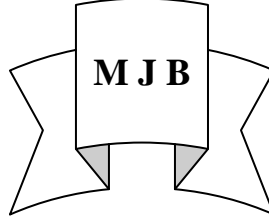


Hirsutism: A Clinical and Biochemical Study among Iraqi Women in Hilla City

Zena Saeed Al-Fadhily

Dept. of Medicine, College of Medicine, University of Babylon, Hilla, Iraq.



Abstract

Background: Hirsutism is defined as excessive terminal hairs that appear in women in a male-like pattern (i.e., sexual hair). It is relatively a common and important medical problem affecting about 5-10% of women in reproductive age. The growth of sexual hair is entirely under the influence of androgens, many hormones have androgenic potential in the human body, but testosterone is the key circulating androgen which is produced by the ovaries and adrenals. Hirsutism can be familial, idiopathic, or caused by excess androgen secretion by the ovary, adrenal glands or exogenous pharmacologic sources of androgen and other miscellaneous endocrine abnormalities.

Aim of the study: To shed light on hirsutism as a rising problem among women in our society, evaluating those hirsute women both clinically and biochemically.

Results: Fifty women with hirsutism were included in this study, their ages ranged from 14-45 years with a mean of 27.64 ± 7.266 . Mean duration of disease was 7.52 ± 6.217 years; while mean age of onset 20.36 ± 4.758 years. All patients had slow progression of hirsutism over years. Positive family history was found in 80% of patients, 38% were married, 6 (12%) are infertile. Irregular menstrual cycles were found in 44%, mostly in a form of oligomenorrhea. The number of hirsute patients who were over weight or obese ($BMI \geq 25$) was 30 patients (60%), [16 patients had android obesity ($WHR \geq 0.85$) and 14 patients had overall (non android) obesity], while the number of non obese hirsute patients ($BMI < 25$) was 20 patients (40%). 31 patients (62%) had elevated serum total testosterone, there was highly significant statistical difference regarding serum testosterone when compared with control group (p-value 0.0001). 20% of patients had elevated serum prolactin, while 4 patients (8%) of control group had elevated serum prolactin. There was no significant correlation between serum total testosterone and serum prolactin levels with BMI or WHR (P-value > 0.05). 36 patients (72%) had mild hirsutism (score 8-15) and 14 patients (28%) had moderate to severe hirsutism (score > 15). Significant correlation was found between the score of the patients with BMI and WHR (P-value < 0.05). Also there was highly significant statistical relationship between the score of the patients and serum androgen level; patients with scores > 15 had significantly higher levels of serum androgen than those with lower scores. Seborrhea, androgenetic alopecia, acne and acanthosis nigricans were recorded in patients with decreasing frequencies, while hypertrichosis if found in 72% of patients especially in those with high score hirsutism.

Conclusion: Hirsutism is not merely aesthetic problem, it can be linked to several metabolic and endocrine abnormalities especially if a patient presents with moderate-severe hirsutism even when it had a slow progression or family history can be obtained.

الخلاصة

المقدمة: يعرف داء الشعرانية بأنه حالة ظهور شعر كثيف خشن بكميات زائدة عند النساء في مناطق من الجسم يشبه ذلك الموجود عند الرجال. تعتبر هذه الحالة شائعة نسبياً ومهمة لأنها تصيب 5-10% من النساء في سن الخصوبة. يسيطر هرمون الأندروجين كلياً على نمو الشعر في المناطق التناسلية، هناك عدة أنواع من الأندروجينات في الجسم لكن يعتبر هرمون الشحمون الخصوي أهم هذه الهرمونات ويتم إفرازه من الغدد الكظرية والمبيض. ظاهرة الشعرانية من الممكن أن تكون وراثية، أو بسبب زيادة إفراز الأندروجين من المبيض والكظرية أو بسبب بعض الأدوية التي تماثل عمل الأندروجين أو غير معروفه الاسباب.

هدف الدراسة: تسليط الضوء على ظاهرة الشعرانية باعتبارها مشكلة متزايدة في مجتمعنا وتقييم هذه الحالة عند المريضات سريريا وايضا عن طريق التحليلات المختبرية.

