

Hirsutism in Jordanian Women

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Abstract

Original Research Article

Objectives: The aim of this study was to assess clinical, hormonal profile, and causes of hirsutism in women who had attended the gynecology clinic at King Hussein Medical Center. **Methods:** A retrospective study where clinical information was collected from the medical records of 120 hirsute women, including age, age at onset of the symptoms, duration, rapidity of the onset of the symptoms, parity, menstrual cycle history, age of menarche, family history of hirsutism. Hirsutism was determined by the modified Ferriman-Gallwey index. Laboratory tests and ultrasound were done. **Results:** The mean age at presentation was 24±4.3. The maximum hirsutism Ferriman-Gallwey score was 20 with a range between 10-20. Sixty women (50%) had a score more than 12. The face was the most common site, 67 women had menstrual abnormalities (55.8%) and 46 were obese (body mass index > 25 kg/M²) (38.3%). A positive family history was obtained in 55 women (45.8%). Polycystic ovary syndrome was assumed to be the cause of hirsutism in 68 women (56.8%), 4 women had microadenoma (3.3%) and 3 women had hypothyroidism (2.5%). **Conclusion:** Polycystic ovary syndrome and idiopathic hirsutism, which were evident in about 94% of the studied patients, are the main causes of hirsutism.

Keywords: Hirsutism Ovary, Idiopathic hirsutism, Ferriman-Gallwey index, Jordanian Women.

Abbreviations: FG, Ferriman-Gallwey; BMI, Body mass index; PCOS, polycystic ovary syndrome; DHEAS, dehydroepiandrosterone sulfate; CAH, congenital adrenal hyperplasia; 17-HP, 17-hydroxyprogesterone; LH, Luteinizing hormone; FSH, follicular stimulating hormone; T, total testosterone

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INTRODUCTION

Hirsutism is defined as the presence of terminal hair in females in a male-like pattern [1]. The accepted amount of excessive hair varies in different cultures. Some women accept severe forms of excessive hair growth without concern, while it is a distressing symptom for other women with minimal degrees of hair growth, who may feel desperate [2]. Hirsutism is a common disorder affecting 5–8% of the whole fertile female population [3, 4]. Race and ethnic origin appears to affect terminal hair growth in healthy women significantly. Northern, fair-skinned Europeans have the least amount of terminal hair whereas southern European, dark-skinned Mediterranean women have the greatest amount of terminal hair.

The affected areas of most concern to the women include face, chest, and areola [5]. In severe cases, shoulder area, lower back, upper abdomen, and upper arms are also affected. Women may show clinical signs of virilization such as (male-pattern) hair loss, amenorrhea, increased muscularity, hypertrophy of

vocal cords, or clitoromegaly [2, 6]. Hirsutism results from an increase in circulating androgens concentrations, an increase in the sensitivity of the pilosebaceous unit to normal androgens or a combination of these factors [7].

Hypertrichosis is a non-androgen dependent diffuse increase in vellus hair growth. Hypertrichosis may be congenital, drug induced, or associated with hypothyroidism, malnutrition, anorexia nervosa, and dermatomyositis. It also may occur after severe head injury presenting at sites of skin trauma [8].

Increased androgen effect that results in hirsutism can be familial, idiopathic, or caused by excess androgen secretion by the ovary (e.g., tumors, polycystic ovary syndrome [PCOS]), excess secretion of androgens by adrenal glands (e.g., congenital adrenal hyperplasia [CAH], Cushing's syndrome, tumors), or exogenous pharmacologic sources of androgens [9].

Idiopathic hirsutism is defined as hirsutism in association with regular menses and normal hormonal levels.

Hirsutism is a very common clinical problem in daily practice and women in Jordan with hirsutism usually come to the gynecology clinics for evaluation.

The aim of this study was to evaluate the causes, clinical, and biochemical patterns of hirsutism among a population of Jordanian women attending the gynecology clinic at King Hussein Medical Center.

SUBJECTS AND METHODS

Medical records of 120 hirsute women aged between 15 and 40 years who had attended the gynecology Clinics of King Hussein Medical Center during the period between January 2021–and October 2022 were reviewed.

Medical history including age, age at onset of the symptoms, duration, rapidity of the onset of the symptoms, parity, menstrual cycle history, age of menarche, family history of hirsutism, and symptoms of virilization (deepening of voice, decreased breast size, increased muscularity, and amenorrhea) was obtained.

