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ORIGINAL ARTICLE

Cutaneous Manifestations in Patients with Polycystic Ovary Syndrome: Their Prevalence and Correlation with Hormonal and Metabolic Parameters

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ABSTRACT

Background: We aimed to determine the prevalence of dermatologic manifestations among an Egyptian PCOS cohort and investigate their correlation with hormonal and metabolic changes.

Methods: A cross-sectional study was conducted on 53 patients with PCOS and 50 non-PCOS controls. Focused dermatologic examination was performed to assess hirsutism, acne, androgenic alopecia (AGA), and acanthosis nigricans (AN) using clinical scoring. Serum levels of total & free testosterone (TT, FT), dehydroepiandrosterone sulfate, prolactin, and homeostatic model assessment for insulin resistance (HOMA-IR) were estimated for patients with PCOS, along with pelvic ultrasound.

Results: Among our cohort, a significantly higher prevalence of hirsutism, AGA, acne & AN was observed in the PCOS group compared to the non-PCOS group, with prevalence rates of 49%, 45.3%, 39.6%, and 32% versus 10%, 12%, 16%, and 12%, respectively. In PCOS, hirsutism is associated with greater TT and FT. A significant positive correlation exists between the degree of hirsutism and TT & FT (P = 0.0001 and P = 0.0007, respectively). No significant difference in any of the measured parameters in PCOS patients with/without AGA. Patients with PCOS who had acne were younger (P = 0.001). Women with PCOS and AN had higher anthropometrics, HOMA-IR, and serum prolactin.

Conclusions: In this Egyptian cohort, cutaneous manifestations were common in PCOS, providing clinical clues. Androgen levels can influence the occurrence & degree of hirsutism in PCOS. Acne and AGA in PCOS can be caused by factors other than hyperandrogenism and insulin resistance. AN in PCOS is related to the metabolic derangement.

Keywords: Polycystic Ovary Syndrome; Hirsutism; Androgenic Alopecia; Acne; Testosterone.

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a highly prevalent endocrine disorder in the female reproductive years with different phenotypes and heterogeneous manifestations, including reproductive, dermatological, and metabolic. [1] Such an extremely prevalent syndrome has a heavy burden on public health, coupled with long-term risks of developing diabetes mellitus, hypertension, dyslipidemia, cardiovascular diseases, and infertility.[2]

Ovarian dysfunction leads to the main signs and symptoms coined to this syndrome. The

ovary is affected by a combination of gonadotrophins, insulin, and several growth factors, which are dependent upon hereditary and environmental variables.[3] The biochemical abnormalities in PCOS include derangement in gonadotropin levels, increased androgens, both ovarian and adrenal, with or without increased insulin levels and markers of insulin resistance. [4]

Dermatologic manifestations, including acne, hirsutism, androgenic alopecia (AGA), and acanthosis nigricans (AN), primarily attributed to both hyperandrogenism and insulin resistance, can be the primary indicators, thus providing a clue for the underlying diagnosis of PCOS. Furthermore, cutaneous manifestations also play a role in disturbed self-image with subsequent anxiety and depression in some PCOS patients. [5,6]

It is well recognized that ethnic background plays an important role not only in determining the prevalence of PCOS in different societies, but also in the phenotypic distribution of cases and clinical manifestations. [7] As most of the available studies were conducted on non-Egyptian populations, we thought it was imperative to investigate the dermatologic signs of PCOS in Egyptian women with this disorder. Thus, our purpose was to elucidate the prevalence of relevant dermatologic manifestations in an Egyptian PCOS cohort and to investigate the correlation between each dermatologic manifestation and hormonal and metabolic alterations.

The current research aimed to estimate the prevalence of key cutaneous manifestations—including hirsutism, acne, androgenetic alopecia, and acanthosis nigricans—in women with PCOS compared to controls, and to explore their associations with hormonal and metabolic parameters.

METHODS

Study design and population:

This cross-sectional study involved 103 adult females aged between 20 and 40 years, divided into two groups: Group I included 53 patients diagnosed with PCOS, and Group II included 50 individuals who did not meet the criteria for PCOS, serving as the control group. The study was conducted from October 2018 to October 2019.

Participants were recruited at the Infertility Clinic, Alexandria University Hospital (Al-Shatby), Alexandria, Egypt.

For group I, the diagnosis was established according to the Rotterdam Consensus criteria of 2003, [8] which requires the identification of two out of the following three criteria: A) Amenorrhea or oligomenorrhea. B) Clinical and/or biochemical hyperandrogenism (HA). C) Polycystic ovarian (PCO) morphology assessed with ultrasonography. Group II consisted of individuals attending the infertility clinic for conditions other than PCOS, such as uterine polyps, fibroids, uterine septa, tubal adhesions, or male factor infertility.

