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Cutaneous Manifestations and Their Relationship with Serum Testosterone and DHEAS Levels in Women with Polycystic Ovary Syndrome: A Cross-Sectional Clinical Study

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ABSTRACT

Context: Polycystic ovary syndrome (PCOS) is one of the most common heterogenous endocrinopathy in women affecting 5–10% of women in reproductive age group. Excess synthesis of ovarian androgens plays an essential role in the clinical and biochemical manifestations of hyperandrogenism in patients with PCOS. Cutaneous manifestations might be the first sign of PCOS. Hence, this study was intended to evaluate the correlation between cutaneous and biochemical markers of hyperandrogenism in PCOS patients.
Aims: To determine the profile of cutaneous manifestations and to evaluate their correlation with biochemical hyperandrogenism in women with PCOS.
Settings and Design: A cross-sectional clinical study was carried out at a tertiary care hospital over a period of 18 months from May 2024 to December 2025
Methods and Material: Seventy-one consecutively diagnosed cases of PCOS were enrolled. Cutaneous manifestations were ascertained, hirsutism was assessed using the modified Ferriman–Gallwey score, severity of acne was assessed, and androgenic alopecia (AGA) was assessed using the Ludwig's scale. Biochemical hyperandrogenism was determined from serum concentration of total testosterone (TT), free testosterone (FT) and dehydroepiandrosterone sulfate (DHEAS). Fasting serum insulin levels and ultrasonological assessment was done in all the cases.
Statistical analysis used: Associations between clinical hyperandrogenism and serum androgen levels were analyzed using Spearman's rank correlation. The strength of association was measured by correlation co-efficient (r). A two-tailed P-value of <0.05 was considered statistically significant.
Results: The prevalence of hirsutism, acne, AGA, acanthosis nigricans, seborrhea, and acrochordons was 67%, 65%, 51%, 43%, 24%, and 10% respectively. Levels of TT, FT, and DHEAS were raised in 66%, 51%, and 31% of patients respectively. There was a statistically significant correlation in the following pairs: hirsutism and FT (P< 0.05); hirsutism and DHEAS (P< 0.05); acne and FT (P< 0.05); acne and TT (P<0.05); and AN and insulin (P< 0.05).
Conclusions: Hirsutism and acne are good predictors for biochemical hyperandrogenism in women with PCOS in our study population. Early correction of biochemical hyperandrogenism will help to ameliorate the cutaneous markers and improve the quality of life of these patients.

***Keywords**: Acne, androgenic alopecia, hirsutism, hyperandrogenism, polycystic ovary syndrome.*

INTRODUCTION

Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders in women of reproductive age with a prevalence of 5-10%. It is characterized by clinical and/or biochemical hyperandrogenism and chronic anovulation. [1] Approximately, 80-85% of women with hyperandrogenism will be diagnosed with PCOS. [2] A revised definition of PCOS was proposed in 2003 at an international joint consensus meeting of the European Society for Human Reproduction and Embryology and the American Society for Reproductive Medicine. [3] It is also called as the Revised 2003 Rotterdam criteria and the most widely used criteria. The group recommended that a PCOS diagnosis could be achieved if two of the following three criteria are present:

- Oligo-ovulation (fewer than 8 menses per 12-month period) and/or anovulation;
- Clinical hyperandrogenism and/or biochemical signs of hyperandrogenism;
- Polycystic ovaries (≥12 follicles in each ovary measuring 2–9 mm in diameter and/or increased ovarian volume >10 ml) by ultrasonography.

Revised 2003 Rotterdam criteria and The Androgen Excess Society (AES) criteria for diagnosis of PCOS requires clinical and/or biochemical evidence of hyperandrogenism as one of the criteria. [3,4] Excess synthesis of ovarian androgens plays an essential role in the clinical and biochemical manifestations of hyperandrogenism in patients with PCOS. However, few studies have shown that minority of PCOS women might not have hyperandrogenism. [3,5]

Cutaneous manifestations might be the first sign of PCOS. Amongst the various cutaneous manifestations, hirsutism is the most common, which is present in up to 80% of the patients with androgen excess followed by acne, androgenic alopecia (AGA) and acanthosis nigricans (AN). Recently, PCOS has been associated with obesity, insulin-resistance (IR), and a risk of developing Type 2 diabetes mellitus (T2DM). [6]