Women who were pregnant or lactating were excluded from the study. Excess body and facial terminal hair growth were measured using a modified Ferriman-Gallwey (mF-G) hirsutism score. This test was done by adding hair scores (0 = none, 4 = frankly virile) in 9 different body locations. In the face (chin, upper lip), areola and chest, upper back, lower back, upper abdomen, lower abdomen, thighs, and upper arms. A total score > 8 is considered hirsute based on the 95th percentile of the data originally collected by Ferriman and Gallwey [10].

Weight and length of the women were measured. The body mass index (BMI) was calculated as kilograms per square meter. Obesity was defined as a BMI \geq 25 kg/m².

The studied women were also examined for clinical signs of virilization, acanthosis nigricans and galactorrhea.

Serum levels of total testosterone (T), free T, dehydroepiandrosterone sulfate (DHEAS), 17-hydroxyprogesterone (17-HP), prolactin and thyroid-stimulating hormone were obtained without regard to the time of the cycle or the day.

Luteinizing hormone (LH), follicular stimulating hormone (FSH) levels were obtained in the second or third day of the cycle.

Pelvic sonography in the early follicular phase (days 5-9 of the menstrual cycle) was carried out. According to the 2003 international consensus [11], PCO was defined as the presence of at least two of the following signs after exclusion of other causes of androgen excess: 1) oligoovulation or anovulation which usually manifested as oligomenorrhea or amenorrhea; 2) elevated levels of circulating androgens (hyperandrogenemia) or clinical manifestations of androgen excess (hyperandrogenism); and 3) Polycystic ovaries in ultrasonography.

Biochemical hyperandrogenism was defined as serum T levels above 60 ng/dl (\geq 2.08 nmol/liter), free T levels of 3 pg/ml (\geq 10.34 pmol/liter) or more, and/or serum DHEAS levels of 3000 μ g/liter (7.8 \geq mol/liter) or more. (21- hydroxylase deficiency was excluded by a basal follicular phase 17-hydroxyprogesterone (17-OHPG) level <6.0 ng/mL [12].

The diagnosis of idiopathic hirsutism was made if there is no menstrual irregularities or any other signs or symptoms of hyperandrogenism, except for hirsutism, and a normal hormone level [14].

STATISTICAL ANALYSIS

Data were entered, checked and analyzed using the SPSS package. Data were expressed as mean \pm SD for quantitative variables, and number and percentage for qualitative ones.

RESULTS

120 women with hirsutism were studied. The age range varied from 16 to 45 years. Most of them were 21-25 years (35%). The age of the onset of disease ranged from 14 to 30 years. The duration of symptoms ranged from 2 to 15 years. 80(66.6 %) of women studied were married and 40 women (33%) had history of infertility.

A positive family history of hirsutism was obtained in 55(45.8%) women; either their mothers or sisters having the same conditions.

8 women complained of galactorrhea (6.6%), 67 (55.8%) women had menstrual abnormalities in the form of oligomenorrhea in 50(41.6%) women, amenorrhea in 6 (5%) women and menorrhagia in 11(9.16%) women.

The maximum hirsutism Ferriman Gallwey score was 20 with a range between 10-20. 60 women (50%) had score more than 12.

The face was the most common site, while the chest and abdomen were the next most common sites. 72 women (60.0)% had acne of varying grades. 46 women were obese (body mass index > 25 kg/M²) (38.3%).

Table 1: Characteristics of the studied patients

Age	24±4.3
Age of onset of the disease	21±8
Duration of the symptoms	3±5.3
Marital Status	
Married	(80/120)66.6%
Single	(40/120)33.3%
Menstrual irregularities	(67/120)55.8%
Infertility	(40/120)33.3%
Presence of Acne	(72/120)60.0%
Body Mass Index>25	(46/120)38.3%
Family history of hirsutism	(55/120)45.8%

Serum total testosterone levels were raised in 32 women (26.6%), free testosterone in 12 women (10%) androstenedione in 36 women (30%). Dehydroepiandrosterone sulfate was raised in 19

women (15.8%), FSH in 2 women (1.6%) and LH in 8(6.6%) patients, The LH/FSH ratio was elevated (>2) in 24women (20%) and serum prolactin was increased in 13 (10.8%) women.