النتائج: ادخلت عينة من النساء المصابات بداء الشعرانية عددها ٥٠ امرأة، وكانت اعمارهن تتراوح بين ١٤-٤٥ سنة بمعدل ٢٧،٦٤ سنة. معدل مدة المرض لديهن كانت ٧،٥٢ سنة، بينما كان معدل اعمارهن لدى بداية المرض ٢٠،٣٦ سنة. جميع المريضا تلاحظن ظهور الشعر المتزايد بصورة بطيئة على مدى سنوات. ٨٠% من الحالات كان لديهن قريبات يعانين من نفس المشكلة، ٣٨% منهن كن متزوجات و ٦ (١٢%) كن عقيمات. عدم انتظام الدورة الشهرية كان موجودا عند ٤٤% غالبا على شكل انقطاع الطمث. عدد المريضا اللواتي كن يعانين من زيادة في الوزن او سمنة (معامل كتلة الجسم ≤ 25) كان ٣٠ (٦٠%)، ١٦ مريضة كان لديهن نسبة قياس الخصر الى الورك ≤ 0.85 ، اما باقي المريضا ١٤ كانت هذه النسبة اقل من ٠،٨٥، بينما عدد المريضا ذوات الوزن الطبيعي كان ٢٠ (٤٠%). ارتفاع الهرمون الذكري لوحظ عند ٣١ مريضة (٦٢%) وكانت هذه النسبة مهمة احصائيا عند المقارنة مع مجموعة النساء الطبيعيات (مجموعة السيطرة)، كما ان ارتفاع هرمون الحليب البيرولاكتين كان مرتفعا لدى ٢٠% من المريضا بينما كان مرتفعا عند ٨% فقط من مجموعة السيطرة. لم تلاحظ هناك علاقة احصائية مهمة بين ارتفاع الهرمون الذكري وهرمون الحليب مع معامل كتلة الجسم ونسبة قياس الخصر الى الورك. ٣٦ مريضة (٧٢%) كان لديهن ظاهرة شعرانية بسيطة (مستوى الشعرانية ٨-١٥) اما المتبقي ١٤ مريضة كان لديهن شعرانية متوسطة او شديدة (مستوى الشعرانية اكثر من ١٥). وجدت علاقة احصائية مهمة بين مستوى الشعرانية مع معامل كتلة الجسم ونسبة قياس الخصر الى الورك، كما وجدت علاقة مهمة ايضا بين مستوى الشعرانية ونسبة ارتفاع الهرمون الذكري في المصل عند المريضا. زيادة الافرازات الدهنية من البشرة، تساقط الشعر، حب الشباب و تتخانات الجلد تواجدت عند المريضا بنسب مختلفة بينما كانت ظاهرة زيادة الشعر في الاماكن غير الخاضعة لسيطرة الاندروجين موجودة عند ٧٢% من المرضى.

الاستنتاجات: داء الشعرانية هو ليس مجرد مشكلة جمالية لدى النساء وانما يمكن ان يشير الى تغيرات غير طبيعية ابيضية وهرمونية عديدة خاصة اذا كانت المريضة تعاني من شعرانية متوسطة او شديدة حتى لو كانت هذه الحالة بطيئة الظهور او كان لدى المريضة اقارب يعانون المشكلة نفسها.

Introduction

Definition:

Hirsutism is defined as excessive terminal hairs that appear in women in a male-like pattern (i.e., sexual hair). **Hypertrichosis** on the other hand, is excessive terminal hair growth in a non sexual pattern. [1]

Prevalence: It is relatively a common and important medical problem affecting about 5-10% of women in reproductive age. [1, 2] The prevalence of hirsutism is dependent on the ethnic and racial origin of the population under study, but it also depends to a certain degree on the method used to diagnose hirsutism. An incidence of hirsutism of 8% was found in the US [3], while in Iraq a study was done in 1992 showed that the incidence among Iraqi women was 59% [4]. Fair skinned Europeans have the least amount of terminal hair, whereas southern European dark skinned Mediterranean women have

the greatest amount of terminal hair [2].

Pathogenesis: The growth of sexual hair is entirely under the influence of androgens. Vellus hair is present before puberty. By the effect of increased level of androgens at puberty, vellus follicles develop into terminal hair at androgen sensitive areas. Hirsutism can results from an increased androgen level or over sensitivity of the hair follicles to androgen. However, the severity of hirsutism does not correlate well with the level of androgen because the response of hair follicles to androgen excess varies considerably within and among persons [5].

