(International Print/Online Journal)

SJIF IMPACT FACTOR: 5.565
PUBMED-National Library of Medicine ID-101739732

ISSN (Print): 2209-2870
ISSN (Online): 2209-2862

International Journal of Medical Science and Current Research (IJMSCR)
Available online at: www.ijmscr.com
Volume 6, Issue 5 , Page No: 442-447
September-October 2023

IJMSCR

A Cross Sectional Study of Lipid Profile in Rural and Urban Population of Udaipur with Polycystic Ovarian Syndrome

Babita Garhwal[1], Ashok.L. Bajentri[1], Varsha Butolia[1], Sumitra Kumari[1]
[1]Department of Physiology, Pacific Institute of Medical Sciences,
Sai Tirupati University, Udaipur, Rajasthan, India

***Corresponding Author:**
Babita Garhwal
Ph.D. Scholar, Department of Physiology, Pacific Institute of Medical Sciences,
Sai Tirupati University, Udaipur, Rajasthan, India

Type of Publication: Original Research Paper
Conflicts of Interest: Nil

Abstract

Background: The name polycystic ovary syndrome (PCOS) describes the numerous small cysts (fluid-filled sacs) that form in the ovaries; however, some women with this disorder do not have cysts, while some women without the disorder do develop cysts. Ovulation occurs when a mature egg is released from an ovary.

Methods: By employing a standard approach and a few carefully chosen biochemical measures, this research aims to analyze the significance of lipid profiles in polycystic ovarian syndrome in rural and urban populations and their implications in the development of the condition.

Results: The present study showed that the value of Lipid Profile were high in polycystic ovarian syndrome of urban patients compared to rural patients.

Conclusions: Our study also shows that urban PCOS Patients have a high risk of critical condition and severity compare to rural PCOS Patients.

Keywords: PCOS,TC,TG,HDL,LDL

Introduction

The primary cause of female infertility is polycystic ovarian syndrome, which is the most prevalent endocrine problem and metabolic disorder in adolescence and reproductive women. [1]

In India, 76.9% of women of reproductive age who have polycystic ovarian syndrome also have insulin resistance. [2]. The average presentation age is 24.8, with ages ranging from 17 to 36.Insulin resistance is the inability of a known amount of exogenous or endogenous insulin to increase glucose absorption and utilization in a person, as it does in a normal person. [1]

A coincidental genetic defect at the level of the ovary or pancreas determines whether polycystic ovarian syndrome and type 2 diabetes mellitus are different clinical manifestations of the same insulin resistance syndrome. As a result, having one increases the risk of having the other develop. The metabolic syndrome, also known as SYNDROME X, is the combination of high blood pressure, glucose intolerance, obesity, lipid abnormalities, and coronary artery disease. Insulin resistance is a pathogenic component for this connection. [3]

Three primary clinical causes are given to women with polycystic ovarian syndrome. Menstrual abnormalities (mean incidence: 70%), infertility (mean incidence: 40%), and androgen excess (mean incidence: 70% hirsuitism, 15% to 30% acne) are all conditions caused by an excess of androgen.[4] .

In a group of patients who presented with amenorrhea, bilateral polycystic ovaries, and hirsutism change in 1935, Irving Stein and Michael

Leventhal initially characterized polycystic ovarian syndrome. At first, they believed that harmonal variation connected to the pituitary was to blame. [5]

Low levels of follicle-stimulating hormone and high levels of luteinizing hormone are characteristics of PCOS. Anovulation is caused by low FSH levels, hyperandrogenism is caused by excessive LH levels, and insulin resistance symptoms include everything from simple cystic acne to cases of oligomenorrhoea or amenorrhoea, sterility, and severe generalized hirsutism.

Originally, in 1935 according to Stein and Leventhal, the diagnosis required classic ovarian findings and the clinical triad of amenorrhea, hirsutism and obesity. 30 years later, next diagnostic milestone was established when researchers in the late 1960s and early 1970s found that derangements in the hypothalamo-pituitary axis was a reason for polycystic ovarian syndrome. Following this the endocrine criteria were added to the diagnosis such as elevated levels of serum testosterone, and an elevated LH: FSH ratio.[4]

The discovery of pelvic ultrasonography enhanced diagnostic specificity to the identification of PCOS further in the 1970s. Following the Rotterdam Consensus meeting, ultrasound diagnostic criteria were developed in 2003[6].

