IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 24, Issue 6 Ser. 2 (June. 2025), PP 32-39
www.iosrjournals.org

PCOS And NAFLD- A Closely Bonded Entity Of Metabolic Syndrome

Vani Malhotra, Portia Sarkar, Parveen Malhotra, Shalini Agarwal, Meenakshi Chauhan,
Pushpa Dahiya
Department Of Obstetrics & Gynecology, Medical Gastroenterology & Radiology,PGIMS, Rohtak, Haryana.

Abstract
Introduction*- Polycystic ovary syndrome (PCOS), also referred as hyperandrogenic an-ovulation (HA), or Stein Leventhal syndrome, is one of the most common endocrine disorders of reproductive age and was first described by Stein and Leventhal in 1935. They are at an increased risk of multiple morbidities including obesity, type II diabetes mellitus (DM-II), cardiovascular diseases (CVD), infertility, cancer, and psychological disorders.*
Aims and objectives: *The aim of study was to assess the association of polycystic ovarian syndrome with Non-Alcoholic Fatty Liver Disease and objectives were to study the association of NAFLD in PCOS patients, association of NAFLD in NON- PCOS controls, to compare the association in both the groups and to assess the correlation between metabolic syndrome and fatty liver.*
Materials & Methods- *It was a prospective case control study conducted at Department of Obstetrics and Gynaecology in PGIMS, Rohtak. A total of 100 women in 20-40 yrs age group were recruited and divided into two groups of 50 each, one group was of women who presented with PCOS and other was age matched healthy controls.*
Conclusion: *This study was done to find the association of NAFLD with PCOS, and also with Metabolic Syndrome. Although, the presence of hepatic steatosis and consequently NAFLD wasn't found to be higher in PCOS group, liver fibroscan score was raised, marking significant hepatic fibrosis in them. NAFLD was found to be significantly associated with Metabolic Syndrome, indicating the need for early liver function assessment in these patients, to avoid long term complications and improve their quality of life.*
Keywords: *Polycystic ovarian syndrome, Non-alcoholic fatty liver disease, Fibroscan, Diabetes Mellitus, Waist circumference*

Date of Submission: 27-05-2025 Date of Acceptance: 07-06-2025

I. Introduction

Polycystic ovary syndrome (PCOS), also referred as hyperandrogenic an-ovulation (HA), or Stein Leventhal syndrome, is one of the most common endocrine disorders of reproductive age and was first described by Stein and Leventhal in 1935.[1] It is a heterogenous, multifactorial, complex genetic disorder, characterized by symptoms associated with menstrual dysfunction and androgen excess, significantly impacting their quality of life. They are at an increased risk of multiple morbidities including obesity, type II diabetes mellitus (DM-II), cardiovascular diseases (CVD), infertility, cancer, and psychological disorders.[2] Prevalence of PCOS is estimated to be 3%-26% worldwide. However, the prevalence of PCOS in India is reported as 3.7%-22.5% in adults, and 9.13%-36% in adolescents.[3,4] According to the Rotterdam Criteria, the diagnosis of PCOS may be made if any two out of the following three abnormalities are present: 1) chronic anovulation; 2) clinical and/or biochemical hyperandrogenism and 3) polycystic ovaries on pelvic ultrasound: a) one or both ovaries demonstrate 12 or more follicles measuring 2-9 mm in diameter or b) the ovarian volume exceeds 10 cubic cm.[5] Each set of diagnostic criteria of PCOS additionally requires exclusion of other causes of hyperandrogenism, such as thyroid dysfunctions, hyperprolactinemia, congenital adrenal hyperplasia, androgen secreting tumour and Cushing syndrome.[6] The principal underlying disorder is insulin resistance, with the resultant hyperinsulinemia stimulating excess ovarian androgen production.[7] Elevated levels of insulin causes abnormalities in hypothalamic-pituitary-ovarian axis (HPO axis) leading to PCOS. Hyperinsulinemia increases the gonadotropin-releasing hormone (GnRH) pulse frequency resulting in increased frequency of pituitary luteinizing hormone (LH) pulse over follicular stimulating hormone (FSH) which further causes an increase in LH stimulated androgen secretion.[8-11] PCOS patients have complaints regarding their menstrual irregularities, heavy menstrual bleeding, infertility, reduced efficacy of infertility treatment, recurrent

