

Oxidative Stress and Anthropometric characteristics in Polycystic Ovary Syndrome (PCOS) -A preliminary investigation

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Abstract

The study was conducted to assess the role of oxidative stress, host factors with respect to PCOS. A total of 131 subjects (69 PCOS and 62 non-PCOS) were enrolled. Demographic, reproductive, anthropometric data, clinical characteristics of hyper-androgenism was recorded, and blood was collected. The mean age was significantly lower while waist, hip circumference, and BMI were significantly higher in PCOS subjects. The polycystic ovary was observed to be ~ 90% in PCOS and ~13% in non-PCOS subjects. Irregular menses, acnes, hirsutism were also more in PCOS cases. The LPO, total thiols, and protein levels were higher, while SOD, GSH and GST levels were lower in PCOS subjects with respect to non-PCOS subjects. The decline in GST level was significant. Further analysis indicated that alterations in OS parameters were more in subjects with three clinical PCOS diagnostic symptoms compared to subjects with two symptoms or non-PCOS subjects. The differences in obesity, waist,

hip circumference, BMI, features of hyperandrogenism and oxidative stress markers were higher in PCOS than non-PCOS subjects.

Keywords: Polycystic Ovary Syndrome, Body Mass Index, Menstruation, Hyperandrogenism, Oxidative stress, Anthropometric characteristic

Introduction

Polycystic ovary syndrome is a common syndrome of women. It causes complications like menstruation irregularities, impaired sex hormones, and infertility in women. In this syndrome, many small cysts are grown in either one or both the ovaries. It is known to be one of the foremost reasons of infertility among female. However, prevalence of PCOS is greatly variable globally because of the different criteria being used by the investigators. Welt et al. (2013) mentioned that occurrence of the PCOS symptoms like clinical signs of hyperandrogenism and menstrual irregularities during adolescence might be developed with the interaction of both genetic and environmental factors that might

occurred during the early life^[1]. It is rational that exposure of predispose PCOS subjects to the environmental factors in combination with genetic factors may lead to the expression of PCOS phenotypes at later life. Abbott et al. (2013) reported that fetal exposure to testosterone, among non-human primate models develops the closest to PCOS-like symptoms, and female rhesus monkeys exposed to testosterone during early-to-mid gestation exhibiting reproductive, metabolic dysfunction and endocrinal characteristics which are co-pathologies of PCOS^[2]. Earlier, in 2003, a consensus workshop on PCOS held at Rotterdam, Netherlands inferred that PCOS is an ovarian dysfunction syndrome occurred with polycystic ovary morphology and the cardinal characteristics of hyperandrogenism. PCOS remains as a syndrome, thus as such only one diagnostic criterion such as Polycystic ovary (PCO) or hyperandrogenism is not sufficient for diagnosis of PCOS clinically. Further, PCOS also have some common features such as elevated level of serum luteinizing hormone (LH) and insulin resistance^[3]. Allahbadia and Merchant (2008) reported that it is linked with hyperinsulinemia, insulin resistance, hypertension, abdominal obesity, and dyslipidemia which might also be accountable for coronary artery disease, type 2 diabetes mellitus and endometrial hyperplasia^[4]. The causes of PCOS are subtle yet, but it is presumed that the manifestation of PCOS features in later life might be due to *in utero* fetal programming^[4].

The disproportion between the production of free radicals and scavenging activity of antioxidants causes oxidative stress (OS) which may be harmful to the human health including the reproductive health and pregnancy outcome. Earlier, Agarwal et al. (2005) reported that OS effects the whole reproductive cycle of

a woman, or even at later life i.e. menopause^[5]. Reactive oxygen species affect many physiological processes right from oocyte development to fertilization and embryo development, thereafter, affecting the pregnancy or its outcome. It also plays a key role in normal pregnancy, parturition, and onset of the preterm labor. The impacts of OS on oocytes and reproductive functions are subtle and the imbalance among pro-oxidants and antioxidants level can lead to reproductive implications such as PCOS, endometriosis, and many other infertility problems with unknown causes^[6]. Zuo et al. (2016) also mentioned that whether the abnormal OS levels in PCOS patients is derived from PCOS or they are associated with other potential complications is not yet fully understood^[7].

The precise mechanism of the role of OS and PCOS is not yet fully elucidated but some inconsistent findings on the role of environmental, lifestyle factors and OS in the etiology of occurrence of PCOS are available along with other host factors. Owing to these, this study was carried out to assess the role of oxidative stress, body mass index (BMI), subject's anthropogenic characteristics, clinical features of hyperandrogenism in PCOS.