Many hormones have androgenic potential in the human body, but testosterone is the key circulating androgen which is produced by the ovaries and adrenals either as testosterone or as prohormones (mainly androstenedione and dehydroepiandrosteron sulfate) which are metabolized into testosterone in the

peripheral tissues such as fat [6]. Testosterone is converted into dihydrotestosterone (DHT) in the peripheral tissue by the enzyme 5- α reductase. DHT is the most potent androgen in the body. The phenomena of increased conversion rate of DHT in the target area may help to clarify the increased sensitivity of hair follicles to androgens [2].

Virilization is the combination of hirsutism plus other signs of masculinization; like: acne, increase sebum production, deepening of the voice, fronto-temporal balding, infrequent or absent menses and clitoral hypertrophy. It is associated with marked increased androgen production by the ovaries or adrenals or both [1, 2, 7]. Other abnormalities associated with androgen augmentation include: dyslipidemia, diabetes, hypertension, insulin resistance, android obesity, coronary heart disease and endometrial carcinoma [8, 9, 10].

Causes: Increased androgen effect that results in hirsutism can be familial, idiopathic, or caused by excess androgen secretion by the ovary (e.g., polycystic ovary syndrome PCOS, tumors), excess secretion of androgens by adrenal glands (e.g., congenital adrenal hyperplasia CAH, Cushing's syndrome, tumor), exogenous pharmacologic sources of androgens (anabolic steroids, danazol, minoxidil, etc.) and other miscellaneous endocrinological abnormalities like hyperprolactinemia, acromegaly and thyroid dysfunction [1, 7, 10, 11].

Over 80% of patients with hirsutism have PCOS which affects approximately 6% of women of reproductive age. PCOS represented by chronic anovulation and hyperandrogenemia. Patients often report menstrual irregularities, infertility, obesity and symptoms associated with androgen excess, and diagnosis usually is based on clinical

rather than laboratory findings. Up to 70% of patients with PCOS have signs of hyperandrogenism [1, 10].

Idiopathic hirsutism is the second most common represents 10% of cases of hirsutism and often familial. It is a diagnosis of exclusion and thought to be related to disorders in peripheral androgen activity. Onset occurs shortly after puberty with slow progression. Patients with idiopathic hirsutism generally have normal menses and normal levels of circulating androgens. Other diseases represent the last 10% of causes [1, 12, 13, 14].

Evaluation: Clinical evaluation of a woman with hirsutism include thorough history and physical examination seeking for a possible cause and also using standardized scoring system for the amount and degree of hirsutism. The most common method of scoring body and facial terminal hair growth used today is the **modified Ferriman-Gallway score**. This grades the hair growth between 0 (absence of terminal hair) to 4 (extensive terminal hair growth) at nine different body sites (upper lip, chin, chest, upper & lower abdomen, upper & lower back, arm and inner thigh). Hirsutism is said to be present if the total score is 8 or more [1, 2, 15].

Aim of the Study

To shed alight on hirsutism as a rising problem among women in our society, evaluating those hirsute women both clinically and biochemically.

Patients and Methods

This study was conducted in Hilla city during a period of one year from September 2010 to September 2011. A group of **50** patients in whom the chief complain was hirsutism and were attending to private clinic for IPL hair removal. Another group of **20** healthy non hirsute age-matched women was

also included in the study as a control group.
All 50 patients were evaluated initially regarding the presence and severity of

hirsutism by using modified Ferriman-Gallway score (a visual scoring system) by a single examiner to avoid bias. (Figure 1).

