Sarcouncil journal of Medical sciences

ISSN(Online): 2945-3526

Volume- 02| Issue- 01| 2023

Research Article

Received: 10-11-2022 | Accepted: 21-12-2022 | Published: 17-01-2023

Hirsutism among Women Attending Al-Yarmouk Teaching Hospital, (Clinical and Investigative Study)

Dr. Ban Muhi Mohammed[1], Dr. Wasan Burhan Abdulatef[2] and Dr. Ammar Faisal Khalaf[3]

[1]*Specialist dermatolagist in Ibn sina hospital IRAQ.*

[2]*Specialist dermatologist in alyarmok teaching hospital IRAQ.*

[3]*Specialist dermatologist in alyarmok teaching hospital IRAQ.*

Abstract: Background: Hirsutism is a cosmetic problem facing dermatologists, gynecologists & buticians. **Aim of study:** The aim of the study is to assess Hirsutism as cosmetic and disfiguring problem. **Patients and Methods:** Outpatient hospital based clinical and investigative study during one year period for female patients attending Al-Yarmouk Teaching Hospital for the period from January 2012 to January 2013. Each patient was interviewing for age, family history of hirsutism, medications and systemic diseases, menstrual and obstetric history, and associated signs of hyperandrogenism. Women who were pregnant or lactating, those who received drugs that could interfere with the hormonal and metabolic studies were excluded from the study. Quantification of hirsutism was done using Ferriman-Gallwey score(appendix1). Body mass index (weight/height²) was also calculated. The patient was examined clinically for any associated conditions like acne, alopecia ,acanthosis nigricans(signs &symptoms of hyperandrogenemia). Investigations included were estimation of thyroid hormones, total testosterone, luteinizing hormone , follicular stimulating hormone , serum prolactin levels ,and ultrasonography for adrenals and ovaries was done also. Menstrual cycle history included a general review since menarche till the last year, to detect the presence of any menstrual irregularities. The study exclude all cases with metabolic syndrome, cases treated with hormones and or LASER therapy. A data were processed in computer program Microsoft-office-Excel-2007 & EPI-info Version 6. **Results:** One hundred five patients with hirsutism aged between 15 and 50 years Fifty seven percent of patients were aged 20 - 30 years. The average F-G score was 10.53±7.19 Associated signs of hyperandrogenism were acne (17.85%), menstrual irregularities (23.8%), androgenic alopecia (21%), acanthosis nigricans (9.45%), Polycystic ovaries were detected in 31.4% patients, luteinizing hormone LH (19%), Follicle-stimulating hormone FSH (3.8%) ,LH/FSH ratio increase in (20%) and 14.28% patients had elevated serum testosterone levels. Family history of hirsutism was present in 33.3% patients **Conclusion:** This study shows that Hirsutism is a common cosmetic problem in Iraqis women and most patients were considered to be idiopathic followed by familial cases.

Keywords: Hirsutism, Among, Women.

INTRODUCTION

Hirsutism:

Hirsutism is defined as the excessive growth of thick dark hair in locations where hair growth in women usually is minimal or absent .Such male-pattern growth of terminal body hair usually occurs in androgen-stimulated locations, such as the face, chest, and areolae. In severe cases shoulder area, lower back, upper abdomen, and upper arms are also affected. Patients may show clinical signs of virilization such as frontoparietal (male-pattern) hair loss, acne, amenorrhea, and sometimes masculinization of the muscle mass, hypertrophy of vocal cords, or clitoromegaly (Ferriman, D. and Gallwey, J.D. 1961; Mofid, A. *et al.*, 2008; Fattah, N. S. A., and Darwish, Y. W. 2009).

Hirsutism is a medical problem that can have significant psychological consequences. Most women seek treatment for hirsutism for cosmetic condition, because excess body hair outside cultural norms can be very distressing (emedicin.medscape.com).