Androgen excess is defined as an elevated serum level of one or more androgens, that is, Total Testosterone (TT), Free Testosterone (FT), Dehydroepiandrosterone Sulphate (DHEAS) and Androstenedione (ADD). [7] The metabolic and reproductive abnormalities predispose women to develop infertility and endometrial cancer, necessitating early diagnosis and appropriate treatment. [8]

Nevertheless, there are evidences that clinical hyperandrogenism can be found in women without hyperandrogenemia, and on the other hand, biochemical hyperandrogenemia can be detected in women without clinical hyperandrogenism. [9]

Limited studies have evaluated the relationship between clinical and biochemical hyperandrogenism and these have reported conflicting findings. [9-11] Confirmed associations between biochemical and clinical characteristics in patients with PCOS have important implications for follow-up and management. As the quality of life is impaired in patients with PCOS due to cutaneous manifestations, hormonal correlations may help to determine the severity and to choose appropriate and efficacious treatment options.

Hence, in this study we sought to determine the profile of cutaneous manifestations and the association between biochemical hyperandrogenism parameters and cutaneous markers for better understanding of the pathophysiology in South Indian women with PCOS.

SUBJECTS AND METHODS

A cross-sectional clinical study was carried out at a tertiary care hospital over a period of 18 months from December 2017 to May 2019. Seventy-one consecutively diagnosed cases of PCOS attending the departments of dermatology/obstetrics and gynecology aged between 15 and 45 years were enrolled in the present study after obtaining an informed consent. The diagnosis of PCOS was made according to the Revised 2003 Rotterdam Criteria. [3] Women were excluded from the present study if they had one of the following conditions: congenital adrenal hyperplasia, androgen secreting tumors, Cushing's syndrome or hypothyroidism; taking medications that affect endocrinal patterns (hormonal therapy or steroid agents within 3 months before enrollment); history of treatment for acne or hirsutism within 3 months before enrollment.

After obtaining institutional ethics committee approval and a written informed consent, each patient was evaluated with a detailed history regarding sociodemographic information, past medical history, menstrual history and any previous investigations for the diagnosis of PCOS. Physical examination with special emphasis on dermatological manifestations– such as hirsutism, acne, seborrhea, androgenetic alopecia, virilization, acanthosis nigricans, striae, skin tags and body mass index (BMI) was done. Hirsutism was scored according to modified Ferriman Gallwey (mFG) score which rates hirsutism on a scale from 0 to 36. [12] Clinical hirsutism was defined on a score of 8 or higher. It was classified as moderate if the

score was 8-15 and severe if the score was ≥15. Acne severity was evaluated using the criteria by Indian authors. [13] Grade 1: Comedones, occasional papules. Grade 2: Papules, comedones, few pustules. Grade 3: Predominant pustules, nodules, abscesses. Grade 4: Mainly cysts, abscesses, widespread scarring. AGA was evaluated using the Ludwig scale. [14] Virilization was diagnosed if the participant had a history of increased muscle mass, decreased breast size, deepening of voice or was found to have clitoromegaly.

The diagnosis of biochemical hyperandrogenemia was made based on the elevated levels of one or more of the following parameters: TT > 80ng/dL, or FT > 6pg/mL, or DHEAS > 350microgram/dL. [10] Estimation of follicle stimulating hormone (FSH), luteinizing hormone (LH), thyroid stimulating hormone (TSH), and fasting serum insulin levels were also done. All the patients underwent pelvic ultrasonography.

Statistical data was analyzed using SPSS 17. Continuous data was presented as mean +/- standard deviation. Associations between scores for diagnosis of clinical hyperandrogenism and serum androgen levels were analyzed using Spearman's rank correlation. The strength of association was measured by correlation co-efficient (r). A two-tailed P-value of <0.05 was considered statistically significant.

RESULTS

The present study comprised 71 female patients diagnosed to have PCOS. The demographic data of 71 women with PCOS are shown in Table 1. Maximum number of patients studied belonged to the age group of 20-25 years (43.66%). Youngest case was 15 years and oldest case was 40 years.