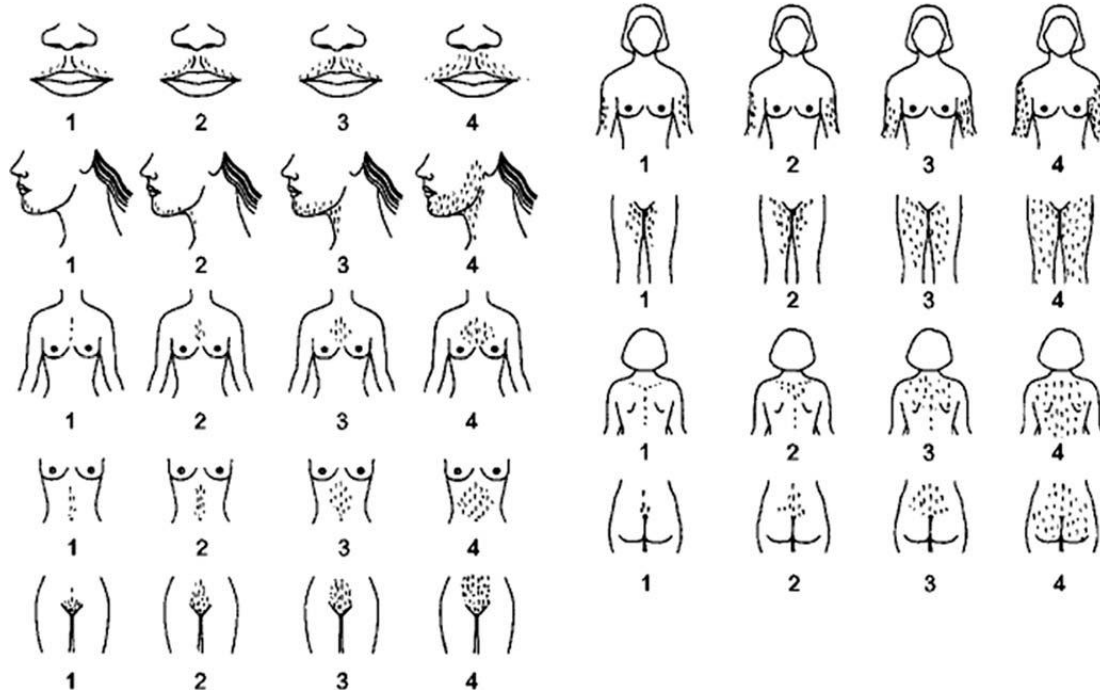


Figure 1 Modified FG scoring system: Clinically, terminal hair hairs can be distinguished from vellus hairs primarily by their length (i.e. greater than 0.5 cm) and the fact that they are usually pigmented.

History regarding the following items was taken from each patient: duration and onset of the disease, rate of progression, family history, menstrual history, marital status, frequency and method of epilation used, drug history (specially androgenic and hormonal therapies).

Physical examination was done for each patient and this include the following anthropometric measures: height (measured without shoes against wall-fixed tape), weight (measured with light cloths and without shoes), waist circumference (with a tape measure 2 cm above the umbilicus), hip circumference was also measured then waist/hip ratio (WHR) was calculated. BMI was calculated as: $\text{weight}/(\text{height in meter})^2$. $\text{BMI} \geq 25$ indicate over weight or overall obesity,

while $\text{WHR} \geq 0.85$ indicate android obesity.

Physical examination also looking for dermatological features of hyperandrogenism like: seborrhea, acne, androgenic alopecia and for acanthosis nigricans. Patients examined for the presence of hypertrichosis.

Any patient take antiandrogenic or hormonal therapy 3 months before participation was excluded from this study.

Fasting blood samples were taken from all patients and control group for hormonal analysis (total testosterone and prolactin hormones). Serum total testosterone level assessed by using ELISA technique, while serum prolactin was assessed by Minividas device.

Statistical analysis was done by using SPSS package 1998.

Results

Fifty women with hirsutism were included in this study, their ages ranged from 14-45 years with a mean \pm

SD of 27.64 ± 7.266 . Duration of the disease was ranged from 1-28 years (mean duration 7.52 ± 6.217); while age of onset of the disease was ranged 12-32 years (mean age of onset 20.36 ± 4.758). (Table 1)

Table 1 Age, duration and age of onset of hirsute women.

	Range (years)	Mean \pm SD
Age of patients	14-45	27.64 ± 7.266
age of onset	12-32	20.36 ± 4.758
Duration of the disease	1-28	7.52 ± 6.217

Rate of progression of the disease was slow for all patients 100% (over years). Positive family history was found in 40 patients (80%) (whether 1st or 2nd degree relatives) with 58% had positive family history in more than two members of the family. 19 patients (38%) were married, of them 6 (12%) are infertile. Irregular menstrual cycles were found in 22 patients (44%),

mostly in a form of periods of oligomenorrhea.

Body weight of patients group ranged 40-125 kg with a mean of 68.4 ± 7.266 , BMI was ranged 18.26-50.07 with a mean of 27.84 ± 6.579 , there was no statistical significant difference with the mean body weight & BMI of the control group (P-value > 0.05).