Hirsutism may be a sign of an underlying endocrine abnormality, it can result from an overproduction of androgen, or a sign of increased sensitivity of hair follicles (pilosebaceous unit) to normal levels of circulating androgen or both of them. The main sources of androgens in women are the ovaries, adrenal glands and peripheral adipose tissues. Up to 15% of patients have idiopathic hirsutism in which there are regular ovulatory cycles and normal androgen levels (emedicine.medscape.com; Carmina, E. 1998). Hirsutism may be associated with obesity, insulin resistance, diabetes, polycystic ovary syndrome (PCOS), hypertension, infertility, and menstrual irregularities (Ferriman, D. and Gallwey, J.D. 1961; Mofid, A. *et al.*, 2008; Carmina, E. 1998).The threshold level for acceptable amount of excessive hair varies in different cultures. Some women with minimal degrees of hirsutism may be psychologically devastated, while others accept more severe forms without much concern (Mofid, A. *et al.*, 2008).

Although the terms hirsutism and hypertrichosis often are used interchangeably, hypertrichosis actually refers to excess hair (terminal or vellus) in areas that are not predominantly androgen dependent. Whether a patient is hirsute often is difficult to judge because hair growth varies among individual women and across ethnic groups. What is considered hirsutism in one

*Corresponding Author: Dr. Ban Muhi Mohammed

DOI-https://doi.org/10.5281/zenodo.7545202

culture may be considered normal in another (Carmina, E., & Lobo, R. A. 2001). For example, women from the Mediterranean and the Indian subcontinent have more facial and body hair than do women from East Asia, sub-Saharan Africa, and Northern Europe. Dark-haired, darkly pigmented individuals of either sex tend to be more hirsute than blond or fair-skinned persons (Carmina, E., & Lobo, R. A. 2001).

In most cases, hirsutism is a benign condition and is primarily of cosmetic concern. However, when hirsutism is accompanied by masculinizing signs or symptoms, particularly when these arise well after puberty, hirsutism may be a manifestation of a more serious underlying disorder such as an ovarian or adrenal neoplasm. Fortunately, these disorders are rare. Some families are affected more than the others (Carmina, E., & Lobo, R. A. 2001).

It should be mentioned that the affected areas most concern to the patients include upper lip, chin, chest, and areola (Carmina, E. 1998). The degree of hirsutism in different parts of the body is assessed by the Ferriman and Gallwey score (Ferriman, D., & Gallwey, J. D. 1961). Hirsutism was classified as mild (score 8 – 16), moderate (score 17 – 24), and severe (score >24) (Ferriman, D., & Gallwey, J. D. 1961).

Stress has been proposed as a contributing factor in hirsutism (Mofid, A. *et al.*, 2008).

Anatomy and physiology:
Hair has no vital function in humans, yet its psychological functions are extremely important (Dry, F.W. 1926).

Types of hair:
Different types of hair may be produced by different kinds of follicle, and the type of hair produced in any particular follicle can change with age or under the influence of hormones (Dry, F.W. 1926).

In humans, a prenatal coat of fine soft unmedullated and usually unpigmented hair, known as lanugo. Postnatal hair may be divided at the extreme into two kinds: vellus and terminal hair (Hughes, C.L. 2003).

Vellus hair which is soft, unmedullated, occasionally pigmented and seldom more than 2 cm long; it is fine hair (peach fuzz) that covers the body of children and adults; growth not affected by hormones. Beard hair in women and children is vellus (Hughes, C.L. 2003).

Terminal hair, which is longer, coarser and often medullated and pigmented.

Before puberty, terminal hair is normally limited to the scalp, eyebrows and eyelashes (Pinkus, H. 1958).

After puberty, secondary sexual ‘terminal’ hair is developed from vellus hair in response to androgens (Pinkus, H. 1958). it is Thick pigmented hair found on scalp, beard, axillae, pubic area; growth is influenced by hormones. However, there is a range of intermediate kinds. Intermediate Hair Shows the characteristics of vellus and terminal hairs (Editorial, 1975).