Material and Methods

One hundred thirty-one subjects who were in reproductive age and attending OPD of Obstetrics and Gynecology Department, Institute of Kidney Disease and Research Centre, Ahmedabad, India for the consultation of gynecological problem were enrolled randomly. Informed consent was taken from each subject after explaining the aims and the benefit of the study. This paper is a part of a major project entitled "*Role of Occupational and environmental factors in PCOS*" for which ethical approval was taken from the Institutional Human Ethical Committee of National

Institute of Occupational Health, Ahmedabad, India. The enrolled women were categorized into two groups as PCOS and non-PCOS using the Rotterdam 2003 criteria for the diagnosis of PCOS, i.e. the subject having minimum two out of the three features 1) oligo-anovulation 2) biochemical and/or clinical signs of hyperandrogenism 3) polycystic ovaries [8]. Women with thyroid dysfunction, hyperplasia, pregnant women, and the other disorders are taken care to follow the exclusion criteria. Among the subjects, 69 women were with PCOS and 62 women were non-PCOS. The socio-demographic information such as age, personal habits, occupational history, and anthropometric data such as weight, height, hip and waist circumference etc. were recorded on predesigned proforma. The BMI and ratio of waist and hip were also calculated. The reproductive history of the subjects such as menstruation disorders, history of pregnancy and the reproductive history of mother, sisters, etc. and information on clinical features of hyperandrogenism i.e., acne in the facial skin and hirsutism were recorded. The data with respect to ovarian morphology was obtained through ovarian observation using ultrasonography. About 5 ml blood samples were drawn through vein puncture during 10 am to 1pm and serum was separated using centrifuge. The biochemical parameters like total protein (TP) (Lowry et al. 1951)^[9], non-enzymatic parameters such as Malondialdehyde (MDA), a stable compound of Lipid Peroxidation (LPO) (Buege and Aust, 1978) ^[10], Reduced glutathione (GSH) level (Ellman's et al. 1959) ^[11], Total thiol (TT) (Hu, 1994) ^[12] and enzymatic parameters i.e. superoxide dismutase (SOD) (Marklund and Marklund, 1974) ^[13] and Glutathione S-transferase (GST) (Habig et al. 1974) ^[14] in the serum of the study subjects was estimated using Multi-mode analyzer

(Bio-Tek, Germany)

Data analysis and Statistics

The data was computerized in excel program and variables were assessed with respect to PCOS and non-PCOS groups and difference between the two groups were determined and significance level was considered at $p < 0.05$. The statistical analysis i.e., Chi square test, t-test, ANOVA post-hoc, Mann-Whitney, Kruskal Willis tests etc. was performed using SPSS 16 for comparing the groups. The data is presented as percentages in the pie charts, mean \pm SE in the tables and graphs.

Results

The mean age was 27.7 years and 31.4 years of the PCOS and non-PCOS subjects respectively, and this difference was statistically significant. The BMI, waist and hip circumferences were also significantly higher in PCOS patients than non-PCOS subjects and ratio of waist to hip circumferences was higher (n.s.) in PCOS subjects as compared to non-PCOS subjects (table 1).

The anthropometric parameters and age variables were further assessed with regards to diagnostic clinical features of PCOS i.e. Group 1) non-PCOS subjects (without or with one diagnostic feature), Group 2) PCOS subjects with two diagnostic features, Group 3) PCOS subjects with three diagnostic features and depicted in table- 2, and found a significantly lower age in PCOS groups (with two and three clinical features) as compare to non-PCOS group. The BMI and waist circumference were higher in group-2 and 3 PCOS subjects than group-1 non-PCOS subjects, which were statistically significant in group-3 as compared to group-1. However, differences in elevations of BMI and waist circumference in group-2 as compare to group-1 and in group-3 as compared to group-2 were insignificant. While the hip circumference was

statistically significantly higher in both PCOS groups as compare to non-PCOS group. In addition, the ratio of waist to hip circumferences was also higher in group-3 compare to non-PCOS and PCOS group -2, which were non-significant.

Cardinal features of the study subjects are depicted in figures 1-4. Polycystic ovaries observed through ultrasonography, were found to be more in PCOS subjects (89.9%) as compare to non-PCOS subjects (12.9%) (Fig.1). Menstruation pattern shows that irregular menstruation was found to be more in PCOS subjects (69.6%) as compared to non-PCOS subjects (4.8%) (Fig.2). Hirsutism was found to be higher i.e. 34.8 % in PCOS subjects which was less than half i.e. 14.5 % in non-PCOS subjects (Fig. 3). Acnes on face were also found to be 47.8 % in PCOS women while it was nearly half i.e. 24.2 % in non-PCOS subjects (Fig.4).