Patients of group I were classified into four phenotypes according to the National Institute of Health (NIH) 2012: [9]

- Phenotype A (Classic PCOS): HA (clinical or biochemical), ovulatory dysfunction (OD), and PCO morphology (HA+OD+PCO).
- Phenotype B (non-PCO PCOS): HA and OD with normal ovarian morphology. (HA+OD)
- Phenotype C (ovulatory PCOS: HA and PCO (HA+PCO))
- Phenotype D (non-hyperandrogenic PCOS: OD and PCO (OD+PCO).

Exclusion criteria included participants who experienced features suggestive of other causes of hyperandrogenism, such as Cushing syndrome, congenital adrenal hyperplasia and androgen-secreting tumors. Patients with hyperprolactinaemia and thyroid dysfunction were ruled out. Patients receiving any drugs known to influence

cortisol or androgen levels, as well as pregnant and nursing females, were also excluded.

Demographic and clinical data: All participants underwent thorough history taking, including menstrual and obstetric histories; anthropometric measurements, including weight, height, waist circumference, and body mass index (BMI), calculated as weight (in kilograms) divided by height (in meters) squared. Detailed dermatologic manifestations, focusing on hirsutism, were assessed using the modified Ferriman-Gallwey score (mFGS); a score of 8 or greater is considered indicative of hirsutism. [10] Androgenic alopecia assessed by Ludwig's classification [11], acne evaluated by the global acne severity scale [12], and acanthosis nigricans defined as plaques of thickening and hyperpigmentation with velvety texture in body flexures, mainly the nape, the axillae, the antecubital fossae, and the groin. [13]

Hormonal analysis: Total testosterone (TT), free testosterone (FT), thyroid stimulating hormone (TSH), prolactin (PRL), dehydroepiandrosterone (DHEA-S), and fasting insulin with calculation of homeostatic model assessment of insulin resistance (HOMA-IR) according to the following equation [fasting glucose (mg/dL) fasting insulin (µU/mL)/405]. [14]

Transvaginal Pelvic ultrasound was performed for all enrolled cases.

Statistical analysis:

The data was entered and analyzed using SPSS version 26. Continuous variables were expressed as **means ± standard deviation**. Categorical variables were presented as **frequencies and percentages**. A comparison was made between patients with PCOS and those without, as well as between patients with and without individualized cutaneous manifestations, using **a Chi-square test or an independent t-test**. A correlation analysis was performed between individual cutaneous manifestations in the PCOS group and all studied demographic, clinical, hormonal, and metabolic parameters. Group comparisons utilized the Student t-test or the Mann–Whitney U test as appropriate. Correlations were analyzed using Spearman's method, with a linear fit used for figure display. Two-sided $\alpha=0.05$

Note: for composite cutaneous HA analysis, only AGA; Ludwig's ≥ 2 was used

Ethical considerations: The Ethics Committee approved the study at Alexandria University (IRB 00012098, Serial 0105667; date 20 Sept 2018). All participants received written informed consent after the nature and purpose of the study were explained.

RESULTS

Demographic and anthropometric characteristics of the two groups studied (I and II):

The mean **age** of patients with PCOS (group I) was 27.87 ± 5.68 years, and 31.18 ± 5.42 years in the control group (group II), with no significant difference between the two groups.

Based on WHO BMI categories, no participants were classified as underweight (BMI < 18.5 kg/m²). In the PCOS group (n = 53), normal weight was seen in 7/53 (13.2%), overweight in 18/53 (34.0%), and obesity in 28/53 (52.8%). In the control group (n = 50), the distribution was as follows: normal weight, 8 (16.0%); overweight, 24 (48.0%); and obesity, 18 (36.0%). The mean BMI of group I was 30.54 ± 5.46 compared to 29.01 ± 4.11 kg/m² in group II ($p>0.05$). Overall, the mean BMI for groups I and II was 30.54 ± 5.46 and 29.01 ± 4.11 kg/m², respectively, with no significant difference between the two groups.

As regards **waist circumference**, the mean values were 99.36 ± 12.31 cm and 98.7 ± 9.7 cm for groups I and II, respectively, with no significant difference. The measurements

ranged from 70 to 140 cm in group I and from 80 to 130 cm in group II.

Distribution of PCOS patients (group I) according to the phenotype classification of NIH 2012:

Analysis of the clinical and laboratory data of the studied PCOS cohort revealed that 30 patients belonged to phenotype A (56.6%), two patients belonged to phenotype B (3.8%), seven patients were of the ovulatory phenotype C (13.2%), and 14 patients were normoandrogenic and belonged to phenotype D (26.4%). **Table supplementary 1 (S1)**

Biochemical and clinical hyperandrogenism in PCOS patients (group I):

Among the PCOS patients (group I), total and free testosterone were elevated in 24/53(45.3%) patients, with a mean value of 0.47 ± 0.22 ng/ml and 5.84 ± 3.83 pg/ml, respectively. The DHEA-S level was elevated in 3 patients (5.6%), with a mean value of 230.85 ± 97.19 µg/dL.

According to clinical and biochemical findings, it was observed that 10/53 patients (19%) showed neither clinical nor laboratory evidence of hyperandrogenism, 17/53 patients (32%) showed only clinical hyperandrogenism, 3/53 cases (6%) showed only biochemical hyperandrogenism, and 23/53 patients (43%) were proven to have both clinical and biochemical hyperandrogenism.