abortions, excessive growth of facial and body hair, acne, seborrhea, and obesity. All these features may impact the women's quality of life profoundly.[12] Metabolic syndrome (MetS) is a cluster of endocrine disturbances, including insulin resistance, dyslipidemia, obesity and hypertension. The prevalence of MetS is as high as 33% in women with PCOS, and is associated with cardiovascular diseases (CVD), diabetes type II, cancers, sleep apnea and psychological problems. Hyperinsulinemia, and the increased responsiveness of the ovarian theca cells to insulin, causes an increase in the levels of free androgens causing hyperandrogenemia which increases a person's predilection for central obesity and worsens insulin resistance and dyslipidaemia.[13] It most likely results from combined effect of genetic predisposition, poor diet habits and sedentary life style, thus compounding pre-existing metabolic derangements.[14] Various diagnostic criteria for Metabolic syndrome have been proposed based on the basic five parameters by different groups which include elevated waist circumference ≥88 cm or 35 inches, elevated triglycerides ≥150 mg/dl, reduced HDL <50 mg/dl, elevated BP - Systolic BP ≥130 or diastolic BP ≥ 85 mmHg or treatment of previously diagnosed hypertension and elevated fasting glucose levels ≥100 mg/dl. According to most commonly used criteria- National Cholesterol Education Programme Adult Treatment Panel III (NCEP: ATP III) any 3 of the above criteria are used to diagnose Metabolic syndrome. The New International Diabetic Federation (IDF), most recent criteria take waist circumference as the integral component along with any two of the four.[15] Non-alcoholic fatty liver disease (NAFLD) is a clinical pathology spanning from simple steatosis to nonalcoholic steatohepatitis (NASH) with or without cirrhosis, which may progress to liver failure and, in some cases, to hepatocellular carcinoma. It is now becoming clear that NAFLD is also a pathogenic determinant of the MetS.[16] NAFLD is histologically defined by the presence of >5% hepatic steatosis. Ultrasonography remains the recommended first line imaging modality for diagnosing hepatic steatosis in clinical practice.[18] Although existing data of NAFLD in women with PCOS is very scarce, hence the present study was conducted to study the association of PCOS with NAFLD.

II. Aim & Objectives-

The aim of study was to assess the association of polycystic ovarian syndrome with Non-Alcoholic Fatty Liver Disease and objectives were to study the association of NAFLD in PCOS patients, association of NAFLD in NON- PCOS controls, to compare the association in both the groups and to assess the correlation between metabolic syndrome and fatty liver**.**

III. Material & Methods-

It was a prospective case control study conducted at Department of Obstetrics and Gynaecology in PGIMS, ROHTAK. A total of 100 women in 20-40 yrs age group were recruited and divided into two groups of 50 each. One group was of women who presented with PCOS and other was age matched healthy controls. The inclusion criterion was women diagnosed to have PCOS based on ROTTERDAM criterion and exclusion criterion were hyperprolactinemia, thyroid disease, known case of liver disease, patient on metformin / hormonal contraceptives for at least three months, alcohol intake and androgen producing tumors. After taking informed written consent, and explaining the nature of study to the participants, detailed history, general physical and systemic examination was carried out. History was taken specially pertaining to recent increase in weight, menstrual history, dietary habits, physical activity, hirsutism, acne. On examination, hirsutism was graded according to Ferriman Gallway Score ≥8. Waist circumference and BMI was calculated. All the cases and control were subjected to laboratory investigations like complete hemogram, fasting blood sugar, complete lipid profile, serum AST, serum ALT, liver functions tests, renal function test, thyroid profile, viral markers (HBsAg, HIV, IgM HAV & HEV, anti-HCV antibody).All the cases and controls were screened for fatty liver disease based on biochemical and radiological features. The biochemical indicators for assessing fatty liver is defined as an elevation of AST and ALT>35-40 U/L. All the subjects were subjected to sonography of the liver with 3-5mhz convex transducer. Liver parenchyma was evaluated for fatty liver.