Physiology:
Seasonal moulting is regulated by the endocrine system under the influence of environmental signals. The most important of these is change in day length (the photoperiod) (Bissonnette, T. H. 1935; Harvey, N. E., & Macfarlane, W. V. 1958).Temperature may act as a modifying factor in some species. Changing levels of melatonin production by the pineal gland have a key role in orchestrating endocrine control of seasonal hair growth (Allain, D., & Rougeot, J. 1930; Rose, J. *et al.*, 1984; Rose, J. *et al.*, 1987). pinealectomy prevents seasonal moulting, whereas administration of melatonin advances onset of the growth of the winter coat and prevents growth of the summer coat. Prolactin production by the pituitary correlates inversely with melatonin levels, being raised during the summer and falling during the winter (Badura, L. L., & Goldman, B. D. 1992).

Prolactin receptors have been identified in the hair follicle, suggesting that prolactin can affect hair growth directly conversely; thyroid hormones accelerate the onset of follicular activity, whereas thyroidectomy or treatment with propylthiouracil delays it (Choy, V. J. *et al.*, 1995).

Seasonal moults are also delayed by testosterone and accelerated by thyroxine in other species (Maurel, D. *et al.*, 1987). Hormones also act on the anagen phase of hair growth (Ebling, F.J. 1990). Estradiol and thyroxine both reduce the duration of anagen in rats, but estradiol decreases the rate of hair growth, whereas thyroxine has the opposite effect, suggesting these hormones have different points of action (Oh, H. S., & Smart, R. C. 1996). The best example of a systemic influence on the human hair growth cycle is pregnancy (Lynfield,

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Y. L. 1960). During pregnancy, there is an increase in the proportion of follicles in anagen, although it is not clear whether this is caused by prolongation of anagen or more rapid shedding of telogen hairs, as there is also a reduction in hair density during the second and third trimesters (Pecoraro, V. *et al.*, 1969). Following childbirth, large numbers of follicles enter telogen, leading to increased shedding from about 3 months postpartum (postpartum telogen effluvium) (Pecoraro, V. *et al.*, 1969).

Telogen shedding may also be caused by a number of drugs and by febrile and other catabolic illnesses (Kligman, A. M. 1961).

The hair cycle: Hair follicles undergo a repetitive sequence of growth and rest known as the hair cycle.

Anagen: Growth phase; lasts variable periods of time depending on body site, e.g., scalp, eyebrows. Duration: 1–6 years; average 3 years; varies with age; Anagen hair matrix has rapidly proliferating epithelial cells; exquisitely sensitive to drugs, growth factors, hormones, stress; immunologic and physical injury (Harding, H. W., and Rogers, G. E. 1971).

Destruction of epithelial stem cells results in permanent hair loss (Harding, H. W., and Rogers, G. E. 1971).

Catagen: Apoptosis-driven phase between telogen and anagen phase. Duration: few weeks.

Telogen : the period between the completion of follicular regression and the onset of the next anagen phase is termed telogen.

Exogen: Active process of hair shaft shedding (Courtois, M. *et al.*, 1995)

Pathophysiology:

Hirsutism can be caused by abnormally high androgen levels or by hair follicles that are more sensitive to normal androgen levels. Therefore, increased hair growth often is observed in patients with endocrine disorders characterized by hyperandrogenism, which may be caused by abnormalities of the ovaries or the adrenal glands (Ferriman, D. and Gallwey, J.D. 1961). The physiologic mechanism proposed for androgenic activity consists of the following 3 stages:

- Production of androgens by the adrenals and ovaries
- Androgen transport in the blood on carrier proteins (principally sex-hormone–binding globulin)
- Intracellular modification and binding to the androgen receptor (Ferriman, D. and Gallwey, J.D. 1961) .
- In short, central overproduction of androgen, increased peripheral conversion of androgen, decreased metabolism, and enhanced receptor binding are each potential causes of hirsutism. For circulating testosterone to exert its stimulatory effects on the hair follicle, it first must be converted into its more potent follicle-active metabolite, dihydrotestosterone. The enzyme, 5-alpha-reductase, which is found in the hair follicle, performs this conversion (Ferriman, D. and Gallwey, J.D. 1961).