Cutaneous manifestations in the two studied groups, groups I & II, are shown in **Table 1.**

It was found that the most common dermatological manifestation in patients with PCOS was hirsutism, followed by AGA, acne, and acanthosis nigricans, with prevalence rates of 49, 45.3, 39.6, and 32%, respectively. The prevalence rates of hirsutism, acne, AGA, and AN in the non-PCOS group (Group II) were 10%, 16%, 12%, and 12%, respectively. There was a significantly higher prevalence of cutaneous manifestations in the PCOS group (Group I) versus the non-PCOS Group (Group II).

Regarding patients with PCOS (group I), 20/26 patients (77%) with hirsutism had mFGS scores of 8-<15, while 23% (6/26) of patients had an mFGS score of 15 or more. Eighteen out of 21 (85.7%) patients with acne had a grade 1, and 3 out of 21 patients (14.3%) were suffering from a grade 2, and none had grade 3, based on the global acne severity score. 18/23 (78.2%) of patients with AGA had stage I, and 5/23 (21.8%) were classified as stage II on Ludwig's scale.

Correlation between individual cutaneous manifestations in PCOS and demographic, hormonal, and metabolic parameters

Hirsutism

Among group I, comparing women with **hirsutism** versus those without hirsutism revealed no significant differences as regards age, BMI, waist circumference, DHEA-S, and HOMA-IR. On the other hand, the mean values of serum total and free testosterone were significantly higher in hirsute women (p = 0.001, p = 0.0095, respectively). Furthermore, Spearman correlation analysis showed a positive correlation was found between mFGS and the serum levels of total and free testosterone (p = 0.0001 and 0.0007, respectively). (**Table 2**, **Figures 1 & 2**)

Acne

It was shown that the mean age of PCOS women with **acne** was significantly lower than that of acne-free women: 24.52 ± 4.19 versus 30.06 ± 5.5 years (p = 0.001). The mean values of the other measured parameters showed no significant difference between women with acne and acne-free PCOS women. (**Table 2**)

Androgenic alopecia

Comparing PCOS women with **AGA** and those without AGA revealed that there is no significant difference regarding all measured parameters. (**Table 2**)

Acanthosis nigricans

The mean BMI was 33.84 ± 6.55 & 28.98 ± 4.11 kg/m², and the mean WC was 104.94 ± 14.38 & 96.72 ± 10.42 cm in women with **AN** and women without AN, respectively, which was significantly higher for BMI only (p = 0.017). Furthermore, the mean value of the measured HOMA-IR was higher in the AN patient (p = 0.002). Serum prolactin levels were significantly higher in patients with AN than in those without AN (p = 0.036). Age, total testosterone, free testosterone, DHEA-S, and TSH levels showed no significant difference between PCOS women with and without AN. (**Table 2**)

Note: Based on our data, Hirsutism: Clinically (mFG) correlates with biochemical androgens—higher TT and FT (both significant); mFG severity positively correlates with TT and FT. Acne: More common in younger women with PCOS; no considerable link with TT/FT, DHEA-S, BMI, or HOMA-IR in this sample. AGA: No significant biochemical or metabolic differences between women with AGA versus those without AGA within the PCOS group. Acanthosis nigricans: Associated with metabolic parameters—higher BMI, waist circumference, HOMA-IR—and higher prolactin; not linked to TT/FT or DHEA-S. Therefore, hirsutism appears to be more driven by androgens; AN reflects metabolic derangements and insulin resistance; and acne and AGA likely involve additional local or genetic factors beyond systemic androgens/IR in this cohort.

Comparison between PCOS patients with and without hyperandrogenic cutaneous manifestations:

A comparison was made between PCOS women with one or more cutaneous hyperandrogenic manifestations (hirsutism, acne, or androgenetic alopecia), 40/53 (75.5%) patients, and those without any hyperandrogenic cutaneous symptoms, 13/53 (24.5%) patients.

A higher mean total testosterone level was observed in the group of PCOS patients with cutaneous manifestations compared to those without (0.50 ± 0.24 vs. 0.39 ± 0.08). Additionally, the mean free testosterone level was higher in the group with cutaneous manifestations than in those without (6,25 ± 4.27 vs. 4.6 ± 1.46). However, the difference was not statistically significant. Furthermore, the other hormones measured showed no significant difference between the two groups. **Table 3**

Table 1: Cutaneous Manifestations in PCOS vs Non-PCOS

		PCOS n=53	Non-PCOS n=50	Fisher test
		No. (%)	No. (%)	P-value
Hirsutism (mFGS≥8)		**26 (49.1%)**	**5 (10%)**	**0.000**
Acne	**Grade1**	**18 (34.0%)**	**6 (12%)**	**0.014**
	Grade2	**3 (5.7%)**	**2 (4%)**	**1**
	Grade3	**0**	**0**	**1**
Total		**21 (39.6%)**	**8 (16%)**	**0.009**
Androgenetic alopecia (Ludwigs's scale)	**I**	**19 (35.8%)**	**6 (12%)**	**0.014**
	II	**5 (9.4%)**	**0**	**0**
	III	**0**	**0**	**1**
Total		**24 (45.3%)**	**6 (12%)**	**0.0002**
Acanthosis nigricans		**17 (32%)**	**6 (12%)**	**0.018**