Statistical Methods- At the end of study, all the data was compiled and descriptive statistics was analyzed with SPSS software. The clinical characteristics and laboratory measurements of patients with PCOS and subjects of the control group was analyzed. Continuous variables were presented as means ± standard deviation (SD) and dichotomic variables were presented as percentage. Univariate regression analysis was done to evaluate the effect of age, BMI, waist circumference, lipid parameter on hepatic steatosis. Multivariate regression analysis was performed for each independent variables significantly related to hepatic steatosis by univariate regression analysis. A p value of <0.05 was considered statistically significant**.**

Observations- The study was conducted in Department of Obstetrics and Gynaecology in PGIMS, Rohtak. 50 PCOD women and 50 non PCOD women were included in the study. NAFLD, metabolic syndrome and fatty liver were assessed and results are as follows.

Table 1: -Comparison of age(years) between PCOD and non-PCOD group.

Age(years)	COD group(n=50)	Non PCOD group(n=50)	Total	P value
20 to 30 years	41 (82%)	31 (62%)	72 (72%)	**0.026**†
31 to 40 years	9 (18%)	19 (38%)	28 (28%)	
Mean ± SD	25.08 ± 4.21	28.78 ± 4.79	26.93 ± 4.86	**<0.0001**‡
Median (25th-75th percentile)	24 (22-27)	28 (25.25-32)	26 (23-31)	
Range	20-36	20-38	20-38	

‡ **Independent t test,** † **Chi square test**

The proportion of patients aged 20 to 30 years was significantly higher in the polycystic ovary syndrome (PCOD) group compared to the non-PCOD group, with 82% versus 62%, respectively. Conversely, the proportion of patients aged 31 to 40 years was significantly lower in the PCOD group compared to the non-PCOD group, with 18% versus 38%, respectively (p-value=0.026). The distribution of various chief complaints varied significantly between the PCOD and non-PCOD groups. In the PCOD group (n=50), the most common complaint was abnormal uterine bleeding (AUB), reported by 60% of patients, which was significantly higher compared to 22% in the non-PCOD group (p-value=0.0001). Pain abdomen was reported by 28% of patients in the non-PCOD group, significantly higher compared to the PCOD group (p-value <.0001). Other chief complaints were reported by 12% of patients in the non-PCOD group, significantly higher compared to the PCOD group (p-value =0.027). Additionally, hirsutism was present in 12% of patients in the PCOD group, while none were reported in the non-PCOD group (p-value=0.027). Secondary amenorrhea was observed in 18% of patients in the PCOD group compared to 2% in the non- PCOD group (p-value=0.016)

Table 2- Comparison of Obstetric history between PCOD and non-PCOD group

Obstetric history	PCOD group(n=50)	Non PCOD group(n=50)	Total	P value
P0	32 (64%)	16 (32%)	48 (48%)	**0.02***
P1	9 (18%)	14 (28%)	23 (23%)	
P2	8 (16%)	13 (26%)	21 (21%)	
P3	1 (2%)	5 (10%)	6 (6%)	
P4	0 (0%)	1 (2%)	1 (1%)	
P5	0 (0%)	1 (2%)	1 (1%)	
TOTAL	50 (100%)	50 (100%)	100 (100%)	

Table 3: -Comparison of chief complaints between PCOD and non-PCOD group.