Overproduction of androgens results in an increased hair follicle size, hair fiber diameter, and duration of time hair follicles spend in the anagen (growth) phase. In addition to a change in hair quality and volume, oilier skin and hair may result from excess androgen secretion. The distribution of hair in women with high androgen levels is also altered. Excessive hair growth occurs in androgen-sensitive regions, but hair loss occurs on the scalp. The severity of hirsutism does not correlate directly with the level of increased circulating androgens because of individual differences in conversion to 5-alpha-reductase and androgen sensitivity of hair follicles (Ferriman, D. and Gallwey, J.D. 1961).

Testosterone stimulates hair growth, increasing the size and intensifying the pigmentation of hair. Estrogens act in opposition, slowing growth and producing finer, lighter hairs. Progesterone has minimal effect on hair growth (Ferriman, D. and Gallwey, J.D. 1961).

The amount of free testosterone—the biologically active androgen that, after conversion to dihydrotestosterone, causes hair growth—is regulated by sex hormone binding globulin (SHBG). Lower levels of SHBG increase the availability of free testosterone. SHBG levels decrease in response to the following (Ferriman, D. and Gallwey, J.D. 1961)**:**

- Exogenous androgens
- Certain disorders that affect androgen levels, such as polycystic ovarian syndrome (PCOS)
- Congenital or delayed-onset adrenal hyperplasia.

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- Cushing syndrome
- Obesity
- Hyperinsulinemia
- Hyperprolactinemia
- Excess growth hormone
- Hypothyroidism

Conversely, SHBG levels increase with higher estrogen levels, such as the levels that occur during oral contraceptive therapy. The resultant increased SHBG levels lower the activity of circulating testosterone (Ferriman, D. and Gallwey, J.D. 1961).

Epidemiology:
Familial hirsutism is found most commonly in southern European and South Asian countries, in which it is considered to be a normal trait (Mofid, A. *et al.*, 2008).

Hirsutism indicative of underlying endocrinopathy varies from culture to culture, depending on the incidence of the various endocrinopathies in a particular society (Mofid, A. *et al.*, 2008).

Hirsutism is a symptom, rather than a disease. Primarily, hirsutism is of cosmetic and psychological concern; however, it may indicate the presence of more serious associations, such as adrenal hyperplasia and ovarian tumors, particularly if it develops well after puberty. Familial hirsutism is noted most frequently in dark-skinned white persons. It is uncommon in sub-Saharan and African American blacks and is observed least commonly in East Asians and Native Americans (Mofid, A. *et al.*, 2008). he onset of hirsutism depends on its cause. Familial or ethnic hirsutism typically begins during puberty. Hirsutism resulting from congenital adrenal hyperplasia (CAH) begins early in childhood, while late-onset CAH and PCOS often have onset after puberty. The growth of facial hair commonly observed in postmenopausal women may be caused by unopposed androgen (Mofid, A. *et al.*, 2008).

Causes:
1. Ovarian causes of hirsute:
PCOS is a disorder that affects androgen levels. The most common cause of androgen excess and hirsutism is PCOS. Virilization is minimal, and hirsutism is often prominent. Polycystic ovarian syndrome (PCOS) was diagnosed by Rotterdam criteria: presence of two of the three elements: clinical or biological hyperandrogenism, polycystic ovaries and chronic unovulation (Rotterdam ESHRE/ASRM. 2004). Characteristic features include menstrual irregularities, dysmenorrhea, occasional glucose intolerance and hyperinsulinemia, and, often, obesity. The hyperinsulinemia is believed to hyperstimulate the ovaries into producing excess androgens. Women with PCOS may show other cutaneous manifestations of androgen excess in addition to hirsutism, such as recalcitrant acne, acanthosis nigricans, and alopecia on the crown area of the scalp (Mofid, A. *et al.*, 2008).

