

Study of the Prevalence of Different Causes of Hair Loss among Females with Polycystic Ovary Syndrome in Delta Area-Egypt

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is a common endocrinal disorder of reproductive aged females. There are many causes of hair loss that could affect females including androgenetic alopecia (AGA), alopecia areata (AA), telogen effluvium (TE), trichotillomania (TTM) and lichen planopilaris (LPP).

Objective: To assess the prevalence of different causes of hair loss among females with PCOS in Delta area-Egypt.

Patients and Methods: This observational cross-sectional study included 246 patients with PCOS and were classified into 5 groups, group (A) included patients with PCOS with no hair loss (normal) hair, group (B) included patients with PCOS and AGA, group (C) included patients with PCOS and AA, group (D) included patients with PCOS and other causes of hair loss and group (E) included patients with more than one type of causes of hair loss.

Results: There were statistically significant associations between AGA and the causes of hair loss and menstrual irregularities ($p=0.005$), puberty onset ($p<0.001$), and family history. No significant associations were detected between groups with hair loss regarding acne vulgaris (AV), seborrhea, hirsutism, high-pitched voice, decreased breast mass, masculine body and body hair hypertrichosis ($p>0.05$) except for hirsutism was more in TE and psychological disturbances were more in TTM.

Conclusion: Hirsutism, AV, AGA and seborrhea have strong association with PCOS in current study. AGA is accompanied by other manifestations of clinical hyperandrogenism but doesn't seem to be accompanied by greater risk of biochemical alterations than PCOS alone. AGA is the most commonest prevalent clinical manifestation of hair loss in our study.

Keyword: Polycystic Ovary Syndrome, Hyperandrogenism, Androgenetic Alopecia, Alopecia Areata, Trichotillomania.

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a complex phenotype that presents heterogeneous manifestations that differ by time. The most recent diagnostic criteria for Rotterdam diagnosis of PCOS is presence of two of the next criteria: Oligo/anovulation, hyperandrogenism (clinical: hirsutism or biochemical: raised free testosterone or free androgen index) and polycystic ovaries on ultrasound. Other etiologies have to be ruled out which include congenital adrenal hyperplasia (CAH), androgen secreting tumours, Cushing syndrome, impaired thyroid functions and hyperprolactinaemia⁽¹⁾.

Some women with PCOS have some clinical manifestations of hyperandrogenism disorders including AGA, AV and hirsutism, there are many forms of hair loss in females as: AGA, AA, anagen effluvium, TE, traction alopecia⁽²⁾.

AGA is the most frequent form of extensive hair loss in PCO. AGA characterizes an extensive miniaturization of the hair follicle causing vellus transformation of terminal hair. The Ludwig classification uses three phases to define AGA: type I (mild), type II (moderate), and type III (extensive). The top and front of the scalp experience hair loss in all three phases, whereas the frontal hairline is comparatively preserved⁽³⁾.

Alopecia areata (AA) is an inflammatory disease characterized by a sudden onset of non-scarring hair loss⁽⁴⁾. Trichotillomania is a psychiatric problem characterized by repeated pulling out of one's hair

causing obvious hair loss and accompanying impairment/distress in spite of trials to reduce or stop pulling⁽⁵⁾. TE is a form of non-scarring alopecia characterized by diffuse hair shedding, often with an acute onset. In addition, a chronic form with a more insidious onset and a longer duration exists⁽⁶⁾. Lichen planopilaris (LPP) is a scarring form of alopecia that mainly occurs in postmenopausal females. The typical lesion presents on the scalp as scaly erythematous plaques with perifollicular erythema follicular hyperkeratosis, and irreversible hair loss⁽⁷⁾. Among the different patterns of hair loss, AGA is most strongly accompanied by PCOS. The primary involvement of androgens, specifically dihydrotestosterone (DHT), in the processes leading to AGA is well-established; it causes the hair follicle to shrink and terminal hair to change into a vellus⁽⁸⁾.

This study was done to assess the prevalence of different causes of hair loss among females with PCOS in Delta area-Egypt.

PATIENTS AND METHODS

This observational cross-sectional study included 246 patients with PCOS. They were chosen from Gynecology and infertility outpatient clinics, Gynecology Department, Mansoura University Hospital, Egypt, during the study period, which was 12 months between March 2022 and February 2023. This study included female patients in child bearing period between (17 – 45 years old) diagnosed as PCOS based on the Rotterdam diagnostic criteria in which PCOS

was defined by the presence of two of the next criteria: oligo-anovulation, hyperandrogenism and polycystic ovaries in ultrasonic radiology⁽⁹⁾.