Further data analysis, based upon the waist circumference (more than and less than 80 cm) in both PCOS and non-PCOS subjects indicated that PCOS subjects are more likely to have waist circumference more than 80 cm (72.5%) than non-PCOS subjects (48.4%) which was statistically significant ($p=0.005$, $X^2(1, N=131) = 7.96$). The BMI groups comparison among PCOS and non-PCOS groups showed that underweight, healthy, and overweight were higher among non-PCOS subjects (12.9%, 46.8% & 30.6%) as compare to their respective PCOS subjects (2.9%, 40.6% & 29%). However, the obese subjects were more in PCOS group (27.5%) than non-PCOS group (9.7%). The test chi-square test showed that BMI is associated significantly ($p=0.018$, $X^2(3, N=131) = 10.06$) with PCOS. In addition, chi square post-hoc test with Bonferroni correction indicated that obesity among the four BMI subgroups ($p=0.009$) are contributing majorly, in

association of BMI with PCOS, which was nearest to Bonferroni $p < 0.006$ (Table 3).

The oxidative stress related data are depicted in fig. 5. The Mann Whitney test for data comparison between PCOS and non-PCOS subjects showed that lipid peroxidation was marginally higher in PCOS subjects as compare to non-PCOS subjects. Total thiols and total protein levels were also higher slightly in PCOS patients as compare to non-PCOS subjects. However, the enzymatic antioxidants like SOD and GST levels were lower among the PCOS subjects as compare to non-PCOS subjects, and the difference in GST level was statistically significant ($p= 0.037$). While level of GSH was slightly lower in PCOS subjects.

The OS parameter were also correlated with respect to non-PCOS subjects (Gr-1), PCOS subjects with two (Gr-2) and with three diagnostic features (Gr-3). The Kruskal-Wallis test for comparison among the groups indicated that SOD level was significantly different amongst the three groups ($p=0.047$) while difference in GST levels was near to significance level ($p= 0.057$). However, alterations in other studied biochemical parameters were non-significant. Further, Mann-Whitney test for the two groups comparison indicated that LPO level was higher in Group-2 and 3 as compare to Group-1. The antioxidants like SOD, GST and GSH levels were higher in group-1 (non-PCOS subjects) than group-2 and 3 (PCOS) subjects, wherein SOD and GST levels were statistically significantly higher in group-1(non-PCOS) as compare to group-3 subjects ($p < 0.05$). However, total thiols and total protein levels were slightly higher (n.s.) in group-2 and 3 as compare to group-1 subjects. In addition, the difference in total thiols and total protein amongst the groups was also non-significant and marginal (fig. 5).

Discussion

A significantly lower mean age and higher hip, waist circumference and BMI were observed in the PCOS women as compared to non-PCOS women. This corroborates with the earlier findings of Chitme et al. (2017), they also found that PCOS subjects had higher levels of body fat distribution, BMI, waist and hip circumference, visceral fat, diastolic and mean blood pressure [15]. Earlier Ahmadi et al. (2013) did not find significant difference in mean BMI between the PCOS and non-PCOS groups; however, they found a significantly higher mean waist circumference in the PCOS subjects and hirsutism was significantly higher among overweight PCOS patients compared to the normal weight PCOS patients [16]. Later, Yuan et al. (2016) reported that PCOS subjects with higher BMI were more susceptible to androgen excess; thus, BMI may be an indicator of hyperandrogenism among PCOS subjects [17]. The BMI, waist circumference, hip circumference and hip to waist circumference ratio were greater in PCOS women with three diagnostic PCOS features as compared to subjects with two features.

The clinical features like characteristics of hyperandrogenism i.e. face with acne and hirsutism were found more in PCOS subjects. Earlier, Akshaya and Bhattacharya (2016) reported that more than 50% of the PCOS subjects were obese and most of the PCOS subjects were with the clinical features like hirsutism, menstrual disorders, acne, infertility, and acanthosis nigricans irrespective of body weight [18]. In present study, about 56% of PCOS subjects were overweight/obese while it was ~ 40% in non-PCOS group and a considerably greater number of obese cases were in PCOS group. Earlier, Lergo (2012) reported that obesity might not be a cause of PCOS, and a