Footnotes: P-values are from Fisher's exact test. $p \leq 0.05$ indicates significance. Acne Total and AGA Total categorize any grade versus none. mFGS: modified Ferriman–Gallwey score (threshold ≥8). PCOS: Polycystic ovary syndrome; n=number

Table 2: Correlation between cutaneous manifestations in PCOS patients (Group I, n=53) and demographic, clinical, hormonal, and metabolic parameters PCOS cases n=53

Parameter	Hirs Yes (n=26)	Hirs No (n=27)	p_hirs	Acne Yes (n=21)	Acne No (n=32)	p_acne	AGA Yes (n=24)	AGA No (n=29)	p_AGA	AN Yes (n=17)	AN No (n=36)	p_AN
Age (years)	28.23 ± 6.13	27.52 ± 5.30	0.708	24.52 ± 4.19	30.06 ± 5.49	<0.001	27.00 ± 5.18	28.59 ± 6.06	0.425	27.59 ± 5.93	28.00 ± 5.64	0.774
BMI (kg/m²)	30.16 ± 5.05	30.91 ± 5.89	0.398	29.22 ± 6.01	31.40 ± 4.97	0.184	31.24 ± 6.30	29.96 ± 4.69	1.000	33.84 ± 6.55	28.98 ± 4.11	0.017
WC (cm)	96.65 ± 13.62	101.96 ± 10.51	0.033	96.05 ± 12.46	101.53 ± 11.91	0.282	100.17 ± 14.72	98.69 ± 10.12	0.809	104.94 ± 14.38	96.72 ± 10.42	0.084
TT (ng/mL)	0.56 ± 0.24	0.38 ± 0.16	<0.001	0.48 ± 0.28	0.46 ± 0.18	0.978	0.48 ± 0.23	0.47 ± 0.22	0.922	0.47 ± 0.24	0.47 ± 0.22	0.789
FT (pg/mL)	7.00 ± 4.46	4.73 ± 2.75	0.009	6.53 ± 4.56	5.39 ± 3.26	0.548	6.14 ± 4.24	5.59 ± 3.51	0.851	6.03 ± 4.34	5.76 ± 3.63	0.939
DHEA-S (µg/dL)	242.75 ± 94.15	219.39 ± 100.45	0.551	255.41 ± 109.92	214.73 ± 85.87	0.292	243.11 ± 114.77	220.70 ± 80.54	0.592	202.77 ± 66.74	244.11 ± 106.93	0.261
HOMA-IR	1.32 ± 0.88	1.61 ± 1.34	0.215	1.29 ± 0.61	1.59 ± 1.37	1.000	1.73 ± 1.34	1.26 ± 0.91	0.027	2.15 ± 1.58	1.15 ± 0.67	0.002
TSH (mIU/L)	4.146 ± 2.004	2.57 ± 1.58	0.756	2.16 ± 0.94	3.444 ± 1.805	0.592	2.30 ± 1.08	3.766 ± 1.897	0.823	2.36 ± 1.55	3.075 ± 1.702	0.746
PRL (ng/mL)	14.35 ± 6.50	15.21 ± 7.63	0.943	14.83 ± 7.71	14.76 ± 6.69	0.849	15.14 ± 7.84	14.50 ± 6.44	0.809	17.57 ± 6.86	13.48 ± 6.83	0.036

Footness: Mann–Whitney U tests for continuous variables. Hirsutism is defined as mFGS ≥8. Acne = any grade (G1–G3). AGA = any Ludwig grade (I–III). AN = acanthosis nigricans present. $p \leq 0.05$ indicates significance. BMI = body mass index; WC = waist circumference; TT = total testosterone; FT = free testosterone; DHEAS = dehydroepiandrosterone sulfate; HOMA-IR = homeostatic model assessment for insulin resistance; TSH = thyroid-stimulating hormone; PRL = prolactin.