Hirsutism may also be seen in women with the following ovarian conditions, most of which are associated with virilization (Fattah, N. S. A., and Darwish, Y. W. 2009):

- Luteoma of pregnancy
- Arrhenoblastomas
- Leydig cell tumors
- Hilar cell tumors
- Thecal cell tumors

2. Familial hirsutisim Familial hirsutism is not associated with androgen excess. Familial hirsutism is both typical and natural in certain populations, such as in some women of Mediterranean or Middle Eastern ancestry (Fattah, N. S. A., and Darwish, Y. W. 2009).

3. Drug-induced hirsutism
Drugs such as phenytoin, minoxidil, diazoxide, cyclosporine, streptomycin, psoralen, penicillamine, high-dose corticosteroids, metyrapone, phenothiazines, acetazolamide, and hexachlorobenzene presumably exert their effects independently of androgens. The exact mode of action of these drugs on hair follicles is not known, but the same mechanisms do not appear to be involved in all patients (Fattah, N. S. A., and Darwish, Y. W. 2009). Drug-induced hirsutism can be distinguished from drug-induced hypertrichosis, in which a uniform growth of fine hair appears over extensive areas of the trunk, hands, and face and is unrelated to androgen-dependent hair growth (Fattah, N. S. A., and Darwish, Y. W. 2009).

4. Adrenal causes of hirsutisim:
Congenital adrenal hyperplasia (CAH)in children (i.e., the classic formofadrenal hyperplasia) may cause hirsutism. These children may be born with ambiguous genitalia, symptoms of salt wasting, and failure to thrive (Mofid, A. *et al.*, 2008).

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Late-onset CAH usually occurs as an incomplete version of CAH and affects approximately 1-5% of women who are hyperandrogenism. In patients with late-onset CAH, hirsutism (without salt-wasting symptoms) may not develop until adulthood (Fattah, N. S. A., and Darwish, Y. W. 2009).

Signs of virilization and menstrual irregularities may not be observed until puberty or adulthood. Patients have clinical features that resemble PCOS.

Hirsutism and oligomenorrhea suggest 21-hydroxylase deficiency (elevated 17-alpha-hydroxyprogesterone). Another uncommon disorder is 3-beta-, 11-hydroxysteroid dehydrogenase deficiency (elevated 3-beta-, 11-hydroxysteroid levels), which may result in early- or late-onset CAH (Fattah, N. S. A., and Darwish, Y. W. 2009).

Cushing syndrome is a non-congenital form of adrenal hyperplasia characterized by an excess of adrenal cortisol production. The excessive growth is predominantly vellus (non–androgen dependent) hair (Fattah, N. S. A., and Darwish, Y. W. 2009).

Adrenocortical carcinoma is rare malignancy in which approximately 60%of patient presented with steroid hormone excess (Ng, L., and Libertino, J. M. 2003).

5. Other causes:
Less common but potentially serious disorders that may be associated with hirsutism include anorexia nervosa, acromegaly, hypothyroidism, hyperprolactinemia, and porphyria (Mofid, A. *et al.*, 2008).

Investigation:
1. Labrotary investigations:
Follicle-stimulating hormone(FSH) ,Luteinizing hormone (LH) ,serum prolactin, Total testosterone, Dehydrobpiandrosteroid sulfate.
2. Ultrasonography for overies and adrenal glands.
3. Computerised tomography and Magnetic resonance imaging for adrenal tumors (Klaus, W. *et al.*, 2005).

AIM OF THE STUDY

The aim of the study is to assess hirsutism as cosmetic and disfiguring problem.

PATIENTS AND METHODS

Outpatient hospital based clinico-epidemological and investigative study during one year period of female patients attending Al-Yarmouk Teaching Hospital for the period from January 2012 to January 2013.