The participants had a mean age of 23.42 ± 5.19 years. The most common presenting symptom was oligomenorrhea or amenorrhea. Menstrual disturbances were seen in 59%, whereas 41% had a normal menstrual pattern. The most common menstrual disturbance noticed was oligomenorrhea, seen in 52%, followed by amenorrhea, seen in 8%. As per BMI, the prevalence of obesity and overweight in our study group was 28% and 52%, respectively, whereas 20% of our patients in the study group had normal weight. The majority of them were single.

Cutaneous manifestations of clinical hyperandrogenism are depicted in Figure 1. The most common presentation of clinical hyperandrogenism was hirsutism followed by acne, which was found in 67% and 65% of the participants respectively. Hirsutism was of moderate severity in 98%, and 2% of the patients had severe hirsutism. The mean mFG score observed was 7.72 ± 4.43. Among 47 cases of acne, majority of the cases (51%) had grade 2 acne followed by grade 3 in 26% of the cases, grade 1 in 21% and grade 4 in 2% of the cases. AGA was seen in 51 cases, amongst which 46% had grade 2 AGA, 43% had grade 1 AGA and 11% of the cases had grade 3 AGA.

Figure 2 shows the increased levels of biochemical hyperandrogenism in the patients of PCOS. Correlation of cutaneous features and biochemical parameters are enlisted in Table 2. Levels of total testosterone were raised in 66% with a mean of 80.13±24.30; free testosterone in 51% with a mean of 5.50± 3.54; and DHEAS in 31% with a mean of 282.56 ± 109.51. Ultrasonographic features of polycystic ovary was found in 72% of participants. The correlations between parameters of clinical and biochemical hyperandrogenism are shown in Table 3. There was a statistically significant correlation between the following pairs of clinical and biochemical hyperandrogenism: hirsutism and FT ($r=0.262$, $p<0.05$); hirsutism and DHEAS ($r=0.243$, $p<0.05$); acne and FT ($r=0.299$, $p<0.05$); and acne and TT ($r=0.40$, $p<0.05$). The other pairs had little or no correlation.

Fasting serum insulin level was increased in 47% of patients with a mean of 23.63 ± 17.12. While correlating clinical findings with fasting serum insulin, statistically significant correlation was observed only between acanthosis nigricans and fasting serum insulin ($r:0.294$, $p<0.05$).

Table 1. Demographic data of 71 women with PCOS

Demographic data		**Mean ± SD Or n (%)**
Age in years		23.42 ± 5.19
Age at Menarche		12.43 ± 1.08
Body Mass Index (kg/m2)		27.66 ± 4.03
Marital status	Married	25 (35)
	Unmarried	46 (65)

Table 2: Correlation of cutaneous manifestations with raised hormonal levels

Cutaneous features	**No. of patients**	**TT**	**FT**	**DHEAS**	**FSH**	**LH**	**LH: FSH**	**Insulin**
Hirsutism	48	25	26	24	3	2	18	25

Acne vulgaris	47	36	28	16	2	3	36	22
Androgenetic alopecia	37	22	14	11	2	1	10	18
Seborrhea	31	21	17	8	0	0	8	10
Acanthosis nigricans	17	12	8	4	1	2	16	16
Acrochordons	7	1	2	2	0	0	13	2

TT=Total testosterone; FT=Free testosterone; DHEAS=Dehydroepiandrosterone sulfate; FSH=Follicle stimulating hormone; LH=Luteinizing hormone

Table 3. Correlation between clinical and biochemical hyperandrogenism

	FT		**TT**		**DHEAS**	
	r score	**p value**	**r score**	**p value**	**r score**	**p value**
Hirsutism	0.262	**<0.05(0.027)**	-0.008(-0.107)	0.94(0.374)	0.243	**<0.05(0.041)**
Acne vulgaris	0.299	**<0.05(0.011)**	0.400	**<0.05(0.0005)**	0.062	0.607
AGA	0.214	0.072	0.183	0.126	0.001	0.99

AGA=Androgenetic alopecia; TT=Total testosterone; FT=Free testosterone; DHEAS=Dehydroepiandrosterone sulfate