(Table 2)

Table 2 Mean weight and BMI of patients group compared with control group.

	Patients group n=50	Control group n=20	P-value
Mean wt./kg	68.4 ± 7.266	62.9 ± 4.854	0.077 not significant
Mean BMI	27.84 ± 6.579	25.30 ± 2.158	0.074 not significant

The number of hirsute patients who are over weight or obese (BMI ≥ 25) is 30 patients (60%), [16 patients had android obesity (WHR ≥ 0.85) and 14

patients had overall (non android) obesity], while the number of non obese hirsute patients (BMI < 25) was 20 patients (40%). Figure (2)

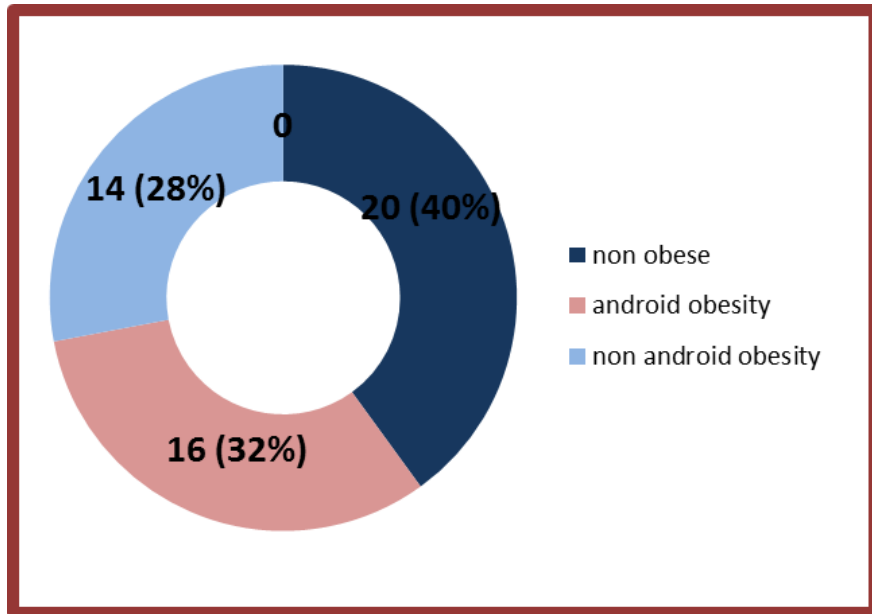


Figure 2 Number and percentage of patients groups according to BMI and WHR.

The mean body wt. and BMI of patients with android obesity are increased over that of patients with non-android obesity, but statistical

significant difference found only between mean body wt. of the two groups (P-value 0.047). (Table 3)

Table 3 Mean body weight and BMI of non-obese, android and non-android obesity patients groups.

	Non-obese (n=20) (BMI<25)	Obese (n=30) (BMI≥25)	
		android obesity (n=16) (WHR≥0.85)	Overall obesity (n=14) (WHR<0.85)
Mean wt./kg	54.8±5.827	81.875±18.278	72.428±9.685
Mean BMI	22.1±1.568	32.4±6.259	29.2±4.377

The level of serum total testosterone of patients group was ranged 0.17± 6.15 ng/ml with a mean of 1.73±1.496, 31 patients (62%) had elevated serum total testosterone (testosterone> 0.9ng/ml), mean level in those patients was 2.5±1.41, while the level of serum total testosterone of control group was

ranged 0.01± 1.67 ng/ml with a mean of 0.4±0.443, 2 patients only (4%) had elevated serum total testosterone, mean level was 1.43±0.33. There was highly significant statistical difference between the two groups (P-value 0.0001). (Table 4)

Table 4 Mean of serum total testosterone level in patients and control groups.

	Range of testosterone (ng/ml)	Mean±SD	Number of patients with ↑ testosterone.	Mean±SD	P-value
Patients group	0.17- 6.1	1.73±1.496	31 (62%)	2.50±1.41	0.0001
Control group	0.01- 1.67	0.4±0.443	2 (4%)	1.43±0.33	

The level of serum prolactin of patients group was ranged 0.5± 54.2 ng/ml with a mean of 17.84±9.915, 10 patients (20%) had elevated serum prolactin (level>23 ng/ml), mean level in those patients was 32.11±9.839, while the level of serum of prolactin in control

group was ranged 3.29± 79.74 ng/ml with a mean of 23.43±22.351, 4 patients (8%) had elevated serum prolactin, mean level was 61.25±25.386. there was no significant statistical difference between the two groups (P-value 0.074). (*Table 5*)

Table 5 Mean of serum prolactin level in patients and control groups.