But we excluded patients with history of taking drugs that causes hair loss as (chemotherapeutic drugs, antidepressant, anticonvulsants, beta blocker, antifungals), patients with other autoimmune diseases as rheumatoid arthritis, psoriasis, multiple sclerosis, systemic lupus erythematosus, Addison's disease and Grave's disease or with other endocrinal disorders as congenital adrenal hyperplasia, thyroid disorders, Cushing syndrome, hyperprolactinaemia.

All patients having PCOS were chosen randomly and classified into 5 groups, group (A) included patients with PCOS with no hair loss (normal) hair, group (B) included patients with PCOS and AGA, group (C) included patients with PCOS and AA, group (D) included patients with PCOS and other causes of hair loss as (telogen effluvium, trichotillomania or lichen planopilaris) and group (E) included patients with more than one type of causes of hair loss.

METHODS

Detailed history was taken from all the study participants including age, menstrual history as, history of pregnancy and lactation, drug history, history of surgical operations and family history of PCOS. General examination included measuring of weight, height, body mass index (BMI), signs of hyperandrogenism as acne, hirsutism, seborrhea, high pitched voice, decreased breast mass, masculine body, or body hair hypertrichosis.

Local scalp examination included, hair pull test where about 20-60 hairs were grasped between the thumb, index and middle fingers from the base of the hairs near the scalp and firmly tugged away from the scalp (without force). If more than ten percent of the hairs are pulled away from the scalp, this constitutes a positive pull test and implies active hair shedding, dermoscopy (trichoscopy) to diagnose AGA, TE, alopecia areata (AA), Trichotillomania (TTM) and LPP.

Ludwig score was used for patients diagnosed as AGA, it is used to describe female pattern genetic hair loss in three grades: grade I (mild), grade II (moderate), and grade III (severe)⁽¹⁰⁾. Severity of Alopecia Tool (SALT) score was used for cases with AA, it was focused on the proportion of the alopecic area to total surface area of the scalp, severity was classified as S0 (no hair loss), S1 (1%–24%), S2 (25%–49%), S3 (50%–74%), S4 (75%–99%), and S5 (100%)⁽¹¹⁾.

Investigations:

Blood samples were taken from all study patients for assessment of serum free testosterone, prolactin, luteinizing hormone (LH), follicle-stimulating hormone (FSH), while dehydroepiandrosterone-sulphate (DHEA-S) was done only for the participants with AGA. Abdominal and pelvic ultrasound were performed to for examination of the participants.

Ethical Consideration:

The Mansoura Faculty of Medicine's Institutional Review Board (IRB) accepted this report (MS. 21. 07.1562). An informed verbal consent was taken before inclusion of patients into the study. Every care was taken to protect the data's privacy. All data were used for scientific purposes. The study adhered to the Helsinki Declaration throughout its execution.

Statistical analysis:

The collected data were revised, coded, and tabulated using SPSS (IBM SPSS Statistics for Windows, Version 25.0. Armonk, NY: IBM Corp.). Data were presented and suitable analysis was done according to the type of data obtained for each parameter. Mean±SD, median, and range were used for numerical data. Frequency and percentage were used for non-numerical data. Student T Test was used to assess the statistical significance of the difference of parametric variable between two study group means. Chi-Square test was used to assess the relationship between two qualitative variables. Fisher Exact or Monte-Carlo test was used to assess the relation between two qualitative variables when the expected count was less than 5. A p value was considered significant if <0.05 at CI 95%.

RESULTS

Table (1) shows that ages were matched with all the groups, and there was no statistically significant relationship between the causes of hair loss and marital state, occupation, pregnancy, lactation, psychological disturbances, weight, height, or BMI. On the other hand, there was a statistically significant relationship between the causes of hair loss and menstrual irregularities, early or late puberty onset and present family history. Also, there were no statistically significant differences between the causes of hair loss and medical ttt or surgical ttt history for PCOS.

Table (1): Comparison between causes of hair loss regarding the history of patients with PCOS and hair loss.