considerable number of PCOS women are with normal weight [19]. However, many phenotypic characteristics were aggravated by obesity, a meager response to infertility treatment and elevated hazard for pregnancy complications could be linked with obesity. Additionally, PCOS obese women had a higher prevalence of hirsutism as well as menstrual disorders compared to non-obese PCOS women [20]. The polycystic ovary and menstruation irregularity were more common in PCOS subjects than the non-PCOS subjects in the study. Earlier, Mahmoud et al. (2015) also reported a significantly higher occurrence of menstrual irregularity, age of menarche and abortion among the overweight and obese PCOS subjects [21]. Obese PCOS subjects have more severe hyperandrogenism and associated clinical features such as menstrual abnormalities, anovulation, and hirsutism than normal-weight PCOS women [22]. Very recently, Kshetrimayum et al. (2019) reviewed that PCOS might be initiated *in utero* and phenotypic appearance of PCOS signs may be established at later life which could be related with host factors (endogenous), and lifestyle, dietary, environmental factors (exogenous) [23].

Recently, Mujica et al. (2018) mentioned that an elevated inflammatory status and disruption in oxidative and antioxidative balance among women with PCOS. They evaluated the metabolic and OS parameter in prenatal and postnatal androgenized rat PCOS models and showed that testosterone propionate exposure after birth could promote the alterations in oxidative stress, which is probably associated with lipid disarrangement and/or anovulation [24]. In the present study MDA, TT and TP level were higher while the SOD, GST and GSH levels was lower in PCOS women even though most of the data were insignificant indicating possible role of OS in PCOS. Earlier, Yeon

Lee et al. (2010) reported that when ROS are out of balance with antioxidants, resulting OS, thereby affecting DNA, and/or causing cell apoptosis. Moreover, reactive nitrogen species, like nitrogen oxide with an unpaired electron are extremely reactive and harmful [25]. Murri et al. (2013) reviewed sixty-eight studies and reported that PCOS patients were having higher concentrations of free homocysteine (23%), malondialdehyde (47%), and asymmetric dimethylarginine (36%), SOD activity (34%) and declined glutathione levels (50%) and paraoxonase-1 activity (32%) and concluded that OS may participate in the pathophysiology of PCOS [26]. Papalou et al. (2016) also mentioned that although mechanisms behind PCOS have not elucidated fully, it is evident that OS has an important role in the pathogenesis of PCOS [27]. The LPO level was higher in both PCOS group (with 2 and 3 diagnostic features of PCOS) subjects as compare to non-PCOS subjects while antioxidants like SOD, GST and GSH levels were higher in non-PCOS subjects than PCOS subjects. The levels of SOD and GST were statistically significantly higher in non-PCOS subjects than the PCOS subjects with three diagnostic features of PCOS. Earlier, Papalou et al. (2016) reported that OS holds a reasonable part in the pathogenesis of PCOS. In fact, PCOS might be considered as a purely oxidative state, where the body antioxidants level cannot encounter the excessive generation of free radicals [27]. Recently, Sulaiman et al. (2018) offers supportive evidence that OS might play an important role in the pathogenesis of PCOS and OS markers could be considered as diagnostic markers for early diagnosis of PCOS [28]. Based upon the data available along with data attained, we also infer that OS might also involve in pathophysiology of PCOS. It can also be suggested that

higher BMI, waist circumference, hyperandrogenism clinical features as well as OS may have some role in occurrence of PCOS. More studies are needed on this issue of human reproduction.

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Conflict of Interest: Kshetrimayum Chaoba has a role in sample as well as data collection, processing, estimation of Oxidative stress parameters and in Manuscript preparation

Dr. Anupama Sharma has a role in manuscript preparation and data analysis.

Dr. Rohina Agarwal and Dr. V V Mishra involved as gynecologist in the study for clinical data collection and interpretation.

Dr. Sunil Kumar has a role in conceptualization, execution of the project, data analysis and preparation of manuscript.

Kshetrimayum Chaoba, Dr. Anupama Sharma, Dr. V V Mishra, Dr. Rohina Agarwal and Dr. Sunil Kumar declare that they have no conflict of interest.