	Range of prolactin (ng/ml)	Mean±SD	Number of patients with ↑ prolactin	Mean±SD	P-value
Patients group	0.5-54.2	17.84±9.915	10 (20%)	32.11±9.839	0.074
Control group	3.29-79.74	23.43±22.351	4 (8%)	61.25±25.386	

Regarding patients group only, there was no significant statistical correlation between serum total

testosterone and serum prolactin hormones with BMI and WHR (P-value >0.05). (*Table 6*)

Table 6 Mean testosterone and prolactin hormones according to BMI and WHR.

	BMI<25 (Non-obese)	BMI≥25 (obese)	
		WHR<0.85 (non-android obesity)	WHR≥0.85 (android obesity)
Mean testosterone	1.71±1.354	1.91±2.022	1.545±0.984
Mean prolactin	18.424±9.826	29.204±11.419	16.745±9.162

The score of hirsutism for all 50 patients was ranged from 8-21with a mean of 13.12±3.777. Patients were divided into two groups regarding their score of

hirsutism:(*Figure 3*) those with mild hirsutism (score 8-15): they were 36 patients (72%) those with moderate to severe hirsutism (score > 15): they were 14 (28%).

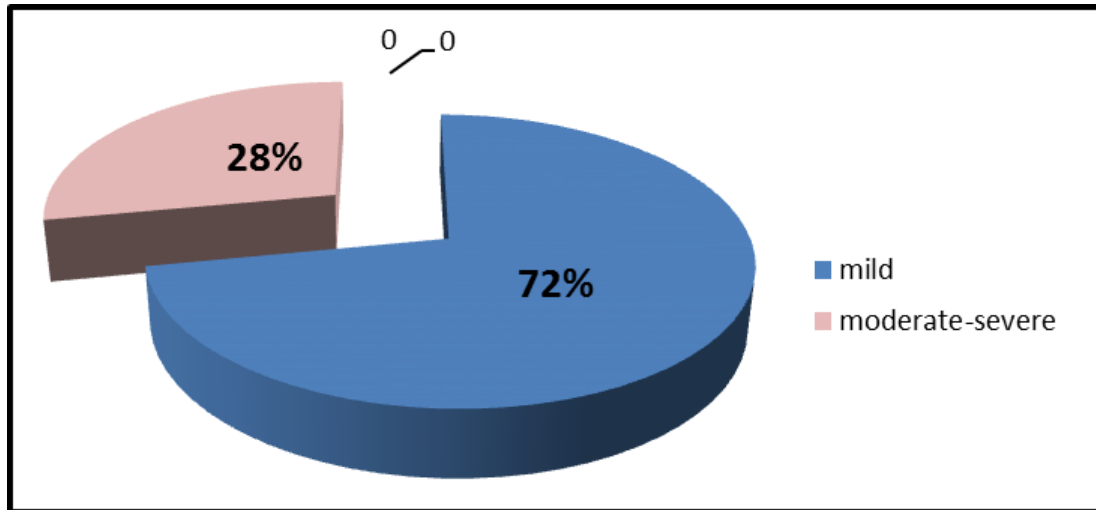


Figure 3 Severity of hirsutism according to Ferriman-Gallway score.

There was significant correlation between score of hirsutism and BMI of the patient (correlation coefficient 0.023), also there was significant relationship between score of hirsutism

and WHR, as patients with android obesity had a higher scores than patients with overall non-android obesity (p-value=0.004). (Table 7)

Table 7 Mean score of hirsutism in patients with android and non-android obesity.