	Group A N = 26		Group B N = 98		Group C N = 37		Group D N = 81		Group E N = 4		Test	P
Age (Years) Mean ±SD.	32.38 ± 6.56		30.57 ± 5.73		30.35 ± 5.14		29.25 ± 5.72		31.75 ± 8.14		F=1.64	0.165
Weight (kg) Mean ±SD.	69.77 ± 14.28		74.38 ± 16.62		76.08 ± 20.59		70.80 ± 16.07		79.50 ± 6.56		H=4.165	0.384
Height Mean ±SD.	162.0 ± 5.19		161.3 ± 5.49		161.2 ± 6.22		161.6 ± 5.70		157.5 ± 6.14		F=0.571	0.684
Marital State	No.	%	No.	%	No.	%	No.	%	No.	%		
Not Married	5	19.2	21	21.4	12	32.4	23	28.4	1	25.0	X ² = 2.696	0.610
Married	21	80.8	77	78.6	25	67.6	58	71.6	3	75.0		
Occupation												
Professional Jobs	3	11.5	13	13.3	6	16.2	8	9.9	1	25.0	X ² = 13.958	MC 0.551
Employee, Worker, Teacher	7	26.9	25	25.5	8	21.6	17	21.0	1	25.0		
Seller	6	23.1	13	13.3	5	13.5	22	27.2	1	25.0		
Student	1	3.8	7	7.1	2	5.4	8	9.9	1	25.0		
Housewife	9	34.6	40	40.8	16	43.2	26	32.1	0	0.0		
Pregnancy Lactation												
None	16	61.5	77	78.6	26	70.3	64	79.0	3	75.0	X ² = 8.677	MC 0.314
Pregnant	7	26.9	8	8.2	5	13.5	8	9.9	1	25.0		
Lactating	3	11.5	13	13.3	6	16.2	9	11.1	0	0.0		
Menstrual Irregularities												
Absent	16	61.5	32	32.7	25	67.6	48	59.3	1	25.0	X ² = 26.757	MC 0.005*
Oligomenorrhea	4	15.4	16	16.3	1	2.7	7	8.6	0	0.0		
Polymenorrhea	5	19.2	33	33.7	7	18.9	16	19.8	3	75.0		
Amenorrhea	1	3.8	17	17.3	4	10.8	10	12.3	0	0.0		
Psychological Disturbances												
No	24	92.3	82	83.7	33	89.2	65	80.2	3	75.0	X ² = 3.335	MC 0.477
Yes	2	7.7	16	16.3	4	10.8	16	19.8	1	25.0		
Puberty Onset												
Normal	20	76.9	38	38.8	25	67.6	59	72.8	1	25.0	X ² = 29.214	<0.001*
Late	6	23.1	60	61.2	12	32.4	22	27.2	3	75.0		
Family History of PCOS												
No	22	84.6	29	29.6	34	91.9	75	92.6	2	50.0	X ² = 98.733	<0.001*
Yes	4	15.4	69	70.4	3	8.1	6	7.4	2	50.0		
Medical History of TTT of PCOS												
No	18	69.2	69	70.4	32	86.5	51	63.0	2	50.0	X ² = 7.464	0.113
Yes	8	30.8	29	29.6	5	13.5	30	37.0	2	50.0		
Surgical History of TTT of PCOS												
No	25	96.2	93	94.9	37	100.0	77	95.1	4	100.0	X ² = 2.190	MC 0.684
Yes	1	3.8	5	5.1	0	0.0	4	4.9	0	0.0		

SD.: Standard deviation, F: One Way ANOVA test, H: Kruskal Wallis test, X²: Chi Square, MC: Monte Carlo, *: Significant.

Table (2) shows that there were statistically significant relationships between the causes of hair loss with seborrhea and hirsutism. A significantly higher percentage of patients with AGA had seborrhea and hirsutism compared to other groups. However, there was no significant differences between the causes of hair loss and presence of acne, high pitched voice, decreased breast mass, masculine body, or body hair hypertrichosis. There were no statistically significant differences as regard of hormonal profile (FT, LH, FSH, Prolactin, TSH, or DHEA-S).

Table (2): Comparison between causes of hair loss regarding general examination and hormonal profile among patients of PCOS