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Legends Tables and Figure

Table 1: Age and anthropometric comparison among PCOS and Non-PCOS groups

Groups	Age (yrs)	BMI (Kg/m ²)	Waist Circumference (cm)	Hip Circumference (cm)	Waist/ Hip Circumference Ratio
Non-PCOS group(n=62)	31.39 ± 0.54	23.79 ± 0.60	80.73 ± 1.54	96.64 ± 1.21	0.834 ± 0.011
PCOS group(n=69)	27.71±0.45*	26.33 ± 0.58*	85.41* ± 1.23	102.30* ± 1.29	0.836 ± 0.008

*Significant as compare to non-PCOS subjects (p<0.05)

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Table 2: Age and anthropometric comparison among two subgroups of PCOS and non-PCOS groups

Groups	Age (years)	BMI (Kg/m ²)	Waist Circumference (cm)	Hip Circumference (cm)	Waist: Hip circumference Ratio
Group-1 (Non-PCOS, n=62)	31.39 ± 0.54	23.79 ± 0.60	80.73 ± 1.54	96.64 ± 1.21	0.834± 0.011
Group-2 (PCOS subjects with two diagnostic features, n=47)	28.09± 0.54*	25.87 ± 0.72	84.31± 1.50	102.03± 1.72*	0.828± 0.011
Group-3 (PCOS subjects with three diagnostic features n=22)	26.91 ± 0.82*	27.29 ± 0.98*	87.75± 2.10*	102.86± 1.71*	0.853 ± 0.014

* Significance with respect to non-PCOS, P<0.05

Table 3: Waist circumference (< and > 80cm) and BMI (Kg/m²) subgroups in PCOS and Non-PCOS groups

Groups	Waist Circumference (cm)		BMI (Kg/m ²) groups in PCOS and Non-PCOS			
	< 80 cm	> 80 cm	Underweight (<18.5)	Healthy (18.5-24.9)	Overweight (25-29.9)	Obese (≥ 30)
Non-PCOS (n=62)	51.6% (32)	48.4% (30)	12.9% (8)	46.77% (29)	30.64% (19)	9.67% (6)
PCOS (n=69)	27.5% (19)	72.5%* (50)	2.9% (2)	40.6% (28)	28.99% (20)	27.54%*(19)

*Significance compare with non-PCOS subjects (p<0.05), Bonferroni correction for BMI groups comparison (p<0.0062)