Patients taken in the study, include all patient attending the outpatient dermatology& Venereology department who were interviewed by using a questionnaire including age ,time of onset of the disease ,marital status, parity ,history of drug use ,medical history, family history (second degree),menstrual cycle history included a general review since menarche till the last year ,to detect the presence of any menstrual irregularities, obstetric history.

The menstrual patterns were defined as regular cycles, if length of cycle was between 24 and 40 days. The cycle was considered irregular if the patient had either polymenorrhea (bleeding at intervals of less than 22 days), oligomenorrhea (bleeding at intervals of greater than 40 days), or amenorrhea (absence of menstruation for 12 months or more) (Hammond, C.B. and Riddick, D.H. 1999).

Examination for associated signs and symptoms of hyperandroginism:

- Acanthosis nigricans
- Obesity
- Pelvic mass
- Signs or symptoms of virility
- Signs or symptoms of Cushing syndrome
- Acne
- Alopecia

A quantitative method of measuring hair growth, the Ferriman-Gallwey model, allows for the determination of the severity of hirsutism by assessing the extent of hair growth in 9 key anatomic sites, as follows (Ferriman, D., & Gallwey, J. 1961):

- Face (particularly, moustache, beard, and temple areas).
- Chest
- Areolae
- Linea alba
- Upper back
- Lower back
- Inner thighs
- External genitalia

The overall score in all of the affected parts was assessed. Hirsutism was classified as mild (score 8

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– 16), moderate (score 17 – 24), and severe (score >24) (Ferriman, D., & Gallwey, J. 1961).

Body mass index (BMI) (weight/height2) was also calculated.BMI<18.5 KG/m2 considered under weight, BMI < 25 kg/m2 considered normal weight, 25-29.9 Kg/m2 over weight, 30-39.9Kg/m2 obesity while >40Kg/m2 considered morbid obesity.

Investigations included were estimation of total testosterone, luteinizing hormone (LH), follicular stimulating hormone (FSH) and serum prolactin levels, thyroid hormones and a transabdominal ultrasonography with two dimension was done for all patients (Transvaginal ultrasound was rejected by the patients).

Blood sampling was done in the early follicular phase after overnight fasting for 10 – 12 hours, blood samples were collected around 8 – 9 AM to measure serum levels of follicular stimulating hormone, luteinizing hormone, prolactin and total testosterone.

Women who were pregnant or lactating and cases treated with hormones and or LASER therapy were excluded from the study.

Data processed using computer program are Microsoft-office-Excel-2007 for descriptive statistics including range mean and standard deviation.

And EPI-info Version 6 for analytic statistic using student's t. test for comparing between 2 means & ANOVA for comparison of more than 2 means.

RESULTS

One hundred and five hirsute patients aged between 15 and 50 years were studied with the mean ±SD age of 27.89±8.32 yrs patients.

The largest number of patients 60 (57%) were 20 to 30 years old, 15-20 years were 14 (13.3%), 31-40 years were 24 (22.85%), and 41-50 years were 7(6.66%).(as shown in : Table1)

Table No. 1: The age distribution of hirsute patients

Age group in years	No.	%	Cumulative%
15-20	14	13.3	13.34
20-30	60	57	70.48
31-40	24	22.85	93.34
41-50	7	6.66	100
Total	105	100	

There were 50 married patients (47.6%), among whom 41 had children. Twenty-nine patients (27.6%) had a BMI < 25 Kg/m2 and, thus, were considered normal weight, 54 patients (51.42%) had a BMI 25-29.9 Kg/m2 thus were considered overweight, and 22(20.9%) with BMI 30-39.9 Kg/m2 and thus considered as obese & non >40 Kg/m2 morbid Obesity. as shown in (Table 2).

