M, Madhuri GLJ, Venu S. Correlations between anthropometry and lipid profile in women with PCOS. Journal of Human Reproductive Sciences. 2017; 10(3): 167.
8. Abbasi M. Nanoparticles as a promising innovative treatment towards infertility. Journal of Infertility and Reproductive Biology. 2017; 5(1): 1-4.
9. Emokpae MA, Eromosele CO, Ajibade HO. Alterations in Lipid Profile and Indices of Atherogenicity among Infertile Women Seeking Conception by Assisted Reproductive Technology. Journal of Infertility and Reproductive Biology. 2020; 8(3): 61-65.
10. Kiranmayee D, Kavya K, Himabindu Y, Sriharibabu M, Madhuri GLJ, Venu S. Correlations Between Anthropometry and Lipid Profile in Women With PCOS. Journal of Human Reproductive Sciences. 2017; 10(3): 167-172.
11. Saghafi-Asl M, Pirouzpanah S, Ebrahimi-Mameghani M, Asghari-Jafarabadi M, Aliashrafi S, Sadein B. Lipid profile in relation to anthropometric indices and insulin resistance in overweight women with polycystic ovary syndrome. Health Promotion Perspectives. 2013; 3(2): 206.
12. Kar S. Anthropometric, clinical, and metabolic comparisons of the four Rotterdam PCOS phenotypes: A prospective study of PCOS women. Journal of Human Reproductive Sciences. 2013; 6(3): 194.
13. Smet ME, McLennan A. Rotterdam criteria, the end. Australasian Journal of Ultrasound in Medicine. 2018; 21(2): 59-60.
14. Casadei K, Kiel J. Anthropometric measurement. 2019.

15. Żurawiecka M, Wronka I. Association between age at menarche and body mass index, waist circumference, waist to hip ratio, and waist to height ratio in adult women. American Journal of Human Biology. 2021; 33(6): e23523.
16. Ashwell M, Gunn P, Gibson S. Waist-to-height ratio is a better screening tool than waist circumference and BMI for adult cardiometabolic risk factors: systematic review and meta-analysis. Obesity Reviews. 2012; 13(3): 275-286.
17. Ng NYH, Jiang G, Cheung LP, Zhang Y, Tam CHT, Luk AOY, et al. Progression of glucose intolerance and cardiometabolic risk factors over a decade in Chinese women with polycystic ovary syndrome: A case-control study. PLoS Medicine. 2019; 16(10): e1002953.
18. Ishrat S, Hussain M. Prevalence of Insulin Resistance, Dyslipidemia and Metabolic Syndrome in Infertile Women with Polycystic Ovary Syndrome. Journal of Bangladesh College of Physicians & Surgeons. 2021; 39(4).
19. Liu Q, Xie Y-j, Qu L-h, Zhang M-x, Mo Z-c. Dyslipidemia involvement in the development of polycystic ovary syndrome. Taiwanese Journal of Obstetrics and Gynecology. 2019; 58(4): 447-453.
20. Amadi CF, Okolonkwo BN, George-Oparati MI, Odiabara KK. Understanding the Relationship between Polycystic Ovarian Syndrome (PCOS) and Dyslipidemia. Asian Journal of Biochemistry, Genetics and Molecular Biology. 2023; 15(3): 1-11.
21. Legro RS, Kunselman AR, Dunaif A. Prevalence and predictors of dyslipidemia in women with polycystic ovary syndrome. The American Journal of Medicine. 2001; 111(8): 607-613.
22. Whitsel EA, Boyko EJ, Matsumoto AM, Anawalt BD, Siscovick DS. Intramuscular testosterone esters and plasma lipids in hypogonadal men: a meta-analysis. The American Journal of Medicine. 2001; 111(4): 261-269.
23. Yildirim B, Sabir N, Kaleli B. Relation of intra-abdominal fat distribution to metabolic disorders in nonobese patients with polycystic ovary syndrome. Fertility and Sterility. 2003; 79(6): 1358-1364.
24. Sahakyan KR, Somers VK, Rodriguez-Escudero JP, Hodge DO, Carter RE, Sochor O, et al. Normal-weight central obesity: implications for total and cardiovascular mortality. Annals of Internal Medicine. 2015; 163(11): 827-835.
25. Ashwell M, Gibson S. A proposal for a primary screening tool:Keep your waist circumference to less than half your height'. BMC Medicine. 2014; 12: 1-6.
26. Ashwell M, Gibson S. Waist-to-height ratio as an indicator of 'early health risk': simpler and more predictive than using a 'matrix' based on BMI and waist circumference. BMJ Open. 2016; 6(3): e010159.
27. Šumarac-Dumanović M, Stamenković-Pejković D, Jeremić D, Dumanović J, Mandić-Marković V, Žarković M, et al. Age, body mass index, and waist-to-hip ratio related changes in insulin secretion and insulin sensitivity in women with polycystic ovary syndrome: minimal model analyses. International Journal of Endocrinology. 2022; 2022.