Chief complaints	PCOD group(n=50)	Non PCOD group(n=50)	Total	P value
Vaginal discharge	0 (0%)	10 (20%)	10 (10%)	**0.001***
Pain abdomen	0 (0%)	14 (28%)	14 (14%)	**<.0001***
Primary infertility	4 (8%)	5 (10%)	9 (9%)	1*
AUB	30 (60%)	11 (22%)	41 (41%)	**0.0001**†
Secondary infertility	0 (0%)	1 (2%)	1 (1%)	1*
Dysmenorrhea	1 (2%)	2 (4%)	3 (3%)	1*
Acne	2 (4%)	0 (0%)	2 (2%)	0.495*
Hirsutism	6 (12%)	0 (0%)	6 (6%)	**0.027***
Secondary amenorrhea	9 (18%)	1 (2%)	10 (10%)	**0.016***
Weight gain	3 (6%)	0 (0%)	3 (3%)	0.242*
Others	0 (0%)	6 (12%)	6 (6%)	**0.027***

* **Fisher's exact test,** † **Chi square test**

The proportion of patients with body mass index (BMI) falling within the overweight category (25 to 29.99 kg/m²) was significantly higher in the PCOS group compared to the non-PCOS group (32% vs. 12%). Conversely, the PCOS group exhibited significantly lower proportions of patients categorized as underweight (<18.5 kg/m²) and with a normal BMI (18.5 to 24.99 kg/m²) compared to the non-PCOS group (underweight: 0% vs. 2%, normal BMI: 68% vs. 86%). This difference was statistically significant with a p-value of 0.028. There were significant differences were noted in BMI and hip circumference between the two groups (p value < 0.05).

Table 4: -Comparison of anthropometric parameters between PCOD and non- PCOD group.

Anthropometric parameters	PCOD group(n=50)	Non PCOD group(n=50)	Total	P value
Body mass index(kg/m²)				
<18.5 kg/m² {Underweight}	0 (0%)	1 (2%)	1 (1%)	**0.028***
18.5 to 24.99 kg/m² {Normal BMI}	34 (68%)	43 (86%)	77 (77%)	
25 to 29.99 kg/m² {Overweight}	16 (32%)	6 (12%)	22 (22%)	
Mean ± SD	23.93 ± 1.96	22.94 ± 2.03	23.44 ± 2.05	
Median (25th-75th percentile)	23.7(22.4-25.5)	23.2(21.375-24.3)	23.5(22.075-24.8)	**0.015†**
Range	20-28	18.3-26.7	18.3-28	
Waist circumference(cm)				
Mean ± SD	32.66 ± 2.49	31.78 ± 3.07	32.22 ± 2.81	0.118†
Median (25th-75th percentile)	33(31.25-34)	32(30-34)	32.5(30-34)	
Range	28-42	24-38	24-42	
Hip circumference(cm)				
Mean ± SD	40.76 ± 3.53	39.06 ± 4.73	39.91 ± 4.24	**0.045†**
Median (25th-75th percentile)	40(38-43)	40(36-42)	40(37-43)	
Range	35-48	30-47	30-48	
Waist hip ratio				
Mean ± SD	0.81 ± 0.07	0.83 ± 0.11	0.82 ± 0.09	
Median (25th-75th percentile)	0.8(0.765-0.837)	0.81(0.782-0.847)	0.8(0.775-0.84)	0.23†
Range	0.71-1.2	0.7-1.2	0.7-1.2	

† Independent t test, * Fisher's exact test

There were no significant differences observed in systolic blood pressure (SBP) (p value = 0.371) and diastolic blood pressure (DBP) (p value = 0.554) between the PCOD and non-PCOD groups. The mean ± SD values for SBP and DBP in the PCOD group were 118.72 ± 9.79 mmHg and 77.08 ± 8.6 mmHg, respectively, while in the non-PCOD group, they were 116.82 ± 11.3 mmHg and 76.06 ± 8.59 mmHg, respectively, indicating no significant variation between the two groups.

Table 5: -Comparison of lipid profile between PCOD and non-PCOD group.