Table No 2: The distribution of patients according to BMI and Hormones**.**

BMI	**< 25**	**25-29.9**	**30- more**	**ANOVA, P-value**
Hormones	**Mean± SD (n=29)**	**Mean± SD (n=54)**	**Mean± SD (n=22)**	
FSH	4.351±1.831	3.864±3.357	4.502±1.932	>0.05
LH	7.00±5.186	6.227±5.081	7.531±4.342	>0.05
LH/FSH	1.55±0.84	2.025±1.269	1.621±0.739	>0.05
Testosterone	0.72±0.563	0.581±0.465	0.615±0.489	>0.05
Prolactin	14.888±8.096	16.592 ±9.497	14.82±6.342	>0.05
T3	1.735±0.696	1.558±0.705	2.546±3.456	>0.05
T4	86.05±28.16	88.52±36.63	81.076±30.45	>0.05
TSH	1.641±0.747	1.790±0.749	2.469±1.231	>0.05

The Ferriman Gallwey score ranged from 1 to75 with a mean±SD of 10.53±7.19,

In this study most of patients felt to have mild scores. According to Ferriman and Gallwey score hirsutism was mild in 82(78.02%), moderate in 16(15.2%), and severe in 7(6.66 %) of the patients as shown in (table 3)

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The involved sites include face in 97 (92.4%), lower abdomen in 45(42.5%), midline of chest 41(39%), neck 32(30.5%), circumareolar in 9(8.5%), Leg 11 (10.5%), inguinal region and upper thighs in 9(8.5%), arm and upper abdomen 6 (5.7%). Involvement of the upper back, lower back, buttocks, upper abdomen, and foot was rare.

Table No 3: The distribution of patient according to F-G score

F-G score	Number of patient	%
Mild (8-16)	82	78.09
Moderate (17-24)	16	15.23
Severe (>24)	7	6.6

The most common associated symptoms was menstrual irregularity (23.8%) as a sign of clinical hyperandrogenism followed by androgenic alopecia (21%), acne (17.85%) , and acanthosis nigricans (9.45%).

Assessment of menstrual cycle revealed that most of the patients (71.4%) had their first menstrual cycle at the age of 11-13 years, which is in accordance with the national demographic data.

Menstrual cycle was irregular in 25 (23.8%) patients including oligomenorrhea (12.5%), amenorrhea (8.8%), or polymenorrhea (1.4%).

Twelve (11.42%) patients with both hirsutism and menstrual irregularity had polycyctic ovaries on U/S. This group was categorized as polycyctic ovarian syndrome (clinical or biological hyperandrogenism, polycystic ovaries and chronic unovulation).

Eight (7.6%) patients had hirsutism, regular menses, abnormal U/S and abnormal hormonal levels, those patients were categorized as PCOS according to the criteria.So after collecting the results PCOS is found in 20(19 %) of our patients.

In this study the hormonal level shows increase LH hormone in(19%) of patients ,followed by increase in serum total testosterone in (14.3%), After evaluating the LH/FSH ratio we found that it increase in 21(20%)of patients but other hormones have no significant increase. as shown in table No 4.

Thirteen (12.3%) of the patients with both hirsutism and menstrual irregularity had normal U/S but none fulfilled the criteria for PCOS thus categorized as Hirsutism of undetermined origin.

Table No 4: The distribution of patients according to Hormonal level findings.

hormone	No. of patient with normal level	%	No. of patient with increase level	%
Total testosterone	90	85.7	15	14.3
Luteinizing hormone LH	85	81	20	19
Follicle-stimulating hormone FSH	101	96.2	4	3.8
LH/FSH RATIO	84	80	21	20
Serum Prolactin PR	97	92.38	8	6.6

Total testosterone: N= 0.1-0.9ng/ml**,** FSH:N= 93.9-12 mlu/ml
LH:N= 1.5-8 mlu/ml ,Prolactin : N=1.7-25 ng/ml ,LH/FSH : N=1/1

So after Collecting the Results:
Thirty seven (35.23%) patients who have normal hormonal level, regular menses and normal U/S this is considered idiopathic as a cause of Hirsutism.

(Idiopathic hirsutism, defined as hirsutism in association with regular menses and normal hormonal levels).