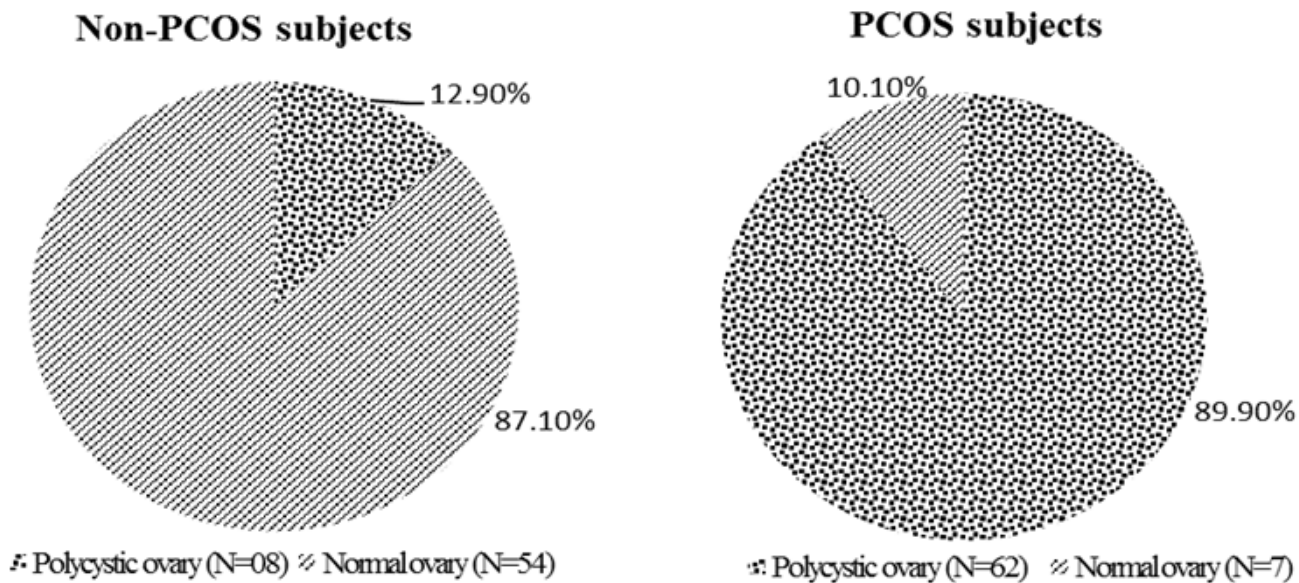


Fig. 1: Ultrasonographic Ovarian morphology of PCOS and non-PCOS subjects

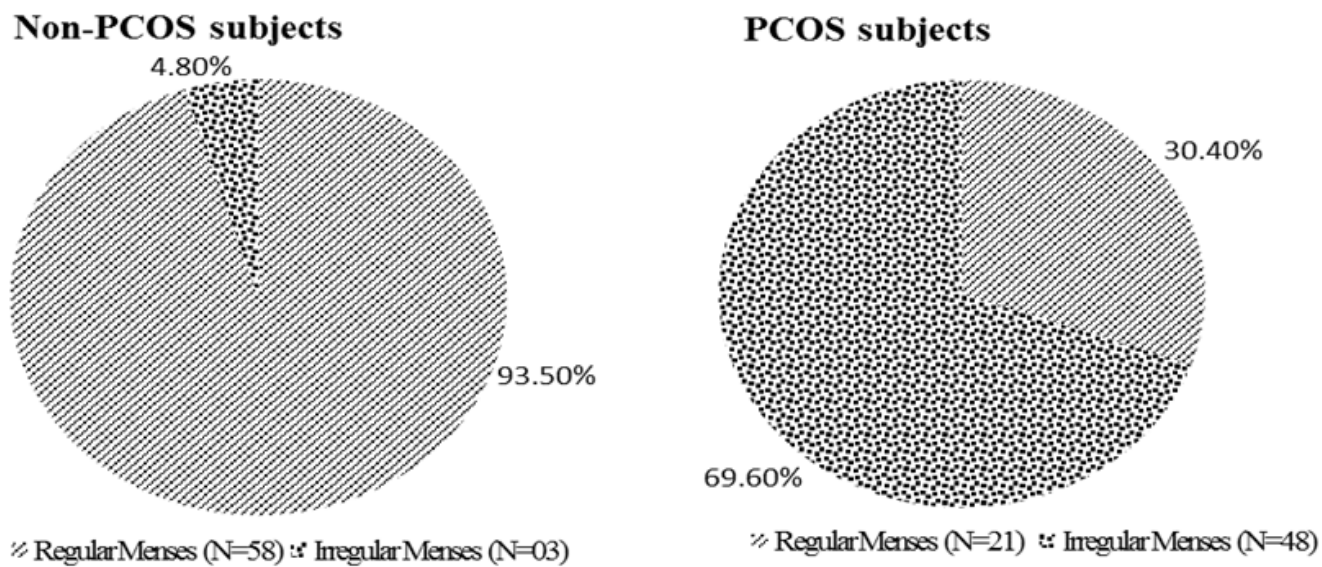


Fig. 2: Menstruation cycle of PCOS and non-PCOS subjects