	Group A n = 26		Group B n = 98		Group C n = 37		Group D n = 81		Group E n = 4		Test	P
	No.	%	No.	%	No.	%	No.	%	No.	%		
Acne												
No	12	46.2	37	37.8	21	56.8	45	55.6	2	50.0	X ² = 7.139	0.129
Yes	14	53.8	61	62.2	16	43.2	36	44.4	2	50.0		
Mild	7	26.9	27	27.6	8	21.6	13	16.0	1	25.0		
Moderate	7	26.9	27	27.6	7	18.9	17	21.0	1	25.0		
Severe	0	0.0	7	7.1	1	2.7	6	7.4	0	0.0		
Seborrhea												
No	21	80.8	53	54.1	25	67.6	61	75.3	2	50.0	X ² = 12.328	0.015*
Yes	5	19.2	45	45.9	12	32.4	20	24.7	2	50.0		
Hirsutism												
No	22	84.6	51	52.0	28	75.7	71	87.7	2	50.0	X ² = 31.431	<0.001*
Yes	4	15.4	47	48.0	9	24.3	10	12.3	2	50.0		
Face	1	3.8	34	34.7	4	10.8	4	4.9	1	25.0		
Body	3	11.5	13	13.3	5	13.5	6	7.4	1	25.0		
High pitched voice												
No	25	96.2	93	94.9	36	97.3	75	92.6	3	75.0	X ² = 3.858	MC 0.403
Yes	1	3.8	5	5.1	1	2.7	6	7.4	1	25.0		
Decreased breast mass												
No	26	100.0	93	94.9	34	91.9	79	97.5	4	100.0	X ² = 3.273	MC 0.455
Yes	0	0.0	5	5.1	3	8.1	2	2.5	0	0.0		
Masculine body												
No	25	96.2	95	96.9	33	89.2	76	93.8	4	100.0	X ² = 3.592	MC 0.428
Yes	1	3.8	3	3.1	4	10.8	5	6.2	0	0.0		
Body hair hypertrichosis												
No	20	76.9	84	85.7	31	83.8	68	84.0	3	75.0	X ² = 2.019	MC 0.712
Yes	6	23.1	14	14.3	6	16.2	13	16.0	1	25.0		
Hormonal profile												
FT Mean ±SD.	1.27 ± 0.74		1.35 ± 0.21		1.43 ± 0.60		1.33 ± 0.20		1.32 ± 0.17		H=0.861	0.930
LH Mean ±SD.	8.66 ± 3.77		10.63 ± 4.23		10.44 ± 3.98		10.01 ± 3.98		7.13 ± 2.81		H=3.838	0.428
FSH Mean ±SD.	5.13 ± 1.90		5.20 ± 1.69		5.38 ± 1.18		5.30 ± 1.57		5.63 ± 2.03		F=0.405	0.805
Prolactin Mean ±SD.	11.28 ± 4.94		11.76 ± 4.74		12.33 ± 4.82		12.93 ± 4.90		14.10 ± 3.27		H=4.879	0.300
TSH Mean ±SD.	2.22 ± 0.59		2.23 ± 0.86		2.34 ± 0.90		2.28 ± 0.79		2.25 ± 0.87		F=0.319	0.865
DHEA-S- AGA Mean ±SD.	-		270.2 ± 94.11		-		-		344.0 ± 70.71		H=2.082	0.353

X²: Chi Square, MC: Monte Carlo, SD.: Standard deviation, F: One Way ANOVA test, H: Kruskal Wallis test, *: Significant.

Table (3) demonstrates that there was no significant relationship between those with and without AA regarding acne, seborrhea, hirsutism, high-pitched voice, decreased breast mass, masculine body, or body hair hypertrichosis. There was a statistically significant relationship between those with and without AGA regarding acne, seborrhea and hirsutism, but there was no statistically significant relationship regarding high pitched voice, decreased breast mass, masculine body, and body hair hypertrichosis.

Table (3): General examination of patients of PCOS and AA and AGA as compared to other patients.

	AA				Test	P	AGA				Test	P
	No (n = 209)		Yes (n = 37)				No (n = 145)		Yes (n = 101)			
	No.	%	No.	%			No.	%	No.	%		
Acne												
No	96	46.2	21	55.3	X ² =1.069	0.301	78	53.8	39	38.6	X ² =5.500*	0.019*
Yes	112	53.8	17	44.7			67	46.2	62	61.4		
Seborrhea												
No	136	65.4	26	68.4	X ² =0.132	0.717	108	74.5	54	53.5	X ² = 11.695*	0.001*
Yes	72	34.6	12	31.6			37	25.5	47	46.5		
Hirsutism												
No	145	69.7	29	76.3	X ² =0.677	0.411	122	84.1	52	51.5	X ² = 30.661*	<0.001*
Yes	63	30.3	9	23.7			23	15.9	49	48.5		
High pitched voice												
No	195	93.8	37	97.4	X ² =0.784	FE 0.702	137	94.5	95	94.1	X ² =0.020	0.888
Yes	13	6.3	1	2.6			8	5.5	6	5.9		
Decreased breast mass												
No	201	96.6	35	92.1	X ² =1.690	FE 0.188	140	96.6	96	95.0	X ² =0.344	FE 0.745
Yes	7	3.4	3	7.9			5	3.4	5	5.0		
Masculine body												
No	199	95.7	34	89.5	X ² =2.467	FE 0.123	135	93.1	98	97.0	X ² =1.834	0.176
Yes	9	4.3	4	10.5			10	6.9	3	3.0		
Body hair hypertrichosis												
No	174	83.7	32	84.2	X ² =0.007	0.932	120	82.8	86	85.1	X ² =0.250	0.617
Yes	34	16.3	6	15.8			25	17.2	15	14.9		

X²: Chi Square, FE: Fisher Exact, *: Significant.