Table 4: Prevalence of clinical features in PCOS among Indian and other studies

Name of the study-year (n)	**Menstrual irregularities**	**Obesity**	**Hirsutism**	**AV**	**AGA**	**Seborrhea**	**AN**	**Acrochordons**
Rajashekar et al.[20]- 2008 (1057)	623 (58.9%)	515 (48.7%)	-	-	-	-	-	-
Ramanand et al.[22]- 2013 (120)	120 (100%)	90 (75%)	53 (41.2%)	24 (20%)	8 (6.7%)	-	53 (44.6%)	-
Gowri et al.[23]- 2013 (40)	-	13 (32.5%)	25 (62.5%)	27 (67.5%)	12 (30%)	21 (52.5%)	9 (22.5%)	4 (10%)
Jayaram et al.[24] - 2016 (87)	29 (33.3%)	63 (72.4%)	36 (41.4%)	56 (64.3%)	6 (6.9%)	1 (1.1%)	41 (47.1%)	1 (1.1%)
Keen et al.25]- 2017 (100)	65 (65%)	80 (80%)	78 (78%)	48 (48%)	31 (31%)	29 (29%)	30 (30%)	9 (9%)
Sharma et al. [11] – 2019 (102)	68 (66.7%)	61 (59.8%)	76 (74.5%)	41 (40.2%)	31 (30.4%)	33 (32.4%)	50 (51%)	37 (36.3%)
Hassa et al [9]-2006 (66)	52 (78.8%)	-	31 (51%)	23 (38%)	3 (4.5%)	-	0	-
Schmidt et al [27] – 2016 (276)	-	-	144 (52.2%)	164 (59.4%)	53 (19.2%)	73 (26.4%)	89 (32.2%)	-
Leerasiri et al [10] – 2016 (145)	144 (99.3%)	-	41 (28.3%	82 (56.6%	63 (43.4%)	-	-	-
Present study (71)	42 (59%)	20 (28%)	48 (67%)	47 (65%)	37 (51%)	31 (43%)	17 (24%)	7 (10%)

PCOS=Polycystic ovary syndrome; AV=Acne vulgaris; AGA=Androgenetic alopecia; AN=Acanthosis nigricans

Table 5: Comparison of biochemical markers of hyperandrogenism in PCOS among Indian and other studies

Name of the study-year (n)	**Total testosterone**	**Free testosterone**	**DHEAS**

Rajashekar et al.[20]- 2008 (1057)	-	472 (44.64%)	687 (65%)
Ramanand et al.[22]- 2013 (120)	-	-	22 (18.3%)
Gowri et al.[23]- 2013 (40)	-	22 (55%)	18 (45%)
Jayaram et al.[24] - 2016 (87)	11 (18.6%)	-	6 (10.2%)
Keen et al.25]- 2017 (100)	28 (28%)	-	-
Sharma et al. [11] – 2019 (102)		35 (34.31%)	9 (8.82%)
Schmidt et al [27] – 2016 (276)	105 (40.7%)	-	-
Leerasiri et al [10] – 2016 (145)	65 (44.8%)	123 (84.8%)	30 (20.7%
Present study (71)	47 (66.1%)	36 (50.7%)	22 (30.98%)

PCOS=Polycystic ovary syndrome

Legends

Figure1: Prevalence of cutaneous manifestations in the study group of PCOS patients

Figure 2: Increased levels of biochemical hyperandrogenism in the study group of PCOS patients

DISCUSSION

PCOS (Stein–Leventhal syndrome) is a common hyperandrogenic disorder in women of the reproductive age group. The cutaneous manifestations of PCOS are key indicators of the underlying hormonal and metabolic dysfunctions associated with this complex endocrine disorder. [36]

It is a multisystem metabolic disorder, which has a significant impact on the quality of life as well as fertility. [15,16] PCOS is a complex disorder wherein numerous genetic variants and environmental factors interact, combine, and contribute to the pathophysiology. The key pathophysiologic components appear to include androgen excess, abnormal gonadotropin dynamics, and insulin resistance. Excess androgen production in the ovary impairs follicle maturation, leading to follicular atresia and decreased reproductive function. In addition, the resultant hyperandrogenemia may produce clinical hyperandrogenism. Whether due to an underlying primary hypothalamic defect in the gonadotropin releasing hormone (GnRH) pulse generator or a secondary effect of low levels of progesterone resulting from oligo- or anovulation, an increased pulse frequency of hypothalamic GnRH is thought to produce elevated levels of LH found in women with PCOS. [17,18] This increase in LH secretion relative to FSH stimulates production of androstenedione by ovarian theca cells. Insulin also plays a central role in PCOS pathophysiology, acting to increase androgen levels by direct and indirect mechanisms. [19]