According to the 2015 recommendations of the American Association of Clinical Endocrinologists (AACE) and the Androgen Excess and PCOS Society (AES), PCOS must be diagnosed when at least two of the following three criteria are met:

i) Chronic ovarian failure

ii) Hyperandrogenism

iii) Polycystic ovaries without evidence of any further causes The following factors are used to define polycystic ovaries: Increased ovarian volume (>10cc) and 12 or more tiny follicles (2 to 9mm) over the entire ovary.The polycystic ovary is significantly defined by its appearance in one ovary.[7]

Although the fundamental cause of polycystic ovaries is unknown, it is becoming increasingly clear that excessive androgen, insulin resistance, and aberrant gonadotropin dynamics are its prominent symptoms. A connection between persistent stress circumstances and various hormone abnormalities is also becoming more and more clear. A condition of an excessive physiological reaction to androgens is polycystic ovarian syndrome. The most significant androgen in circulation is testosterone.

In between 70 and 75 percent of women with polycystic ovarian syndrome, DHEA levels are also increased. Ovarian volume and ovaries with a polycystic look were found to positively correlate with DHEA, androstenedione, and testosterone levels in a study of adolescent women with PCOS, supporting the idea that hormone dysregulation may be a significant contributing cause. The most common symptoms were oligomenorrhea and significant hyperandrogenism without hyperinsulinemia in adolescents with PCOS.[8,9,10]

Despite having more beta cells that produce insulin, polycystic ovarian syndrome individuals have relatively inefficient insulin receptors that bind to insulin, which results in incorrect glucose transport to intracellular compartments and relative hyperglycemia. The number of GLUT4 glucose transporters is significantly reduced in adipocytes from patients with polycystic ovarian syndrome. [11]

Hepatic lipase levels rise as a result of hyperandrogenism. In polycystic ovarian syndrome, the liver secretes more VLDL particles, which raises the level of TG.[12]

Measures of obesity like BMI and the waist to hip ratio are not related to insulin resistance and the accompanying dyslipidemia. 20% of polycystic ovarian syndrome reproductive age women have abnormal OGTT results overall. Screening IR in this particular group is probably advantageous to provide.[13]

The two most significant determining variables of polycystic ovarian syndrome are hereditary and environmental factors.[14] Early sexual maturation, early fetal development, and a family history of PCOS among first-degree relatives are some of the genetic variables that have been implicated [14, 15]. This has been linked to a rise in androgen secretion brought on by early puberty [14]. Premature prenatal development has been linked to an earlier and faster start to puberty as well as a higher risk of developing PCOS [16].

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Reactive oxygen species (ROS) are produced in excess compared to how quickly the body's defense mechanisms can neutralize them, which leads to oxidative stress. When the synthesis of ROS and other radical species surpasses the scavenging capacity of antioxidants due to excessive production of ROS and/or inadequate intakes or increased use of antioxidants, oxidative stress is present. One of the stable end products of lipid peroxidation that can act as a biomarker of oxidative stress is malondialdehyde (MDA). Reactive oxygen species are continuously created in live cells as a result of both metabolic processes and outside influences. The etiology of inflammatory disorders is significantly influenced by an increase in free radicals, and inflammation may be a major factor in atherosclerosis. Active oxygen generated radicals and oxidative damage have a well-established function in the pathogenesis of myocardial ischemia, and mounting evidence points to their involvement in the atherogenesis process as well.[17]

Materials And Methods

Study Design: This observational analytical study was carried out to assess and compare physiological and biochemical parameters in the PCOS women population in urban and rural areas.

Source of Data: The information was gathered from patients who visited the Obstetrics & Gynecology and Biochemistry outpatient departments over a three-year period at the Pacific Institute of Medical Sciences, Udaipur.