Table 2: Frequency of Hormonal abnormalities in the studied hirsute women

Hormone	Value	Number (%)
Total testosterone (ng /ml)	60+ 20	32(26.6%)
Free testosterone(ng/ml)	1.1+ 0.4	12(10%)
Androstenedione(ng/ml)	2.9+ 1.1	36(30%)
Dehydroepiandrosterone sulfate= DHEA-S (ng/ml)	970+ 230	19(15.8%)
Follicle stimulating Hormone =FSH(IU/L)	6.3+ 1.3	2(1.6%)
Luteinizing hormone =LH (IU/L)	8.4+ 3.1	8(6.6%)
LH/FSH ratio>2	1.9+ 1.4	24(20%)
Prolactin(ng/ml)	18+ 6	13(10.8%)

Ultrasonographic findings consistent with polycystic ovary syndrome were seen in 68(56.8%) patients and 3 women had dermoid cysts (2.5%). 8 women complained of galactorrhea (6.6%), 4(3.3%) of them underwent brain MRI which revealed microadenoma.

Thyroid symptoms and signs suggestive of hypothyroidism were seen in3 (2.5%) women, TSH, T3, T4 levels were consistent with hypothyroidism. None of the patients had elevated level of17-hydroxyprogesterone (17-o-HPG).

Table 3: Causes of hirsutism in the studied patients

Polycystic ovary syndrome	68 (56.8%),
Idiopathic	45(37.5%),
Microadenoma	4(3.3%),
Hypothyroidism	3(2.5%),

DISCUSSION

Hirsutism is a common clinical condition that usually has a benign course. In rare cases, however, it may be the presenting feature of a serious underlying disease which needs diagnosis and aggressive treatment [6].

PCOS has an estimated prevalence of 5 –10% [15]. It is the most common endocrine abnormality affecting women in the reproductive age group.

In this study, the majority of the women were young with mean age 24±4.3 years. Hirsutism in this age group is generally of benign nature. The most common cause was PCOS followed by idiopathic hirsutism 45(37.5%), microadenoma in 4 women

(3.3%) and hypothyroidism was seen in 3women (3.3%).

The reported the incidence of PCO in of hirsute women in the United Arab Emirates is 91 % [16], 75% in India [17], 37.3% in Kashmir, India [18]. 53% in Mexico [19], 33% In Finland [20], 60% in England [21], 70 – 78% in the United States. (22) and 49% in Iran [23]. This is comparable with our results as the incidence of PCOS in Jordanian women is (56.8%).

A positive family history of hirsutism was obtained in (45.8%) of our Jodanian women; either their mothers or sisters were hirsute. Hirsutism may occur on a familial basis. This may be due to the familial clustering of some of its underlying diseases (e.g., CAH or PCOS). Azizz *et al.*, had shown that 35% of mothers

and 40% of sisters of women with PCOS are affected by PCOS, themselves [24].

Approximately (38.3%) of women studied were obese (body mass index > 25 kg/M²). This is much higher than the prevalence of obesity in Mexico (18%) and Finland (7%) [19, 20].

In our study, the diagnosis of idiopathic hirsutism applied only to hirsute women with regular menses and normal hormonal levels. (37.5%) of our women were diagnosed as idiopathic hirsutism, our figure is almost the same as in Finland and England [20, 21] and higher than what is reported in the United Arab Emirates (5%), in the USA 15%, in Saudi Arabia 11% [16, 22, 25].

The clinical presentation and the etiology of hirsutism in our women does not differ from what is described in the literature, further studies are needed.