Maximum number of cases in our study belonged to the third decade of life (62%), which agrees with relevant other studies. [20-26] The mean age of the cases was 23.42±5.19, which was similar to other studies that reported a mean age of 23.7±5.8 years by Hassa et al. [9] 25.18±3.61 years by Keen *et al.* [25] and 25.5± 6.5 years by Leerasiri et al. [10]

Reported prevalence of obesity among the participants of Indian studies of PCOS as per BMI ranged from 32.5 % [23] to 80%, [25] whereas in our study it was 52%. The mean BMI of the cases was 27.66 ± 4.03, which was similar to other studies that reported a mean BMI of 27.32±6 by Ramanand et al. [22] 26.1±5.2 by Hassa et al. [9] and 25.5±6.5 by Leerasiri et al. [10] In a systemic review and meta-analysis by Lim *et al.* [28] concluded that women with PCOS had a greater risk of overweight, obesity, and central obesity.

Menstrual disturbances were seen in 59% of the PCOS women in our study group, whereas 41% of the patients had normal menstrual cycles. Among these, oligomenorrhea was the most common menstrual disturbance, seen in 52%, followed by amenorrhea seen in 7% of the PCOS women in our study group. Keen *et al.* [25] reported menstrual disturbances in 65% and normal menstrual cycles in 35% of the PCOS women respectively. Among these, oligomenorrhea was seen in 57%, followed by amenorrhea seen in 8%. Ramanand *et al.* [22] reported irregular cycles in 100% of their study participants, whereas infertility was present in 21%.

Prevalence of clinical features in PCOS among Indian and other studies including our study is presented in Table 4. Most common presentation of clinical hyperandrogenism in our study was hirsutism (67%), other studies which have also reported hirsutism as the most common presentation of clinical hyperandrogenism are by Keen et al. [25] in 78%, Hassa et al. [9] in 51%, and Ramanand et al. [22] in 44.16%. In contrast, acne was reported as the most common cutaneous marker of hyperandrogenism by Gowri et al. [23] in 67.5%, by Schmidt et al. [27] in 61.2% and by Leerasiri et al. [10] in 56.6%.

Maximum mFG score in our patients was 18 with a mean ± SD of 7.72 ± 4.43. Keen *et al.* [25] reported maximum mFG score of 16 with a mean ± SD of 12 ± 2.44, while Schmidt *et al.* [27] reported mean mFG score of 8.6.

Comparison of biochemical markers of hyperandrogenism in Indian and other studies on PCOS including our study are presented in Table 5. Amongst the biochemical markers of hyperandrogenism in our study, TT was raised in 66%, FT in 51% and DHEAS in 31%, which was similar to other studies by Gowri et al. [23] who reported raised levels of TT in 55% and DHEAS in 45% and Schmidt et al. [27] who reported raised levels of TT in 41%, FT in 37% and DHEAS in 37%. In contrast to our study, TT and FT was found to be normal and DHEAS was raised in 31% in a study conducted by Ramanand et al. [22]

In our study, the mean levels of TT were 62.45 ± 24.36, mean levels of FT was 3.56 ± 2.23 and mean levels of DHEAS was 282.56 ± 109.51. Other studies conducted by Keen et al. [25] reported mean levels of TT as 58.59± 24.19 and mean levels of DHEAS as 124.34± 39.47 and Leerasiri et al. [10] reported mean levels of TT as 73.55 ± 38.77, mean levels of FT as 1.42 ± 0.99 and mean levels of DHEAS as 256.27 ± 107.27.

Ultrasonographic features of polycystic ovary were found in 72% of participants in our study. Sharma *et al.* [11] and Leerasisi *et al.* [10] reported ultrasonographic features of polycystic ovary in 79.41% and 81.4% respectively.