Table 3: Comparison between PCOS patients with one or more cutaneous hyperandrogenic manifestations versus those without

Parameter	With cutaneous HA (n=40)	Without cutaneous HA (n=13)	Mann–Whitney p
Total testosterone (ng/mL)	0.50 ± 0.24	0.39 ± 0.08	0.092
Free testosterone (pg/mL)	6.25 ± 4.27	4.60 ± 1.46	0.301
DHEAS (µg/dL)	241.16 ± 98.33	199.14 ± 89.83	0.482
Prolactin (ng/mL)	14.69 ± 7.22	15.07 ± 6.73	0.836
TSH (mIU/L)	2.09 ± 0.98	3.13 ± 1.91	0.090
HOMA-IR	1.50 ± 1.17	1.38 ± 1.05	0.967

Footnotes: Hirsutism (mFGS ≥8), Acne (any grade), AGA (Ludwig ≥ II). AGA stage I alone is not counted as cutaneous hyperandrogenism. With HA n=40; without HA n=13. P-values are shown for the Mann-Whitney U test. $p \leq 0.05$ indicates significance. PCOS = polycystic ovary syndrome; HA = hyperandrogenism; mFGS = modified Ferriman–Gallwey score; AGA = androgenetic alopecia; AN = acanthosis nigricans; BMI = body mass index; WC = waist circumference; TT = total testosterone; FT = free testosterone; DHEAS = dehydroepiandrosterone sulfate; HOMA-IR = homeostatic model assessment for insulin resistance; TSH = thyroid-stimulating hormone; PRL = prolactin.

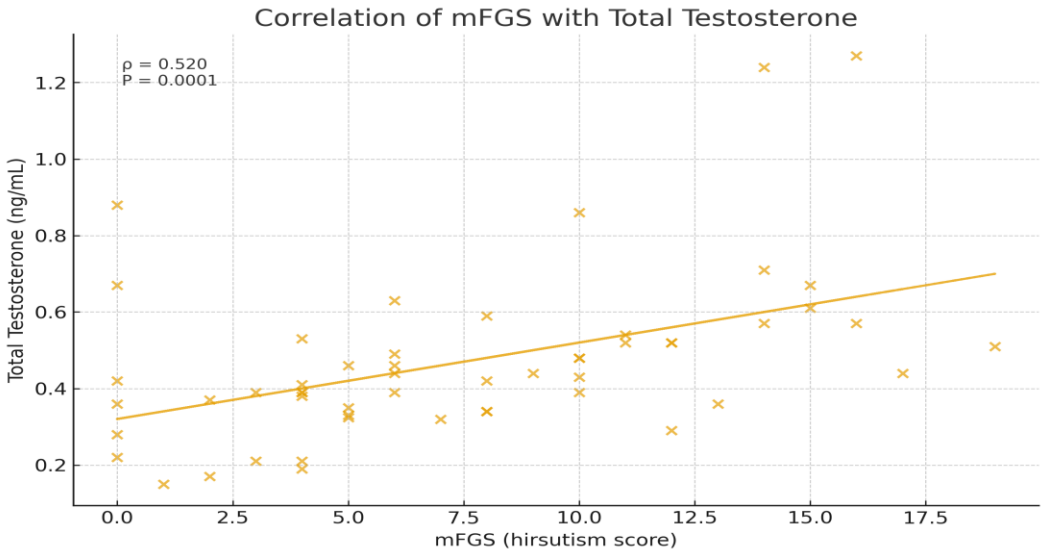

Figure (1): Correlation between mFGS and total testosterone in PCOS group (p=0.520, p= 0.0001)

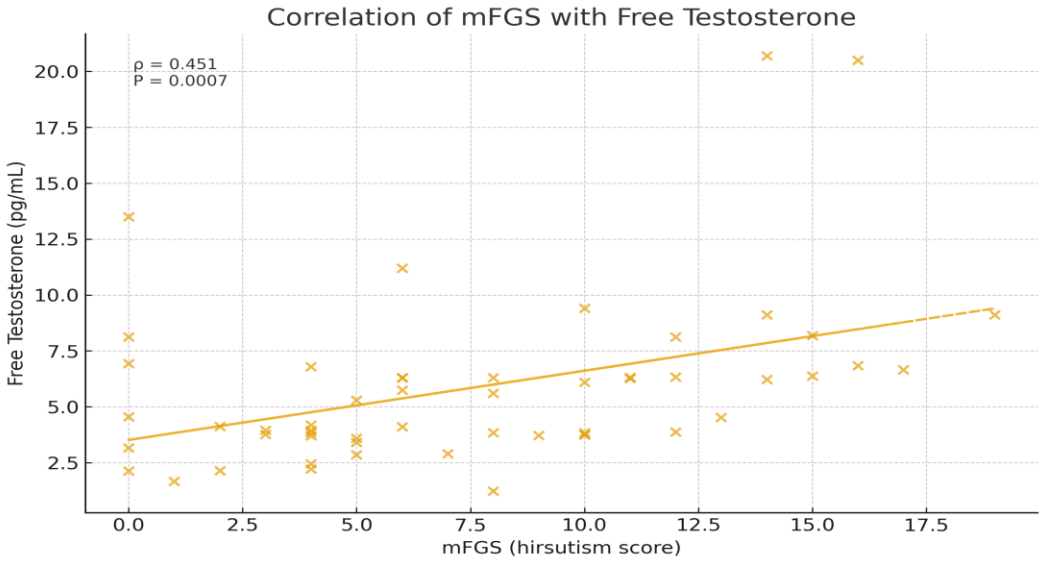

Figure (2): Correlation between mFGS and free testosterone in PCOS group (p = 0.451, p= 0.0007)