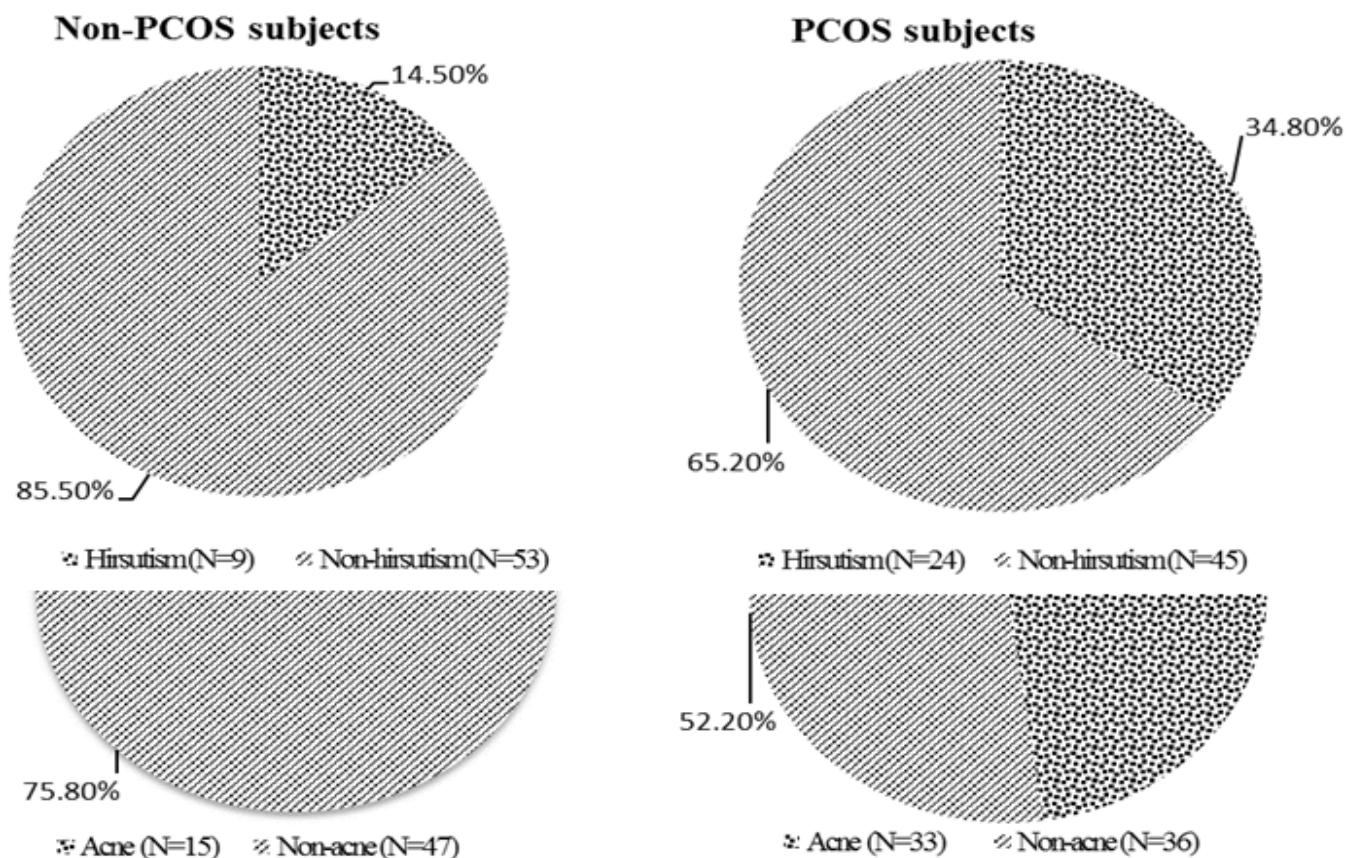


Fig. 3: Acne on the face of PCOS and non-PCOS subjects

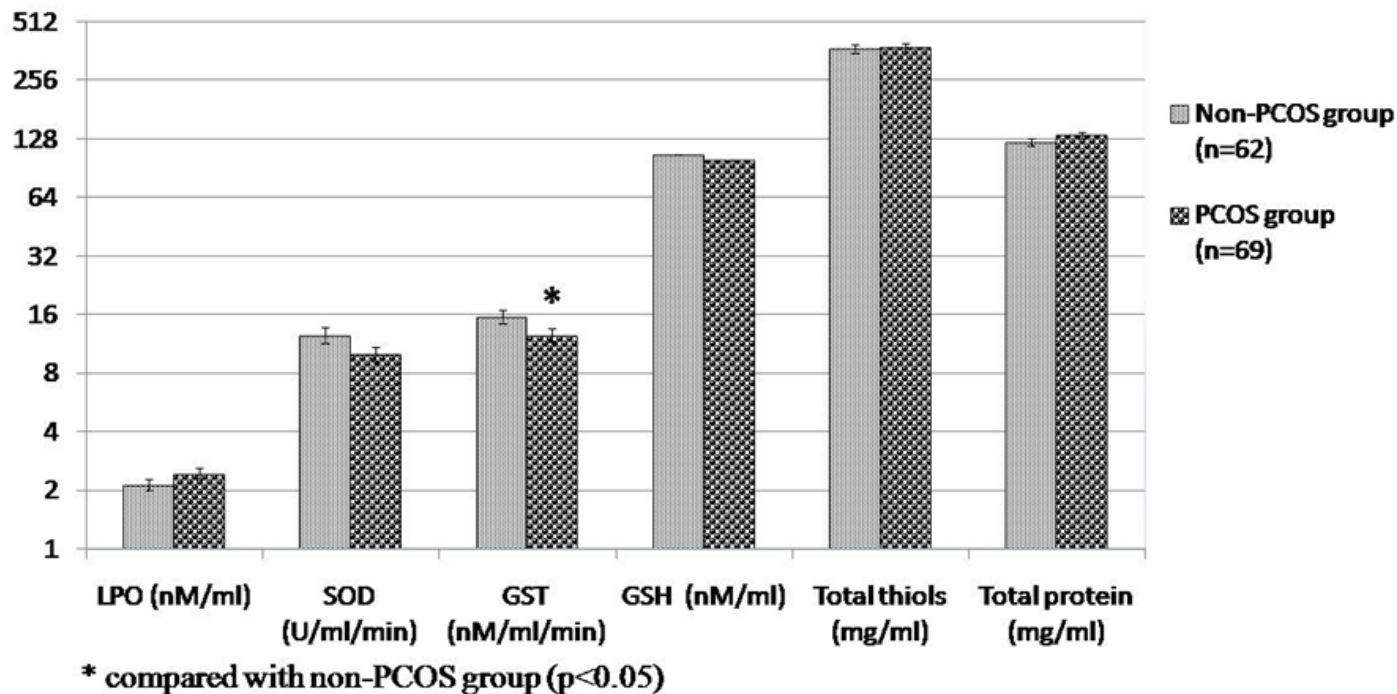


Fig. 4: Oxidative stress parameters in PCOS and non-PCOS groups.