28. Spritzer PM, Lecke SB, Satler F, Morsch DM. Adipose tissue dysfunction, adipokines, and low-grade chronic inflammation in polycystic ovary syndrome. Reproduction. 2015; 149(5): R219-R227.
29. Zou Y, Sheng G, Yu M, Xie G. The association between triglycerides and ectopic fat obesity: An inverted U-shaped curve. PLoS One. 2020; 15(11): e0243068.
30. Graner M, Siren R, Nyman K, Lundbom J, Hakkarainen A, Pentikäinen MO, et al. Cardiac steatosis associates with visceral obesity in nondiabetic obese men. The Journal of Clinical Endocrinology & Metabolism. 2013; 98(3): 1189-1197.
31. Peng K, Mo Z, Tian G. Serum lipid abnormalities and nonalcoholic fatty liver disease in adult males. The American Journal of The Medical Sciences. 2017; 353(3): 236-241.
32. Amor AJ, Perea V. Dyslipidemia in nonalcoholic fatty liver disease. Current Opinion in Endocrinology, Diabetes and Obesity. 2019; 26(2): 103-108.
33. Broughton DE, Moley KH. Obesity and female infertility: potential mediators of obesity's impact. Fertility and Sterility. 2017; 107(4): 840-847.
34. Alves-Bezerra M, Cohen DE. Triglyceride metabolism in the liver. Comprehensive Physiology. 2017; 8(1): 1.
35. Bril F, Barb D, Portillo-Sanchez P, Biernacki D, Lomonaco R, Suman A, et al. Metabolic and histological implications of intrahepatic triglyceride content in nonalcoholic fatty liver disease. Hepatology. 2017; 65(4): 1132-1144.
36. Sousa RMLd, Chein MBdC, Silva DSMd, Dutra MB, Navarro PAdAS, Figueiredo Neto JAd, et al. Metabolic profile in women of different body mass indices with polycystic ovary syndrome. Revista Brasileira de Ginecologia e Obstetrícia. 2013; 35: 413-420.
37. Rasool SO. Anthropometric measurements in PCOS & Non PCO Infertile patients. International journal of chemical,

environmental and biological sciences. 2015; 3: 14-16.
38. Shah AK, Sarin M, Karunanand B, Mohapatra S, Bhat SA. Association of hormonal status with anthropometric & biochemical parameters in women with polycystic ovarian syndrome. Hypertension. 2017; 3(4): 5.
39. Guo F, Gong Z, Fernando T, Zhang L, Zhu X, Shi Y. The lipid profiles in different characteristics of women with PCOS and the interaction between dyslipidemia and metabolic disorder states: a retrospective study in Chinese population. Frontiers in Endocrinology. 2022; 13: 892125.
40. Studen KB, Pfeifer M. Cardiometabolic risk in polycystic ovary syndrome. Endocrine Connections. 2018; 7(7): R238-R251.
41. Ollila M-ME, Piltonen T, Puukka K, Ruokonen A, Järvelin M-R, Tapanainen JS, et al. Weight gain and dyslipidemia in early adulthood associate with polycystic ovary syndrome: prospective cohort study. The Journal of Clinical Endocrinology & Metabolism. 2016; 101(2): 739-747.
42. Klop B, Elte JWF, Castro Cabezas M. Dyslipidemia in obesity: mechanisms and potential targets. Nutrients. 2013; 5(4): 1218-1240.
43. Su X, Peng D. The exchangeable apolipoproteins in lipid metabolism and obesity. Clinica Chimica Acta. 2020; 503: 128-135.
44. Funcke J-B, Scherer PE. Beyond adiponectin and leptin: adipose tissue-derived mediators of inter-organ communication. Journal of Lipid Research. 2019; 60(10): 1648-1697.
45. Abruzzese GA, Cerrrone GE, Gamez JM, Graffigna MN, Belli S, Lioy G, et al. Lipid accumulation product (LAP) and visceral adiposity index (VAI) as markers of insulin resistance and metabolic associated disturbances in young argentine women with polycystic ovary syndrome. Hormone and Metabolic Research. 2017; 49(01): 23-29.
46. Cree-Green M, Bergman BC, Coe GV, Newnes L, Baumgartner AD, Bacon S, et al. Hepatic steatosis is common in adolescents with obesity and PCOS and relates to de novo lipogenesis but not insulin resistance. Obesity. 2016; 24(11): 2399-2406.
47. Rocha MP, Marcondes JA, Barcellos CR, Hayashida SA, Curi DD, da Fonseca ÂM, et al. Dyslipidemia in women with polycystic ovary syndrome: incidence, pattern and predictors. Gynecological Endocrinology. 2011; 27(10): 814-819.
48. Costa EC, Ferezini de Sa JC, Mafaldo Soares EM, Araujo Moura Lemos TM, de Oliveira Maranhão TM, Dantas Azevedo G. Anthropometric indices of central obesity how discriminators of metabolic syndrome in Brazilian women with polycystic ovary syndrome. Gynecological Endocrinology. 2012; 28(1): 12-15.