DISCUSSION

Polycystic ovary syndrome is a common syndrome with heterogeneous presentation, primarily comprising ovarian dysfunction, hyperandrogenism, and PCO morphology. [15] These elements constitute the basis of its clinical presentation, resulting in different phenotypes with various clinical, metabolic, and hormonal milieus. The present study revealed that the majority of PCOS patients belonged to phenotype A (classic, hyperandrogenic), constituting 64.1%, followed by phenotype D (the normoandrogenic phenotype) at 18.9%. Phenotype C (hyperandrogenic, ovulatory with regular cycles) at 13.2%, and the least common was phenotype B (classic, hyperandrogenic but with normal ovarian

morphology) at 3.8%. This distribution closely aligns with what was reported by the Androgen Excess – Polycystic Ovarian Syndrome Society (AE-PCOS), which indicates that approximately 75% of referred PCOS patients exhibit classic PCOS (phenotypes A & B), while the remaining 25% are evenly distributed between the ovulatory and normoandrogenic PCOS phenotypes. [9]

Sachdeva et al. [16] reported that phenotype A was the most common phenotype in their study, accounting for almost two-thirds of the enrolled sample. In contrast, phenotype D was the least common presenting phenotype. In addition, he concluded based on his research that phenotype A is coupled with an elevated risk of metabolic and hormonal derangements, displaying the worst outcomes when treated with ovarian-stimulating agents.

Hyperandrogenism in the present study was defined either clinically, biochemically, or both (according to the Rotterdam consensus criteria 2003). [8] Having both clinical and biochemical hyperandrogenism was observed in 43% of PCOS patients, cutaneous signs of hyperandrogenism without biochemical elevation in 32%, and isolated biochemical hyperandrogenemia in 6%. Overall, cutaneous hyperandrogenism occurred in 75.5%, while biochemical hyperandrogenism was detected in 45% of PCOS patients. On the other hand, 19% of PCOS patients did not show clinical or biochemical evidence of hyperandrogenism and are still being diagnosed as PCOS according to the Rotterdam criteria. These observations draw significant clinical implications; that clinical hyperandrogenism can manifest despite normal levels of androgens, which indicates that androgen levels are not the only contributors to the cutaneous manifestations encountered in PCOS. It seems that other factors lead to these manifestations, including insulin resistance, where studies have shown a positive correlation between clinical scoring of hirsutism and HOMA-IR levels. In addition, insulin and insulin growth factors exert a dose-dependent relationship on hair cycle growth. Moreover, the sensitivity of the pilosebaceous gland and the hair follicle to the circulating androgens rather than the androgen level itself is an essential determinant in the causation of hirsutism, acne, and AGA. [17] The second important implication is that biochemical hyperandrogenism without clinical evidence is less likely to be encountered in clinical practice.

In the present study, one or more cutaneous manifestations of hyperandrogenism occur in almost three-quarters of PCOS patients. Higher prevalence rates were reported by Gowri et al. [15], who reported a 90% prevalence rate. In contrast, a Jordanian study reported a 100% prevalence rate, emphasizing that dermatological manifestations serve as early indicators of PCOS and are relatively prevalent. [18]

The most prevalent dermatologic manifestation in our PCOS patients' sample was hirsutism at 49%, followed by AGA at 45.3% and then acne at 39.6%. Acanthosis nigricans was the least common disorder, at 32%. There was a significant difference regarding the prevalence of the four manifestations between the patient and control groups. In the control group, acne was the most common manifestation, occurring in 16% of cases, followed by AGA and acanthosis nigricans, which were equally common at 12% each. The least common manifestation was hirsutism, occurring in 10% of cases.

Schmidt et al. [19] observed a higher prevalence of cutaneous manifestations in patients meeting PCOS criteria compared to those who did not, in agreement with our findings. However, he found a higher prevalence of acne and lower rates of AGA

among his studied population, which contrasts with our results.

The order of frequency of dermatologic signs in patients with PCOS is different according to the studied population, reflecting that ethnic and racial differences play essential roles. Whereas most of the studies reported that acne was the most common dermatological manifestation in patients with PCOS, as reported by Abusailik et al. [18], Feng et al. [20], and Gowri et al. [15] On the other hand, Ozedemir et al. [21] and Aljefri YE et al. [4] reported a higher prevalence of hirsutism in their studies than that observed in our sample studied. The predominance of hirsutism or acne in the clinical presentation of PCOS may stem from distinct underlying mechanisms, with acne more closely associated with insulin resistance and hirsutism more directly linked to biochemical hyperandrogenism. [19]

In the present study, the prevalence of hirsutism in PCOS patients was 49% and 10% in the non-PCOS group. The figure of hirsute females in patients with PCOS aligns with the figure reported by Schmidt et al. [19], revealing a prevalence of 53.3% in a racially diverse group of patients. Other authors recorded a higher prevalence of hirsutism in their PCOS study populations, accounting for 58.1% in a Chinese study and 62.5% in an Indian study. [15]

Given that hirsutism is a significant sign of hyperandrogenism, [10] it was found in the present study that patients with hirsutism had higher levels of serum total and free testosterone as compared to patients without hirsutism, in agreement with previous studies. [19,20,22] However, Feng et al. [20] have revealed that PCOS patients with hirsutism, in contrast to patients without, were younger and experienced higher BMI and DHEAS serum levels. Schmidt et al. [19] noticed that patients experiencing hirsutism also had higher BMI, HOMA-IR, free and total testosterone levels, elevated triglycerides, and lower HDL-C levels, suggesting that hirsutism reflects both hormonal and metabolic derangement encountered in patients with PCOS.