REFERENCES

- Lee, H. J., Ha, S. J., Lee, J. H., Kim, J. W., Kim, H. O., & Whiting, D. A. (2002). Hair counts from scalp biopsy specimens in Asians. *Journal of the American Academy of Dermatology*, 46(2), 218-221.
- Rabinowitz, S., Cohen, R., & Le Roith, D. (1983). Anxiety and hirsutism. *Psychological reports*, 53(3), 827-830.
- Knochenhauer, E. S., & Azziz, R. (1995). Advances in the diagnosis and treatment of the hirsute patient. *Current Opinion in Obstetrics and Gynecology*, 7(5), 344-350.
- Azziz, R., Carmina, E., & Sawaya, M. E. (2000). Idiopathic hirsutism. *Endocrine reviews*, 21(4), 347-362.
- Zargar, A. H., Wani, A. I., Masoodi, S. R., Laway, B. A., Bashir, M. I., & Salahuddin, M. (2002). Epidemiologic and etiologic aspects of hirsutism in Kashmiri women in the Indian subcontinent. *Fertility and sterility*, 77(4), 674-678.
- de Berker, D. A. R., Messenger, A. G., & Sinclair, R. D. (2004). Disorders of hair. In: Burns, T., Breathnach, S., Cox, N., Griffiths, C., eds. *Rook's Textbook of Dermatology*. 7th ed. Oxford: Blackwell Science.
- Conn, J. J., & Jacobs, H. S. (1997). The clinical management of hirsutism. *European Journal of Endocrinology*, 136, 339-348.
- Leung, A. K., & Robson, W. L. M. (1993). Hirsutism. *International journal of dermatology*, 32(11), 773-777.
- Gilchrist, V. J., & Hecht, B. R. (1995). A practical approach to hirsutism. *American family physician*, 52(6), 1837-1846.
- Ferriman, D., & Gallwey, J. D. (1961). Clinical assessment of body hair growth in women. *The Journal of Clinical Endocrinology & Metabolism*, 21(11), 1440-1447.
- The Rotterdam ESHRE /ASRM -sponsored PCOS Consensus Workshop Group. (2004). Revised 2003 consensus on diagnostic criteria and long term health risks related to polycystic ovary syndrome. *Fertil Steril*, 81, 19-25.
- Azziz, R., Hincapie, L. A., Knochenhauer, E. S., Dewailly, D., Fox, L., & Boots, L. R. (1999). Screening for 21-hydroxylase-deficient nonclassic adrenal hyperplasia among hyperandrogenic women: a prospective study. *Fertility and sterility*, 72(5), 915-925.
- Rossi, R., Tauchmanova, L., Luciano, A., Valentino, R., Savastano, S., Battista, C., ... & Lombardi, G. (2001). Functional hyperandrogenism detected by corticotropin and GnRH-analogue stimulation tests in women affected by apparently idiopathic hirsutism. *Journal of endocrinological investigation*, 24(7), 491-498.
- Carmina, E. (1998). Prevalence of idiopathic hirsutism. *European journal of endocrinology*, 139(4), 421-423.
- Archer, J. S., & Chang, R. J. (2004). Hirsutism and acne in polycystic ovary syndrome. *Best Practice & Research Clinical Obstetrics & Gynaecology*, 18(5), 737-754.
- Gatee, O. B., Al Attia, H. M., & Salama, I. A. (1996). Hirsutism in the United Arab Emirates: a hospital study. *Postgraduate medical journal*, 72(845), 168-171.
- Mithal, A., Ammini, A. C., Godbole, M. M., Khurana, M. L., Raj, D., Karmarkar, M. G., & Ahuja, M. M. S. (1988). Late-onset adrenal hyperplasia in North Indian hirsute women. *Hormone Research in Paediatrics*, 30(1), 1-4.
- Zargar, A. H., Wani, A. I., Masoodi, S. R., Laway, B. A., Bashir, M. I., & Salahuddin, M. (2002). Epidemiologic and etiologic aspects of hirsutism in Kashmiri women in the Indian subcontinent. *Fertility and sterility*, 77(4), 674-678.
- Morán, C., Tapia, M. D. C., Hernández, E., Vázquez, G., García-Hernández, E., & Bermúdez, J. A. (1994). Etiological review of hirsutism in 250 patients. *Archives of medical research*, 25(3), 311-314.
- Erkkola, R., & Ruutiainen, K. (1990). Hirsutism: definitions and etiology. *Annals of Medicine*, 22(2), 99-103.
- O'Driscoll, J. B., Mamtara, H., Higginson, J., Pollock, A., Kane, J., & Anderson, D. C. (1994). A prospective study of the prevalence of clear-cut endocrine disorders and polycystic ovaries in 350 patients presenting with hirsutism or androgenic alopecia. *Clinical endocrinology*, 41(2), 231-236.
- Hunter, M. H., & Carek, P. J. (2003). Evaluation and treatment of women with hirsutism. *American family physician*, 67(12), 2565-2572.

23. Farnaghi, F., & Seyrafi, H. (2002). Descriptive study of patients with hirsutism in Razi Hospital, Tehran. *Iranian J Dermatol*, 1, 21-25.
24. Azziz, R., & Kashar-Miller, M. D. (2000). Family history as a risk factor for the polycystic ovary syndrome. *Journal of pediatric endocrinology & metabolism: JPEM*, 13, 1303-1306.
25. Al-Ruhaily, A. D., Malabu, U. H., & Sulimani, R. A. (2008). Hirsutism in Saudi females of reproductive age: a hospital-based study. *Annals of Saudi medicine*, 28(1), 28-32.