Patients group	Mean score \pm SD	p-value
Android obesity (n=16)	13.1 \pm 6.067	0.004
Non- android obesity (n=14)	11.14 \pm 3.393	

Regarding patients with mild hirsutism (n=36): mean of serum total testosterone was 1.284 \pm 1.994, 21 patients (58.3%) had elevated testosterone with a mean \pm SD of 1.894+0.796, while patients with moderate-severe hirsutism (n=14): mean of serum total testosterone was 2.88 \pm 1.994, 10 patients (71.4%) had

elevated testosterone with a mean \pm SD of 3.79+1.587. Mean testosterone level in patients with severe hirsutism was significantly higher than that of patients with mild hirsutism (P-value 0.0003); while there was no significant relationship between mean serum prolactin levels of the two groups (P-value 0.170). (Table 8)

Table 8 Mean serum testosterone and prolactin according to severity of hirsutism.

	Pt. score 8-15 (n=36)	Pt. score >15 (n=14)	P-value
Mean testosterone ng/ml	1.284 \pm 1.994	2.88 \pm 1.994	0.0003 (significant)
Mean prolactin ng/ml	16.63 \pm 7.434	20.94 \pm 14.402	0.170

Some other cutaneous manifestations of hyperandrogenism were found during examination of the patients,

their frequencies and percentages are found in (Figure 4).

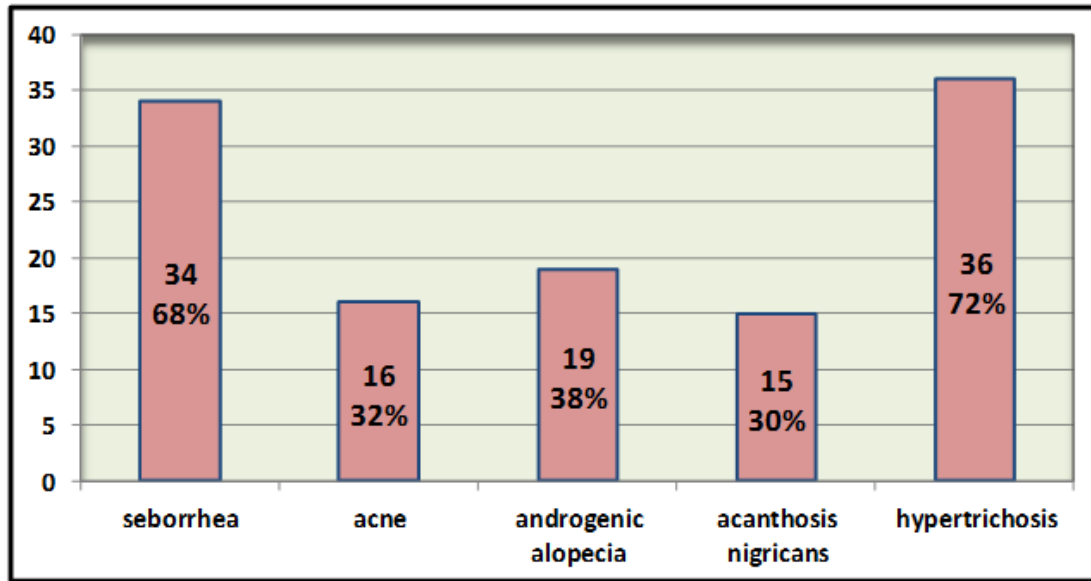


Figure 4 Frequencies of other skin manifestations in patients with hirsutism.

Discussion

Perception of hirsutism is by definition subjective, and women present with a wide variation in severity. Both the severity of hirsutism and the degree of its acceptance are dependent on racial, cultural and social factors [7]. Most women who seek treatment for hirsutism do so for cosmetic reasons because excess body hair outside of cultural norms can be very distressing [10].

All 50 patients who involved in this study showed great degree of distress, because hirsutism interferes with their social, marital life, as well as it significantly affects women perception of her femininity.

Mean age of patients was 27.64±7.266 with age of onset was ranged 12-32 years; these results were comparable with other studies [16, 17].

All patients had slow progression of hirsutism over years (mean duration was 7.52±6.217) and none of them gave a history of rapidly

progressive disease or severe virilization.

Family history was found in 80% of cases, which is a significant result as family history was positive in a rate of 14-50% in most studies [7, 10, 17, 18]. In two previous Iraqi studies, family history was present in 48-49% of patients [4, 19]. Many of our patients had more than one family member who complains from hirsutism (58%), especially sisters and aunts. This tendency to familial clustering might be anticipated since some of the underlying disorders that result in hyperandrogenism have familial basis ex: PCOS and congenital adrenal hyperplasia, also genetic studies conducted to date suggest a polygenic etiology for hyperandrogenic disorders, but the results are inconsistent [2, 7].