The correlation between the severity of hirsutism (recognized by mFGS) and the levels of total and free testosterone in the sera of PCOS patients revealed a significant positive correlation. Consistent with the results reported by Panidis et al. [23]

Hirsutism results from either increased androgen levels or enhanced follicle sensitivity to circulating androgen. Women with hirsutism exhibit increased follicular-based 5α-reductase activity, which mediates the conversion of testosterone to the more potent dihydrotestosterone (DHT), a hormone that stimulates hair growth. This might explain why hirsutism does not correlate well with serum levels of androgens. [3] Moreover, it has been suggested that altered expression levels of androgen receptors within the pilosebaceous units may result in increased action in an otherwise normoandrogenic environment. [23]

In the present study, androgenetic alopecia is the second most common cutaneous manifestation, accounting for 45.3% of the PCOS group and 12% of the control group. This prevalence is higher than what was reported in previous studies, with percentages ranging from 3.7% to 34.8%. [15, 19, 20, 24], which might be attributed to ethnic differences. Regarding the severity of hair loss on Ludwig's scale, 33.9% had alopecia at stage I, and 9.4% had stage 2; none were recorded as having grade 3.

In agreement with previous studies [15, 19, 20, 24], AGA, when present in PCOS patients, was not associated with higher serum androgen levels compared to PCOS patients without AGA, suggesting that it is not a marker for biochemical hyperandrogenemia. [21] Genetic and local

factors are certainly operative in association with the clinical setting of PCOS. [1]

In general, the pathophysiology of AGA remains poorly understood. Studies have shown a wide range of prevalence of androgen excess in female pattern hair loss, making the association of low strength. Enhanced androgen action may be mediated by increased local production of DHT or increased androgen binding to its receptors. [25]

Moreover, the present study showed no significant difference regarding age, BMI, waist circumference, and HOMA-IR between PCOS women with and those without AGA. This agrees with the work of Schmidt et al. and Aljefri et al. [4,19]. On the other hand, Feng et al. [20] and Abusailik et al. [18] reported higher ages among PCOS patients with AGA, consistent with previous reports that the prevalence of female pattern hair loss increases with age in general.

The next common manifestation of hyperandrogenism in PCOS, noted in our work, was acne, which was seen in 39.6% of PCOS patients and 16% of the non-PCOS control subjects, with a significant difference between the two groups. A wide variation in the prevalence of acne has been reported by different studies, ranging from 15% to 95%. Our results were close to what was reported by Aljefri et al. [4], who observed a prevalence rate of 40.6%; however, our acne prevalence rates were much lower than previous studies made by Schmidt et al., Gowri et al., and Abusailik et al. [15,18,19] who reported a prevalence rate of 61.2, 67.5, and 75% in their PCOS study groups, respectively. However, these studies have documented the high prevalence of acne among their non-POS control groups, ranging from 40% to 44%. [15,18] This finding indicates that these variations could be related to the clinical setting from which patients and control groups were recruited. Additionally, other confounding factors might be involved, including stress, smoking, obesity, dietary habits, ethnic and racial backgrounds, and cosmetic applications. [26]

In the present study, among the PCOS patients' group, 34% experienced only comedones and a few papules (grade 1), and only 5.6% had papules and pustules (grade 2). This is consistent with the results of Feng et al. [20] and Hong et al. [24], who reported that the majority of PCOS acne patients had mild to moderate acne. This observation contrasts with what was previously known about adult female acne: that it is more inflammatory, with a high incidence of scarring.[3]

Patients with PCOS suffering from acne were significantly younger than patients who were free of acne, with a significant difference between them. This agrees with what was reported by Schmidt et al. [19], who concluded that the presence of acne was associated with younger age, affirming that, in general, acne vulgaris has a lower prevalence in the older age group. It seems that PCOS does not constitute an exception.

In the current investigation, we didn't find a significant correlation between the presence of acne and the metabolic and hormonal parameters studied, consistent with the findings of multiple studies [19,24], indicating that the association between acne and hyperandrogenemia is sophisticated and that the presence of acne does not uniformly indicate androgen excess. On the other hand, a Chinese study has reported higher BMI in PCOS women with acne than those without acne. [20]

It is well established in the literature that women with PCOS have a higher prevalence and a greater degree of hyperinsulinemia and insulin resistance than weight-matched control subjects, with an increased risk of obesity, a contributing factor causing psychological distress and depression

frequently associated with this syndrome. [27] In the present study, AN was the least frequent cutaneous association (32% in the PCOS patients' group and 12% of the control group), with a significant difference between the two groups. This finding is consistent with other studies conducted by Schmidt et al. [19], who reported a prevalence rate of 36%, and Keen et al. [28], who reported a prevalence of 30%. While others reported lower prevalence rates, as stated by Feng et al. [20] and Gowri et al. [15], who reported rates of 15.8 and 22.5%, respectively.