Lipid profile	PCOD group(n=50)	Non PCOD group(n=50)	Total	P value
WNL	46 (92%)	46 (92%)	92 (92%)	1*
Deranged	4 (8%)	4 (8%)	8 (8%)	
Total	50 (100%)	50 (100%)	100 (100%)	

*** Fisher's exact test**

There was no significant difference observed in liver function test parameters between the PCOD and non-PCOD groups. The median (25th-75th percentile) values of SGOT (IU/L) and SGPT (IU/L) in the PCOD group were 22 (20-32) and 22 (19-29.5), respectively, while in the non-PCOD group, they were 21.5 (18-29.5) and 22 (19.25-28), respectively (p values = 0.249 and 0.843, respectively). Similarly, no significant difference was noted in serum albumin (g/dL), serum protein (g/dL), and serum bilirubin (mg/dL) levels between the two groups (p values = 0.073, 0.241, and 0.626, respectively). The mean ± SD values of serum albumin, serum protein, and serum bilirubin in the PCOD group were 3.87 ± 0.67 g/dL, 7.22 ± 0.56 g/dL, and 0.54 ± 0.21 mg/dL, respectively, while in the non-PCOD group, they were 4.14 ± 0.81 g/dL, 7.07 ± 0.7 g/dL, and 0.57 ± 0.23 mg/dL, respectively, with no significant difference between the groups. The proportion of patients with significant ovarian volume >10cc was markedly higher in the PCOD group compared to

the non-PCOD group (100% vs 0%, respectively). Conversely, the proportion of patients with ovarian volume <10cc was significantly lower in the PCOD group compared to the non-PCOD group (0% vs 100%, respectively). This disparity was statistically significant (p value < 0.0001). The distribution of hepatic steatosis was similar between the PCOD and non-PCOD groups. In the PCOD group, 82% had no hepatic steatosis, 8% had mild, 8% had moderate, and 2% had severe steatosis, while in the non-PCOD group, these percentages were 92%, 6%, 2%, and 0% respectively. The comparison yielded a p- value of 0.234, indicating no statistically significant difference between the groups. The mean ± SD of fibroscan readings in the PCOD group was 6.28 ± 2.29, which was significantly higher than that in the non-PCOD group, which had a mean ± SD of 3.6 ± 0.61 (p value=0.046). This suggests a notable difference in fibroscan measurements between the two groups. The distribution of metabolic syndrome was similar between the PCOD and non- PCOD groups, with 94% and 98% of participants respectively classified as without metabolic syndrome and 6% and 2% respectively classified as with metabolic syndrome (p value=0.617). This indicates no significant difference in the prevalence of metabolic syndrome between the two groups. The distribution of NAFLD was similar between the PCOD and non-PCOD groups, with 82% and 92% of participants respectively classified as without NAFLD and 18% and 8% respectively classified as with NAFLD (p value=0.234

Table 6- Showing LFT comparison between PCOD and non-PCOD group

Liver function test parameters	PCOD group(n=50)	Non PCOD group(n=50)	Total	P value
SGOT(IU/L)				
Mean ± SD	27.66 ± 14.78	24.64 ± 10.5	26.15 ± 12.85	0.249‡
Median (25th-75th percentile)	22(20-32)	21.5(18-29.5)	22(18-30.5)	
Range	15-97	12-57	12-97	
SGPT(IU/L)				
Mean ± SD	27.06 ± 15.84	25 ± 8.97	26.03 ± 12.85	0.843‡
Median(25th-75th percentile)	22(19-29.5)	22(19.25-28)	22(19-28)	
Range	13-117	14-66	13-117	
Serum albumin(g/dL)				
Mean ± SD	3.87 ± 0.67	4.14 ± 0.81	4.01 ± 0.75	0.073†
Median (25th-75th percentile)	3.75(3.35-4.35)	4.05(3.6-4.5)	3.85(3.5-4.4)	
Range	2.8-5.8	3-6.2	2.8-6.2	
Serum protein(g/dl)				
Mean ± SD	7.22 ± 0.56	7.07 ± 0.7	7.14 ± 0.64	0.241†
Median (25th-75th percentile)	7.2(6.9-7.6)	7.05(6.525-7.6)	7.2(6.8-7.6)	
Range	6-8.2	4.5-8.2	4.5-8.2	
Serum bilirubin(mg/dL)				
Mean ± SD	0.54 ± 0.21	0.57 ± 0.23	0.55 ± 0.22	0.626†
Median (25th-75th percentile)	0.49(0.4-0.7)	0.54(0.4-0.76)	0.5(0.4-0.73)	
Range	0.1-1.02	0.2-1.02	0.1-1.02	

Table 7: -Comparison of ovarian volume between PCOD and non-PCOD group.