The majority (77.2%) of patients with PCOS and AGA had a Ludwig score of type 1. Table (4) shows that there was no statistically significant relationship between Ludwig score with general parameters among AGA cases regarding: average age, marital status, occupation, pregnancy, lactation, or non-pregnant or lactating states, menstrual irregularities, or puberty onset.

Table (4): Association of Ludwig score with general parameters among patients with PCOS and AGA.

		Ludwig 1		Ludwig 2		Ludwig 3		test	P
		N=78	%=77.2	N=19	%=19.2	N=4	%=4.0		
Age (years)	Mean±SD	31.13±5.84		28.42±5.32		28.25±4.72		H=3.116	0.211
	Median (min-max)	30(21-45)		29(17-39)		26.5(25-35)			
BMI (kg/m ²)	Mean±SD	29.31±6.15		27.37±5.9		34.45±4.71		H=5.001	0.082
	Median (min-max)	28.7(21.4-45.7)		24.4(21.7-40.3)		33.95(29.6-40.3)			
Acne	No	29	38.2%	8	42.1%	1	25.0%	X ² =0.416	0.853
	Yes	47	61.8%	11	57.9%	3	75.0%		
Seborrhea	No	37	48.7%	14	73.7%	2	50.0%	X ² =3.840	0.147
	Yes	39	51.3%	5	26.3%	2	50.0%		
Hirsutism	No	37	48.7%	11	57.9%	3	75.0%	X ² =1.437	0.488
	Yes	39	51.3%	8	42.1%	1	25.0%		
High pitched voice	No	72	94.7%	18	94.7%	3	75.0%	X ² =2.626	0.269
	Yes	4	5.3%	1	5.3%	1	25.0%		
Menstrual irregularities	Absent	22	28.9%	7	36.8%	2	50.0%	X ² =4.251	0.643
	Oligo	12	15.8%	3	15.8%	1	25.0%		
	Poly	30	39.5%	4	21.1%	1	25.0%		
	Ameno	12	15.8%	5	26.3%	0	0.0%		
Decreased breast mass	No	73	96.1%	17	89.5%	4	100.0%	X ² =1.594	0.451
	Yes	3	3.9%	2	10.5%	0	0.0%		
Masculine body	No	73	96.1%	19	100.0%	4	100.0%	X ² =0.936	0.626
	Yes	3	3.9%	0	0.0%	0	0.0%		
Body hair hypertrichosis	No	64	84.2%	17	89.5%	3	75.0%	X ² =0.642	0.725
	Yes	12	15.8%	2	10.5%	1	25.0%		

SD: standard deviation, H: Kruskal Wallis test, X²: chi square test.

Table (5) shows that women with TE were significantly more likely to have hirsutism compared to those without TE. However, there were no statistically significant relationships between those with and without TE regarding acne, seborrhea, high-pitched voice, decreased breast mass, masculine body, or body hair hypertrichosis. There were no statistically significant relationships between those with and without TTM regarding acne, seborrhea, hirsutism, high-pitched voice, decreased breast mass, masculine body, or body hair hypertrichosis. There were no statistically significant relationships between those with and without LLP regarding general examination and hormonal profile.

Table (5): General examination in patients with PCOS and TE and TTM and LLP as compared to other groups