According to the present study, PCOS women with AN had a higher BMI, waist circumference, and HOMA-IR when compared to PCOS women without AN. These results are expected because the primary suggested pathogenic mechanism of AN is mediated by insulin resistance, with a compensatory increase in insulin levels, which stimulates IGF-1 receptors present in keratinocytes and fibroblasts, inducing their proliferation. [13]

Dong et al. [29] conducted a study on 339 women with PCOS and a normal BMI (<23 kg/m2) to exclude the effect of obesity, and reported that 9.7% had AN, which was associated with elevated HOMA-IR levels. They concluded that, as a dermatological manifestation, AN could be a reliable marker of insulin resistance; however, it lacks the sensitivity to become a non-invasive diagnostic screening tool for insulin resistance in women with a normal BMI.

Schmidt et al. [19] also added that AN, when present in PCOS patients, was associated with elevated free testosterone levels in the serum. This was explained by the stimulatory effect of elevated insulin levels on ovarian androgen production. [30] However, in the present study, serum androgen levels showed no significant difference between PCOS patients with and those without AN.

Mild hyperprolactinemia has long been associated with PCOS, with a reported prevalence of 5% probably related to low dopaminergic tone encountered in this syndrome. [31] We didn't find a significant difference in mean prolactin levels in patients with or without cutaneous manifestations with hyperandrogenism. However, in the present study, prolactin was found to be higher in PCOS women with AN versus those without. An association between prolactin and insulin sensitivity, obesity, and altered glucose and lipid metabolism has been proposed in the literature. [32,33] Multiple studies have noted an association between lowering prolactin levels using bromocriptine or even adenectomy and an improvement in insulin sensitivity.[34] Some authors explained this association by suggesting that chronic elevation of prolactin causes increased food intake and weight gain, or affects the CNS regulation of glucose metabolism, or that prolactin acts as an adipokine released from visceral fat. [35,36] However, these explanations were debated by other studies. [37]

Strengths and Limitations:

Our study had several strengths; we utilized validated, standardized scoring methods (mFG, Ludwig, global acne grading) alongside comprehensive hormonal and metabolic profiling (TT, FT, DHEAS, prolactin, TSH, HOMA-IR) and Rotterdam/NIH phenotyping. A concurrent control group with similar age and BMI reduces confounding factors. Additionally, we implemented strict exclusion criteria (such as other hyperandrogenic disorders, thyroid dysfunction, hyperprolactinemia, pregnancy/lactation, and interfering medications) to ensure the validity of results. The project addresses a geographic evidence gap by systematically collecting data from an Egyptian cohort and establishes a reproducible foundation for the planned

multicenter extension. Our limitations include that this single-center study, with a modest sample size (PCOS, n = 53; controls, n = 50), was sufficiently powered only to detect moderate to large prevalence differences. Although the differences between groups were large for hirsutism and androgenetic alopecia (AGA), the estimates for acne and acanthosis nigricans (AN)—despite being statistically significant—had broader confidence intervals and should be considered preliminary. We were unable to perform phenotype-specific (A–D) comparisons because the counts per phenotype, especially for phenotype B, were too small. Clinical photographs were not included because our ethical approval letter did not cover the publication of identifiable images; additionally, several comparable epidemiologic cohorts have reported standardized scoring without patient photos. Therefore, to improve external validity, a multicenter study is recommended, recruiting participants from additional Egyptian governorates.

CONCLUSIONS

In this Egyptian cohort, cutaneous manifestations were common in PCOS. Hirsutism was linked to biochemical hyperandrogenism, while acne and androgenic alopecia showed no consistent biochemical association, suggesting other mechanisms. Acanthosis nigricans correlated with adiposity and insulin resistance, emphasizing a metabolic pathway.

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Supplementary material: Table supplementary 1 (S1)

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Supplementary material:

Table supplementary 1 (S1): NIH 2012 PCOS Phenotype Distribution

Phenotype	n	% of PCOS (n=53)
Phenotype A (HA + OD + PCO)	**30**	**56.6%**
Phenotype B (HA + OD)	**2**	**3.8%**
Phenotype C (HA + PCO)	**7**	**13.2%**
Phenotype D (OD + PCO)	**14**	**26.4%**

Definitions per NIH 2012: HA = hyperandrogenism (clinical and/or biochemical); OD = ovulatory dysfunction; PCO = polycystic ovarian morphology on ultrasound.

Abbreviations: PCOS = polycystic ovary syndrome; HA = hyperandrogenism; OD = ovulatory dysfunction; PCO = polycystic ovary.

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