Ovarian volume	PCOD group(n=50)	Non PCOD group(n=50)	Total	value
<10cc	0 (0%)	50 (100%)	50 (50%)	P <0.0001*
Significant(>10cc)	50 (100%)	0 (0%)	50 (50%)	
Total	50 (100%)	50 (100%)	100 (100%)	

Table 8: -Comparison of hepatic steatosis between PCOD and non-PCOD group.

Hepatic steatosis	PCOD group(n=50)	Non PCOD group(n=50)	Total	P value
No	41 (82%)	46 (92%)	87 (87%)	0.234*
Mild	4 (8%)	3 (6%)	7 (7%)	
Moderate	4 (8%)	1 (2%)	5 (5%)	
Severe	1 (2%)	0 (0%)	1 (1%)	
	50 (100%)	50 (100%)	100 (100%)	

Table 9: - Comparison of different variables between patients with and without NAFLD.

VARIABLES	NAFLD PRESENT (N=13)	NAFLD ABSENT (N=87)	P VALUE
Age (years)	28.15 ± 4.85	26.75 ± 4.86	0.332‡

Body mass index(kg/m²)	24.85 ± 1.91	23.22 ± 1.99	**0.007**
Waist circumference(cm)	34.31 ± 3.73	31.91 ± 2.53	**0.004**
Hip circumference(cm)	41.46 ± 3.07	39.68 ± 4.35	0.158
Waist hip ratio	0.86 ± 0.12	0.81 ± 0.09	0.131
Systolic blood pressure(mmHg)	120.62 ± 11.5	117.34 ± 10.42	0.3
Diastolic blood pressure(mmHg)	77.38 ± 11.27	76.45 ± 8.17	0.715
Hemoglobin(g/dL)	10.86 ± 1.3	10.15 ± 1.22	0.054
Fasting blood sugar(mg/dL)	95.46 ± 16.8	86.2 ± 11.51	0.075
TSH(mIU/L)	2.68 ± 1.66	1.71 ± 1.05	0.052
SGOT(IU/L)	50.15 ± 15.94	22.56 ± 7.37	**<0.0001**
SGPT(IU/L)	45.85 ± 22.42	23.07 ± 7.19	**<0.0001**
Testosterone(ng/dL)	30.77 ± 11.56	29.39 ± 15.61	0.281
Endometrial thickness(mm)	6.42 ± 3.08	7.24 ± 2.61	0.306
Metabolic syndrome	3 (23.08%)	1 (1.15%)	**0.007**

Table 10: -Comparison of fibroscan between PCOD and non-PCOD group.

Fibroscan	PCOD group(n=9)	Non PCOD group(n=4)	Total	P value
Mean ± SD	6.28 ± 2.29	3.6 ± 0.61	5.45 ± 2.29	
Median (25th-75th percentile)	5.8(5.2-6.9)	3.55(3.15-4)	5.2(3.9-6.2)	
Range	3.9-11.7	3-4.3	3-11.7	**0.046**[†]

Table 11: -Comparison of metabolic syndrome between PCOD and non-PCOD group.

Metabolic syndrome	PCOD group(n=50)	Non PCOD group(n=50)	Total	P value
Absent	47 (94%)	49 (98%)	96 (96%)	
Present	3 (6%)	1 (2%)	4 (4%)	
Total	50 (100%)	50 (100%)	100 (100%)	0.617[*]

Table 12: -Comparison of NAFLD between PCOD and non-PCOD group.

NAFLD	PCOD group(n=50)	Non PCOD group(n=50)	Total	P value
Absent	41 (82%)	46 (92%)	87 (87%)	
Present	9 (18%)	4 (8%)	13 (13%)	0.234[*]
Total	50 (100%)	50 (100%)	100 (100%)	

Table 13: -Association of NAFLD with metabolic syndrome.