	TE				Test	p	TTM				Test	P	LLP				Test	P
	No		Yes				No		Yes				No		Yes			
	n = 180	n = 66	n = 239	n = 7			n = 238	n = 8										
N	%	N	%	N	%	N	%	N	%	N	%	N	%					
o.		o.		o.		o.		o.		o.		o.						
Acne																		
No	78	44.1	39	56.5	X ² =	0.079	114	47.9	3	37.5	X ² =	FE	112	47.1	5	62.5	X ² =	FE
Yes	99	55.9	30	43.5	3.087		124	52.1	5	62.5	0.336	0.725	126	52.9	3	37.5	0.740	0.483
Seborrhea																		
No	113	63.8	49	71.0	X ² =	0.287	154	64.7	8	100	X ² =	FE	156	65.5	6	75.0	X ² =	FE
Yes	64	36.2	20	29.0	1.136		84	35.3	0	0.0	4.288	0.054	82	34.5	2	25.0	0.308	0.719
Hirsutism																		
No	63	35.6	9	13.0	X ² =	<	169	71.0	5	62.5	X ² =	FE	166	69.7	8	100.0	X ² =	FE
Yes	114	64.4	60	87.0	12.194*	0.001*	69	29.0	3	37.5	0.271	0.695	72	30.3	0	0.0	3.422	0.109
High pitched voice																		
No	170	96.0	62	89.9	X ² =	FE	224	94.1	8	100	X ² =	FE	224	94.1	8	100.0	X ² =	FE
Yes	7	4.0	7	10.1	3.544	0.071	14	5.9	0	0.0	0.499	1.000	14	5.9	0	0.0	0.499	1.000
Decreased breast mass																		
No	169	95.5	67	97.1	X ² =	FE	228	95.8	8	100	X ² =	FE	228	95.8	8	100.0	X ² =	FE
Yes	8	4.5	2	2.9	0.335	0.730	10	4.2	0	0.0	0.350	1.000	10	4.2	0	0.0	0.350	1.000
Masculine body																		
No	168	94.9	65	94.2	X ² =	FE	225	94.5	8	100	X ² =	FE	226	95.0	7	87.5	X ² =	FE
Yes	9	5.1	4	5.8	0.050	0.761	13	5.5	0	0.0	0.461	1.000	12	5.0	1	12.5	0.860	0.356
Body hair hypertrichosis																		
No	148	83.6	58	84.1	X ² =	FE 0.9	200	84.0	6	75.0	X ² =	FE	199	83.6	7	87.5	X ² =	FE
Yes	29	16.4	11	15.9	0.007		38	16.0	2	25.0	0.464	0.620	39	16.4	1	12.5	0.086	1.000

X²: Chi Square, FE: Fisher Exact, *: Significant.

Table (6) shows the relationship between AA and various aspects of patient's history. The results showed that there were no statistically significant differences between those with and without AA regarding average age, marital status, occupation, pregnancy, lactation, menstrual irregularities, psychological disturbances, or puberty onset. However, there was a significant relationship between AA and family history. 38.9% of patients without AA reported a family history compared to 7.9% of those with AA. There were no statistically significant relationships between those with and without TE regarding average age, marital status, occupation, pregnancy, lactation, menstrual irregularities, or the presence or absence of psychological disturbances. However, there were significant relationships between TE and puberty onset and family history.

Table (6): History of patients with PCOS and AA and TE as compared to other groups.

	AA				Test	P	TE				Test	P
	No n = 209		Yes n = 37				No n = 180		Yes n = 66			
Age (years) Mean ± SD.	30.26 ± 5.90		30.58 ± 5.26		t=	0.759	30.67 ± 5.83		29.41 ± 5.67		t=1.536	0.126
	No.	%	No.	%			No.	%	No.	%		
Marital state												
Not Married	49	23.6	13	34.2	X ² = 1.934	0.164	44	24.9	18	26.1	X ² = 0.040	0.842
Married	159	76.4	25	65.8			133	75.1	51	73.9		
Occupation												
Professional jobs	24	11.5	7	18.4	X ² = 2.831	0.586	23	13.0	8	11.6	X ² = 7.806	0.099
Employee, worker, teacher	50	24.0	8	21.1			44	24.9	14	20.3		
Seller	42	20.2	5	13.2			27	15.3	20	29.0		
Student	17	8.2	2	5.3			12	6.8	7	10.1		
Housewife	75	36.1	16	42.1			71	40.1	20	29.0		
Pregnancy lactation												
None	159	76.4	27	71.1	X ² = 0.816	MC 0.716	134	75.7	52	75.4	X ² = 0.206	0.902
Pregnant	24	11.5	5	13.2			20	11.3	9	13.0		
Lactating	25	12.0	6	15.8			23	13.0	8	11.6		
Menstrual irregularities												
Absent	96	46.2	26	68.4	X ² = 7.095	MC 0.065	80	45.2	42	60.9	X ² = 4.880	0.181
Oligo	27	13.0	1	2.6			22	12.4	6	8.7		
Poly	57	27.4	7	18.4			50	28.2	14	20.3		
Ameno	28	13.5	4	10.5			25	14.1	7	10.1		
Psychological disturbances												
No	174	83.7	33	86.8	X ² = 0.245	0.621	145	81.9	62	89.9	X ² = 2.343	0.126
Yes	34	16.3	5	13.2			32	18.1	7	10.1		
Puberty onset												
normal	118	56.7	25	65.8	X ² = 1.083	0.298	95	53.7	48	69.6	X ² = 5.152*	0.023*
Late	90	43.3	13	34.2			82	46.3	21	30.4		
Family history												
No	127	61.1	35	92.1	X ² = 13.77*	<0.001*	97	54.8	65	94.2	X ² = 34.274*	<0.001*
Yes	81	38.9	3	7.9			80	45.2	4	5.8		

SD.: Standard deviation, t: Student t test. X²: Chi Square, MC: Monte Carlo, *: Significant.