After receiving informed consent, the study was conducted on PCOS cases that had been clinically verified. The study comprised 360 patients with clinically confirmed PCOS who were between the ages of 18 and 40 and who visited the Obstetrics & Gynecology OPD at PIMS Hospital. According to the inclusion and exclusion criteria, the study recruited 180 women who met the criteria for PCOS in urban areas (Rotterdam 2004) and 180 women from rural areas who were age-matched controls with regular menstrual cycles (28-35 d) and normal ovaries from the same geographic area.

Participant size is furthers divided into following groups:

- **Group 1:** .Consists of 180 known PCOS from urban cases.
- **Group 2**. Consists of 180 known PCOS from rural cases

Inclusion Criteria

1. Patient who are willing to participate.
2. Radiologically (ultrasonography) confirmed multiple small cysts in ovary.
3. Biochemical parameters confirmed PCOS.
4. Irregular Menses, hirsutism, acne and oligomenorrhea.

Exclusion Criteria

1. Patients below 18 years and above 45 years of age will be excluded in the study.
2. Other endocrinal disorders like diabetes mellitus, untreated hypothyroidism
3. Patients those on drug treatment like antihypertensive, antiepileptic, lipid lowering agents, drug affecting glucose
4. Vitamin D drugs
5. Androgen-secreting tumour
6. Impaired glucose tolerance
7. Pregnancy
8. breast feeding

Methodology: Symptoms (irregular periods, excessive pain during periods accompanied with heavy flow,), serum Lipid Profile were recorded by using Autoanalyzer XL-640.

Statistical Analysis: For the quantitative analysis, we used the software SPSS software.

Result

Table 1. Comparison of Lipid Profile between Rural PCOS Patients and Urban PCOS Patients

S. No	Test	Rural Pcos Patients	Urban Pcos Patients	P Value

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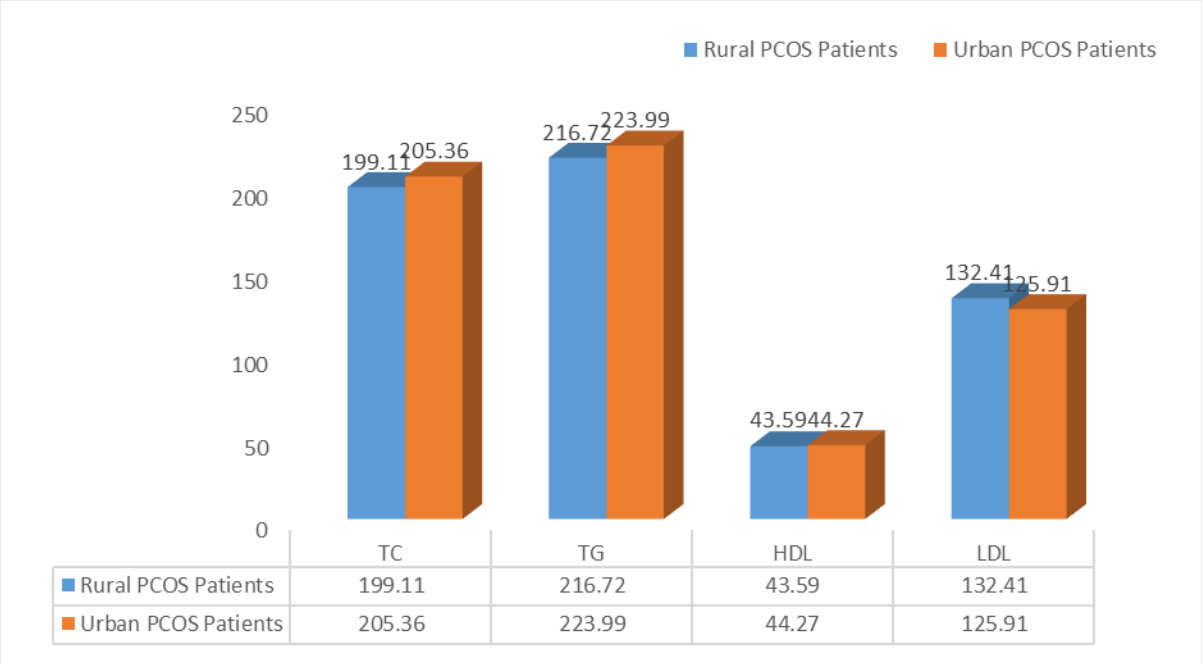
		Mean	SD ±	Mean	SD ±	
1	TC	199.11	30.63	205.36	37.61	P= 0.08
2	TG	216.72	42.81	223.99	56.45	P= 0.16
3	HDL	43.59	5.06	44.27	7.47	P=0.31
4	LDL	132.41	27.79	125.91	26.92	P=0.02