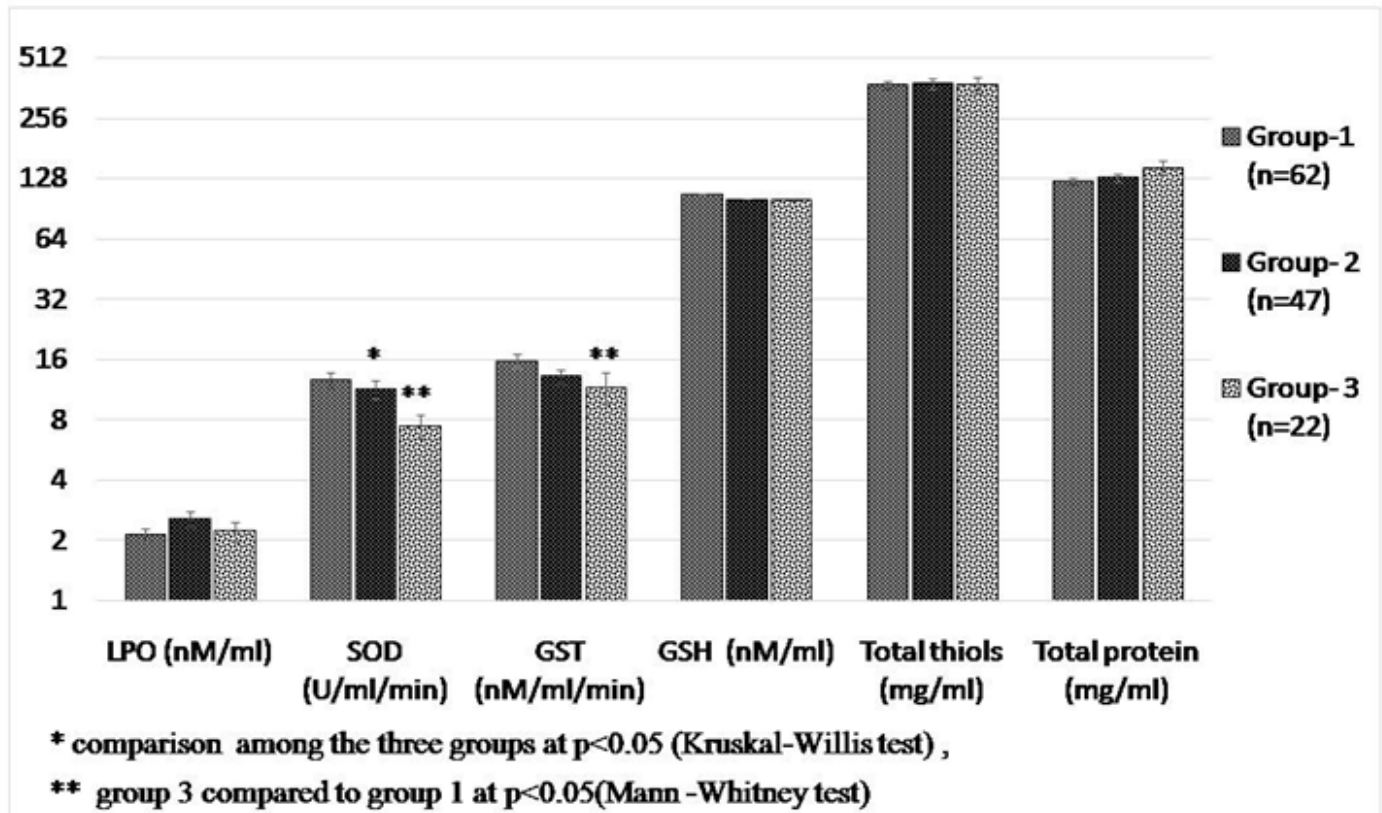


Fig. 5: Oxidative stress parameters in two PCOS groups and non-PCOS group.