Rate of infertility and irregular menstrual cycles among patients group were 12% and 44% respectively. These features might go with the underlying hyper-androgenemia and other endocrine disorders like PCOS in those

patients and it matches other studies and literature [7, 16, 18, 20].

Mean body weight and BMI among hirsute patients were elevated (68.4 ± 7.266) and (27.84 ± 6.579) respectively, it still not statistically different from that of control group. Number of obese and over weight patients was 30 (60%). This is comparable with other Iraqi study [20]. Obesity is a common clinical sign in hirsute women since it reported in 50-80% of patients with PCOS which represents a major etiological factor for hirsutism [1, 8, 19, 20].

16 patients (32%) had android obesity ($WHR \geq 0.85$). Mean weight and BMI are elevated over that of patients with non-android overall obesity whose number was 14 (28%); this is comparable with results of a study about the relationship of mean wt., BMI and WHR with hirsutism in Iran [8]. Hirsutism and menstrual abnormalities are frequently seen in women with android obesity. These conditions are probably due to the peculiar endocrine profile with a preponderance of androgen activity, elevated free testosterone, and low sex-hormone binding globulin concentrations in plasma [8, 21].

31 patients (62%) had elevated serum total testosterone, mean level in those patients was 2.5 ± 1.41 , while mean level of serum total testosterone of control group was 0.4 ± 0.443 ; it was elevated in 4% only. There was highly significant statistical difference between the two groups (p-value 0.0001). This result disagrees with other studies that show less percent of hirsute patients with androgen excess, although still elevated androgen level is more in patients group than control [16, 17, 19].

We expect that androgen excess could be found in even more than 62% of patients because we measure only serum total testosterone

which is less sensitive than serum free testosterone in establishing hyperandrogenemia [1, 7, 22]. 50% of patients with mild hirsutism (score 8-15) have elevated androgen level [1] and consequently much more if patients have severe hirsutism [1, 11]. We agree with literature and other similar studies [18].

20% of patients had elevated serum prolactin, mean level in those patients was 32.11 ± 9.839 , while 4 patients (8%) of control group had elevated serum prolactin; there was no significant statistical difference between the two groups (p-value 0.074). This match well some studies [16, 18]. The exact relationship between hirsutism and prolactin is not clear. The incidence of hirsutism in the amenorrhea-galactorrhea syndrome has been reported as 22-60%. Women with hyperprolactinemia may have an increase in functional androgens through adrenal overproduction and through a decrease in sex hormone-binding globulin caused by a diminution of ovarian estrogen production.

There was no significant correlation between serum total testosterone and serum prolactin levels and BMI or WHR (P-value > 0.05). This result disagrees with some studies [8]. We found that BMI and WHR are more related to score of hirsutism rather than serum androgen level.

Regarding the severity of hirsutism; 36 patients (72%) had mild hirsutism (score 8-15) and 14 patients (28%) had moderate to severe hirsutism (score > 15). This matches well with some studies [17] and not with others [19].

Significant correlation was found between the score of the patients with BMI and WHR (P-value < 0.05). This result is comparable with other studies [8]. Obesity with lowering sex binding globulin can results in a higher

level of free testosterone which can cause hirsutism [22].

Also there was highly significant statistical relationship between the score of the patients and serum androgen level; patients with scores >15 had significantly higher levels of serum androgen than those with lower scores. This result does not match well with some studies [16], but it is comparable with others [2, 15, 17]. A clinician may suspect gross abnormalities of androgen level associated with some endocrine

Recommendations

Hirsutism is frequently seen medical problem in our practice that may *need attention from dermatologists, endocrinologists and even gynecologists* (as many cases represent PCOS).

Hirsutism can give a clue to underlying endocrine disorder especially if patient have high score of hirsutism, thus it *should not be ignored* as it simply represents a cosmetic, racial or familial problem.

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disorder in patients with marked hirsutism [17].

Seborrhea, androgenetic alopecia, acne and acanthosis nigricans were recorded in patients with decreasing frequencies. They represent other features of hyperandrogenism [1, 2, 7, 11], while hypertrichosis is found in 72% of patients especially in those with high score hirsutism. Whether this is related to their high androgen level or due to the hypersensitivity of hair follicles to androgen is still unknown for us and may need further investigations.

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