NAFLD	Met S absent(n=96)	Met S present(n=4)	Total	P value
Absent	86 (89.58%)	1 (25%)	87 (87%)	
Present	10 (10.42%)	3 (75%)	13 (13%)	
Total	96 (100%)	4 (100%)	100 (100%)	**0.007**[*]

IV. Summary-

The present study was planned with an aim to assess the association of Polycystic Ovarian Disease with Non-Alcoholic Fatty Liver Disease. The study included a total of 100 participants divided in two groups i.e., 50 participants in PCOD group and 50 in Non PCOD group. NAFLD as well as MetS were assessed in all the study participants. In the present study, 72% of participants were under 30 years of age and 28% were in 31 to 40 years age group. The difference of age group between PCOD and non PCOD group participants was statistically significant (p-value 0.026). Abnormal uterine bleeding was most common chief complaints among all participants in the study (41%), with significantly higher in PCOD group participants (60%) as compared to non PCOD group (22%). Secondary amenorrhea (18%) and hirsutism (12%) were other major chief complaints among participants of PCOD group. Majority (46%) of participants were nulliparous, with significantly higher in PCOD group (62%) as compared to non PCOD group (30%). BMI of study participants in both groups was significantly different with higher BMI observed in PCOD group (23.93 ± 1.96) as compared to non PCOD group (22.94 ± 2.03). Waist circumference was not observed to be statistically different in both groups. Hip circumference was significantly higher in PCOD group (40.76 ± 3.53) as compared to non PCOD group (39.06 ± 4.73). Waist Hip ratio was similar between both groups with no

significant difference. Systolic blood pressure and Diastolic blood pressure readings were similar in both groups and no statistically significant difference was observed. It was observed in present study that hemoglobin levels were not significantly different between two groups (p-value = 0.556). Fasting blood sugar were also observed to have no statistically significant difference between both groups (p-value = 0.887). Lipid profile was observed to be deranged in 4 participants in each group. TSH levels were found to be similar in both groups, with no statistically significant difference (p-value = 0.989). SGOT, SGPT, Serum albumin, Serum protein and Serum bilirubin were not found to be significantly different between both groups (p-value >0.05). It was observed that all the patients were non-reactive for viral markers. Testosterone levels were observed to be similar in both groups (p-value = 0.464). Ovarian volume >10cc were observed in 100% of participants in PCOD group, whereas none in non PCOD group. Endometrial thickness was significantly less among participants in PCOD group as compared to non PCOD group. Hepatic steatosis was observed in 18% of participants of PCOD group as compared to 8% in non PCOD group (p-value = 0.234). Fibro scan readings were significantly higher in participants of PCOD group as compared to non PCOD group (p-value = 0.046), with 1 participant in the PCOS group having significant liver fibrosis (>8.2kpa). Metabolic syndrome was present in 6% of participants in PCOD group and only among 2% in non PCOD group (p-value = 0.617). NAFLD was present in 18% of participants in PCOD group and only among 8% in non PCOD group (p-value = 0.234). Prevalence of NAFLD was significantly higher among participants with Metabolic syndrome (75%) as compared to those without metabolic syndrome (10.42%) (p-value = 0.007).

V. Conclusion-

This study was done to find the association of NAFLD with PCOS, and also with Metabolic Syndrome. Although, the presence of hepatic steatosis and consequently NAFLD wasn't found to be higher in PCOS group, liver fibroscan score was raised, marking significant hepatic fibrosis in them. NAFLD was found to be significantly associated with Metabolic Syndrome, indicating the need for early liver function assessment in these patients, to avoid long term complications and improve their quality of life.

Conflict Of Interest- The authors declare that there was no conflict of interest and no funding was taken from any source for conducting this study**.**

Ethical Clearence- The required ethical clearance was taken before conducting this study from Ethical Committee of University of Health Sciences, Rohtak.