Table (7) shows that there were no statistically significant relationships between those with and without TTM regarding average age, marital status, occupation, pregnancy, lactation, or non-pregnant or lactating states, menstrual irregularities, puberty onset, or family history.

Table (7): History of patients with PCOS and TTM as compared to other groups.

	TTM				Test	P
	No n = 239		Yes n = 7			
Age (years) Mean ± SD.	30.32 ± 5.73		30.25 ± 8.01		t=0.031	0.975
	No.	%	No.	%		
Marital state						
Not Married	58	24.4	4	50.0	X ² = 2.697	FE 0.113
Married	180	75.6	4	50.0		
Occupation						
Professional jobs	30	12.6	1	12.5	X ² = 1.661	MC 0.839
Employee, worker, teacher	57	23.9	1	12.5		
Seller	46	19.3	1	12.5		
Student	18	7.6	1	12.5		
Housewife	87	36.6	4	50.0		
Pregnancy lactation						
None	178	74.8	8	100.0	X ² = 1.269	MC 0.571
Pregnant	29	12.2	0	0.0		
Lactating	31	13.0	0	0.0		
Menstrual irregularities						
Absent	118	49.6	4	50.0	X ² = 1.071	MC 0.866
Oligo	28	11.8	0	0.0		
Poly	61	25.6	3	37.5		
Ameno	31	13.0	1	12.5		
Psychological disturbances						
No	206	86.6	1	12.5	X ² = 31.818*	FE <0.001*
Yes	32	13.4	7	87.5		
Puberty onset						
normal	138	58.0	5	62.5	X ² = 0.065	FE 1.000
Late	100	42.0	3	37.5		
Family history						
No	157	66.0	5	62.5	X ² = 0.041	FE 1.000
Yes	81	34.0	3	37.5		

SD.: Standard deviation, t: Student t test, X²: Chi Square, FE: Fisher Exact, MC: Monte Carlo, *: Significant.

DISCUSSION

Polycystic ovary syndrome is considered the commonest endocrinal disorder of reproductive-aged females globally. It affects about 4.5% of women in the general population. Its pathogenesis isn't totally identified. Impairment of gonadotropic regulation, genetics, and environmental factors have been involved (12).

Cases with PCOS are usually first detected by a dermatologist. It is estimated that 72% to 82% of women with PCOS are seen with dermatological manifestations typically accompanied by hyperandrogenism including androgenetic alopecia, acne vulgaris, hirsutism, seborrhea and acanthosis

nigricans. There are many causes of hair loss that could affect females such as AGA, alopecia areata, anagen effluvium, telogen effluvium, trichotillomania and lichen planopilaris (13).

The current study aimed to evaluate the prevalence of different causes of hair loss among females with PCOS in Delta area - Egypt. The current study included 246 females in childbearing period with confirmed diagnosis of PCOS according to Rotterdam diagnostic criteria (14). Patients were divided into 5 groups based on the type of hair loss as Group A (PCOS and no hair loss), Group B (PCOS and AGA), Group C (PCOS and AA), Group D (PCOS and other causes of hair loss like telogen effluvium, trichotillomania and lichen

planopilaris) and Group E with PCOS and combined causes of hair loss.

In literature, the incidence of hair loss in PCOS isn't clear, about 25% of women with PCOS have troublesome hair loss. In general, it is accompanied by other symptoms like hirsutism, acanthosis nigricans or acne⁽¹⁵⁾. However, in our study, hair loss percentage in PCOS patients was 89.5%. **Prasad et al.**⁽¹⁶⁾ confirmed in their study on 38 women (38%) presenting for hair thinning had a known diagnosis of PCOS at the time of hair loss consultation.