Fig 1. Comparison of Lipid Profile between Rural PCOS Patients and Urban PCOS Patients



The present study showed that the Mean and Standard deviation of Total Cholesterol (TC), Low density Lipoprotein (LDL), Triglyceride (TG), And HDL high in polycystic ovarian syndrome patients in urban population compare to rural population with polycystic ovarian syndrome disease patients. (Show in table 1 and figure 1)

Discussion

Serum Triglycerides: The mean serum concentration of triglycerides in rural and urban PCOD cases was noted as 216.72±42.81 and 223.99±56.45 respectively. In present study we found that the mean serum levels of triglycerides are significantly increased in subjects with PCOS, however there was no significant difference in between them (p = 0.84). It is in accordance to the studies by Xian S et al ,Kalra A et al ,Valkenburno O et al and Mandrelle K et al

Serum Total cholesterol: The mean serum concentration of cholesterol in rural and urban PCOD cases was noted as 199.11±30.63 and 205.36±37.61 respectively. In present study we found that the mean serum levels of triglycerides are significantly increased in subjects with PCOS, however there was no significant difference in between them (p = 0.169). It is in accordance to the studies by Jayashree R et al,Ioan C et al ,Liu N et al and Mandrella K et al

Serum HDL cholesterol: The mean serum concentration of HDL in rural and urban PCOD cases was noted as 43.59±5.06 and 44.27±7.47respectively. In present study we found that the mean serum levels of triglycerides are significantly increased in subjects with PCOS, however there was no significant difference in between them (p = 0.314). It is in accordance to the studies by Valkenburn O et al ,Wonwananuruk et al ,Saxena P et al and Ioan C et al .

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Serum LDL cholesterol: The mean serum concentration of LDL in rural and urban PCOD cases was noted as 132.41±27.79 and 125.91±26.92 respectively. In present study we found that the mean serum levels of triglycerides are significantly increased in subjects with PCOS, however there was no significant difference in between them (p = 0.024). It is in accordance to the studies by Mutib M et al , Liu N et al , Mandrella K et al and Sanghafiasl Met al

Both insulin resistance and hyperandrogenemia contribute to dyslipidemia in PCOS. Testosterone decreases lipoprotein lipase activity in abdominal fat cells, and insulin resistance impairs the ability of insulin to exerts its antilipolytic effects by altering expression of lipoprotein lipase and hepatic lipase.

Insulin plays a key role in TG metabolism as it normally reduces availability of large TGRLs particles, synthesized by a distinct pathway compared with smaller VLDL particles. In insulin resistant subjects, insulin fails to suppress synthesis of large VLDL particles. In addition, insulin resistance is associated with increased flow of free fatty acid to liver, increased lipid synthesis in the liver, and decreased clearance of VLDL particles, all of which increase VLDL concentrations in plasma. Increased secretion of very low density lipoprotein (VLDL) particles by the liver.

Conclusion

The present study done on rural and urban polycystic ovarian syndrome patient admitted in Pacific Institute of Medical Sciences, Umarda, Udaipur. Total 360 patients were included for this study .180 was rural patient and 180 was urban polycystic ovarian syndrome patient. 19-44 age group was taken for this study the study shows that the Mean and Standard deviation of Total Cholesterol (TC), Low density Lipoprotein (LDL), Triglyceride (TG), HDL high urban polycystic ovarian syndrome patients compare to rural patient. Our study also shows that population from urban polycystic ovarian syndrome patients have a high risk of critical condition and developing sever.

Ethical Issues: Research project approved by the ethics committee of Pacific Institute of Medical Sciences, Umarda Udaipur- 313005, Rajasthan, INDIA**.**

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