References

[1] Doi SA, Al-Zaid M, Towers PA, Scott CJ, Al-Shoumer KA. Ovarian Steroids Modulate Neuroendocrine Dysfunction In Polycystic Ovary Syndrome. J Endocrinol Invest. 2005; 28:882-92.
[2] EL Hayek S, Bitar L, Hamdar HL, Mirza FG, Daoud G. Poly Cystic Ovarian Syndrome: An Updated Overview. Front Physiol. 2016; 7:124.
[3] Joshi B, Mukherjee S, Patil A, Purandare A, Chauhan S, Vaidya R. A Cross- Sectional Study Of Polycystic Ovarian Syndrome Among Adolescent And Young Girls In Mumbai, India. Indian J Endocrinol Metab. 2014;18(3):317-24.
[4] Nidhi R, Padmalatha V, Nagarathna R, Amritanshu R. Prevalence Of Polycystic Ovarian Syndrome In Indian Adolescent. J Pediatr Adolesc Gynecol. 2011; 24:223- 7.
[5] The Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 Consensus On Diagnostic Criteria And Long-Term Health Risk Related To Polycystic Ovary Syndrome. Fertil Steril. 2004;81(1):19-25.
[6] Azziz R, Carmina E, Dewailly D, Diamanti-Kandarakis E, Escobar-Morreale HF, Futterweit W, Et Al. Position Statement: Criteria For Defining Polycystic Ovary Syndrome As A Predominantly Hyperandrogenic Syndrome: An Androgen Excess Society Guideline. J Clin Endocrinol Metab. 2006; 91:4237-45.
[7] Talbott E, Guzick D, Clerici A, Berga S, Detre K, Weimer K, Et Al. Coronary Heart Disease Risk Factors In Women With Polycystic Ovarian Syndrome. Arterioscler Thromb Vasc Biol. 1995; 15:821–6.
[8] Dahlgren E, Janson PO, Johansson S, Lapidus L, Oden A. Polycystic Ovary Syndrome And Risk For Myocardial Infarction: Evaluated From A Risk Factor Model Based On A Prospective Study Of Women. Acta Obstet Gynecol Scand. 1992; 71:599-604.
[9] Conway G, Agrawal R, Betteridge DJ, Jacobs HS. Risk Factors For Coronary Heart Disease In Lean And Obese Women With PCOS. Clin Endocrinol. 1992; 37:119-25.
[10] Dunaif A. Insulin Resistance And The Polycystic Ovarian Syndrome: Mechanisms And Implications For Pathogenesis. Endocr Rev. 1997; 18:774-800.
[11] Blank SK, Mccartney CR, Marshall JC. The Origins And Sequelae Of Abnormal Neuroendocrine Function In Polycystic Ovary Syndrome. Hum Reprod Update. 2006;12(4):351-361.
[12] Gainder S, Sharma B. Update On Management Of Polycystic Ovarian Syndrome For Dermatologists. Indian Dermatol Online J. 2019;10(2):97-105.
[13] Coviello AD, Legro RS, Dunaif A. Adolescent Girls With Polycystic Ovary Syndrome Have An Increased Risk Of The Metabolic Syndrome Associated With Increasing Androgen Levels Independent Of Obesity And Insulin Resistance. J Clin Endocrinol Metab. 2006; 91:492-7.
[14] Chandrasekhran S, Sagili H. Metabolic Syndrome In Women With Polycystic Syndrome. Obstet Gynecol. 2018; 20:245-52.
[15] Alberti KG, Zimmet P, Shaw J. Metabolic Syndrome: A New Worldwide Definition. A Consensus Statement From The International Diabetes Federation Diabet Med. 2006;23(5):469-480.
[16] Abd El-Kader SM, El-Den Ashmawy EM. Non-Alcoholic Fatty Liver Disease: The Diagnosis And Management. World J Hepatol.

2015;7(6):846-858.
[17] Brunner KT, Henneberg CJ, Wilechansky RM, Long MT. Nonalcoholic Fatty Liver Disease And Obesity Treatment. Curr Obes Rep. 2019;8(3):220-228
[18] Castera L, Friedrich-Rust M, Loomba R. Noninvasive Assessment Of Liver Disease In Patients With Nonalcoholic Fatty Liver Disease. Gastroenterology.2019;156(5):1264-1281.E4.