In accordance with our results, higher prevalence of AGA in patients with PCOS (42.5%) was found in **Abusailik et al.**⁽⁹⁾ study, however, **Salman et al.**⁽¹⁷⁾ reported that 25.5% of women with PCOS had AGA. According to **Agarwal et al.**⁽¹⁸⁾, among the patients having PCOS, 28.6% of patients presented with telogen effluvium. This finding was in accordance with our result where telogen effluvium was found in 26.8 % of PCOS patients

Our study revealed insignificant relationships between the studied groups and age, marital status, occupation, pregnancy, lactation, psychological disturbances, weight, height, or BMI ($p>0.05$). However, there were statistically significant relationship between groups regarding menstrual irregularities ($p=0.005$). Additionally, in our study, a higher percentage of patients with AGA (61.2 %) had a late puberty onset. Over 70% of patients with AGA reported +ve family history of PCOS.

In the present study, patients with menstrual irregularities and AGA in group (B) had polymenorrhea (33.7%) and oligomenorrhea (16.3%) while patients with no hair loss (group A) had the highest percentage (61.5%) of regular menses. However, **Quinn et al.**⁽¹⁹⁾ found menstrual irregularities in 24.7% of PCOS patients. Also, **Keen et al.**⁽²⁰⁾ reported that oligomenorrhea was seen in 57 % followed by amenorrhea seen in 8% of PCOS patients.

In addition, in our study, there was a significant relationship between AA and family history of AA ($p<0.001$), 38.9% of patients with AA reported a family history compared to 7.9% of those without AA. The rate of a positive family history of AA varied widely from around 10% to 50%⁽²¹⁾. Positive family history of AA in PCOS patients was significantly more common in cases with AA than those without AA⁽²²⁾.

There were insignificant differences between groups with hair losses (AA, TE, TTM and LPP) regarding average age, marital status, occupation, pregnancy, lactation, menstrual irregularities, psychological disturbances, or puberty onset ($p>0.05$) except in patients with trichotillomania. The current study showed that over 85% of individuals with TTM were more likely to report psychological disturbances compared to those without TTM (13.4%). This result was concomitant with that of **Shah et al.**⁽²³⁾ where the prevalence of psychiatric problems in females with PCOS has been recorded to range between 57% and 67%. **Mehrabadi et al.**⁽²⁴⁾ also, reported that the

prevalence of depression in women with PCOS was 28 to 64% and the prevalence of anxiety was 34 to 57%.

The current study revealed no statistically significant associations between groups with hair loss (AA, TE, TTM, LPP) regarding acne, seborrhea, hirsutism, high-pitched voice, decreased breast mass, masculine body and body hair hypertrichosis ($p>0.05$) except for hirsutism. Cases with TE were significantly more likely to have hirsutism compared to TE free ones ($p<0.001$).

This is nearly in accordance with **Awrahman and Qurtas**⁽²⁵⁾ study in which three types of non-scarring hair loss were accompanied by hirsutism in PCOS patients (AGA with TE). The commonest type of hair loss accompanied by hirsutism was AGA with TE.

Regarding to laboratory abnormalities, our study found no significant differences ($p>0.05$) between our groups concerning hormonal profile (FT, LH, FSH, Prolactin, TSH) or DHEA-S in AGA only. However, in literature, assessment of biochemical profile was done only in PCOS patients with AGA and LPP.

Our results were in accordance with that of **Quinn et al.**⁽¹⁹⁾ who recorded that there were no statistically significant differences in overall biochemical hyperandrogenemia between PCOS patients with and without AGA. Meanwhile, in **Abusailik et al.**⁽⁹⁾ study, 38.4% of patients with PCOS and AGA had normal hormonal profiles, whereas the rest (61.6%) manifested by at least one hormonal abnormality, which include an abnormal LH:FSH ratio (35.6% of cases had one abnormal hormonal level and 26.1% had at least two abnormal hormonal levels).

Gowri et al.⁽²⁶⁾ reported that raised testosterone levels was the most common finding in PCOS (in 55% patients). Moreover, **Keen et al.**⁽²⁰⁾, reported that the commonest findings were raised LH/FSH in 38% of patients followed by raised testosterone levels in 28% patients.

Additionally, **Aljefri et al.**⁽²⁷⁾ showed that the most frequent hormonal changes encountered in PCOS cases were raised LH levels (in 49.1% of cases) and raised LH/FSH ratio (in 35.5% of cases). A major number of cases had normal levels of TSH, prolactin 72.1%, FSH, testosterone, glycosylated haemoglobin, and fasting blood glucose.

CONCLUSION

Acne, hirsutism, AGA and seborrhea have strong association with PCOS in current study. AGA is accompanied by other manifestations of clinical hyperandrogenism but doesn't seem to be accompanied by greater risk of biochemical changes than PCOS alone. AGA is the commonest prevalent clinical manifestation of hair loss in our study, followed by AA, telogen effluvium, trichotillomania and lichen planopilaris.

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