Acne, hirsutism, AGA, seborrhea, AN, skin tags and stria have strong association with PCOS. [37]
Hirsutism, the most common cutaneous manifestation in our study revealed a statistically significant correlation with raised FT (r=0.26, p=0.027) and raised DHEAS levels (r=0.243, p=041). Sharma et al. [11] also showed statistically significant correlation between hirsutism and raised FT (P = 0.012) and raised DHEAS (P = 0.016). Leerasiri et al. [10] reported a statistically significant correlation between hirsutism and raised FT (r=0.30, p<0.001) and raised TT levels (r=0.26, p<0.001). Schmidt et al. [27] also reported a statistically significant correlation between hirsutism and raised FT levels (p=0.04, <0.05). A meta-analysis done by Amiri et al. [29] reported a statistically significant correlation between hirsutism and raised DHEAS levels(p=0.22), whereas, no statistically significant correlation was reported between hirsutism and FT and TT. Hirsutism is the result of an interaction between plasma androgens and the apparent androgen sensitivity of the hair follicle, which is determined by the local metabolism of androgens (conversion of testosterone to dihydrotestosterone [DHT]). [30] By prolongation of the anagen phase and induction of vellus follicles to develop into terminal hair, androgens increase the hair shaft thickness and also the hair follicle size. [31]

In our study, there was a statistically significant correlation between acne and raised FT levels (r=0.299, p=0.011) and raised TT levels (r=0.400, p=0.0005), which was similar to another study conducted by Leerasiri et al. [10] who reported a statistically significant correlation between acne and raised TT levels (r=0.26, P=0.002). However, no statistically significant correlation was reported between acne and any of the hormonal parameters in studies done conducted by Sharma et al. [11] and Schmidt et al. [27] Androgens directly stimulate sebaceous glands and increase sebum secretion and also cause sebaceous gland hyperplasia. [32] Testosterone and 5 alpha-DHT, which act on the sebocytes via androgen receptors, are considered the most effective androgens in the pathogenesis of acne. [33]

There was no statistically significant correlation between AGA and biochemical parameters of hyperandrogenism in our study. Similar results were reported by studies conducted by Sharma et al. [11] and Leerasiri et al. [10] As AGA is caused by dihydrotestosterone, the patients might not have high circulating androgen levels. [10] Even though scalp hair loss is an essential feature of hyperandrogenism in women, several investigations failed to demonstrate the same. [34] Androgens cause loss of hair at one site (AGA) and overgrowth of hair at other site (hirsutism) and this variable response of hair follicles in different regions to androgens could be due to the difference in the sensitivity or the number of receptors or the difference in androgen metabolism in the hair follicles. [35]

Raised serum fasting insulin levels were seen in 49% of the cases in our study. Gowri et al. [23] and Schmidt et al. [27] reported raised fasting insulin levels in 67.5% and 36.9% respectively. The mean insulin levels in our study was 23.63 ± 17.12, while Leerasiri et al. [10] reported the mean insulin levels as 15.6± 34.2. We noted a statistically significant correlation between insulin levels and AN (r:0.294, p<0.05). However, this association has not been reported in other studies on PCOS. There was no statistically significant correlation between insulin and obesity or other cutaneous markers. The presence of AN in hyperandrogenic women depends on the presence and severity of hyperinsulinemia. The mechanism responsible for the development of AN is uncertain. Conflicting studies suggest mediation through various growth factor receptors, not just insulin or insulin-like growth factor. [22]

Limited Indian studies, [11,24,25] have investigated the correlation of cutaneous markers of hyperandrogenism with biochemical markers of hyperandrogenism. We found a statistically significant correlation between hirsutism and raised FT and DHEAS; acne and raised TT and FT; and acanthosis nigricans and raised serum fasting insulin levels. Hence, hirsutism and acne are good predictors for biochemical hyperandrogenism in women with PCOS in our study population. Early correction of biochemical hyperandrogenism will help to ameliorate the cutaneous markers and improve the quality of life of these patients.

Key Messages: Hirsutism and acne are the most reliable cutaneous markers of PCOS in our study population and are good predictors for biochemical hyperandrogenism. Early correction of biochemical hyperandrogenism will help to ameliorate the cutaneous markers and improve the quality of life of these patients.

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