Thirty five (33.33%) patients with regular menses ,normal U/S and normal hormonal level ,assotiated with positive family history ,this group considered

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familial Hirsutism PCOS is found in 20(19 %) of our patients.

Thirteen (12.3%) of the patients with both hirsutism and menstrual irregularity had normal U/S but none fulfilled the criteria for PCOS thus categorized as Hirsutism of undetermined origin.

All these findings are shown in (table 6)

Table No 5: The end results of the study

Diagnosis	No.	%
Idiopathic Hirsutism	37	35.23
Familial Hirsutism	35	33.33
PCOS	20	19
Undetermined origin	13	12.3

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. It should be differentiated from hypertrichosis, which is defined as the excessive growth of hair independent of androgens and is usually of vellus type (de Berker, D.A.R. *et al.,* 1990).

The intensity of hirsutism in particular areas of the body varies in different patients and depends on the rate of androgen excess, or increased sensitivity of hair follicles to normal levels of androgens in the serum. The most frequently involved sites are face, periumbilical and lower abdomen, areola, and chest (Sharma, N. L. *et al.*, 2008).

The largest group 60 (57%) of patients with hirsutism was in the age group 20-30 year, this indicates that this age group is more vulnerable for Hirsutism, which is also found in Indian study (de berker, D.A.R. 2004).

This is explained by the fact that hirsutism is more in younger age group age for two possible reasons: either younger age group are more sensitive for this unpleasant condition which reflect false rate in community or there wariness of serious disease.

Excessive production of androgens from adrenals or ovaries accounts for 60–80% cases of hirsutism in different studies (Morán, C. *et al.,* 2004). In this study, hyperandrogenemia was found in 14.3% of the patients so further investigations is needed other than US to reach the causes of increase secretion of androgen like CT scan &MRI ,24 hour urine collection test for cortisol ,dexamethasone suppression test .

One of the contributing factors to hyperandrogenemia is increased adiposity, in this study 54 patients (51.42%) had a BMI 25-29.9 Kg/m2 thus were considered overweight, and 22(20.9%) with BMI 30-39.9 Kg/m2 considered obese so we have obesity (BMI ≥ 25 kg/m2) in 76 (72.38%) of our patients. The prevalence among hirsute Mexicans was lower (18%), which might be explained by the higher prevalence of overwieght in Iraq for the genetic susceptibility or environmental factors as diet.

According to Ferriman and Gallwey score Hirsutism was mild in 82(78.02%), moderate in 16(15.2%), and severe in 7(6.66 %) of the patients, in this study most of patients felt to have mild scores, this could be explained by the fact that patients are complaining from Hirsutism early in the course of the problem, or because most of the severe cases of Hirsutism usually consult the gynecological and endocrine department. Twenty tow (21 %) of the patients with both Hirsutism, regular menses and normal hormonal levels had polycyctic ovaries on U/S. These groups were considered within the entity familial polycyctic ovaries. (PCO was defined as the presence of bilaterally normal or enlarged ovaries containing at least 10 microcysts (2-8 mm in diameter) on ultrasonography) (Rotterdam ESHRE/ASRM, 2004).

One of the most common endocrine abnormalities is PCOS with prevalence in the world of 5-10% (Gatee, O. B. *et al.,* 1996), One study from United Arab Emirates reported the presence of PCOS in 91% of 102 patients (Zargar, A. H. *et al.,* 2002), in similar study found an incidence of 37.3% of 150 patients in India. In this study we found 20 (19%) of patients fulfill the criteria of PCOS, within which 7.6% of patients have normal menstrual cycle which does not rule out the diagnosis of PCOS (Rotterdam ESHRE/ASRM, 2004).

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Idiopathic hirsutism was the final diagnosis made in 35.23% of our patients. The diagnosis of idiopathic hirsutism should be applied only to hirsute patients with normal ovulatory function and circulating androgen levels. Some authors question the existence of "idiopathic hirsutism" and believe that all patients with hirsutism will show some metabolic or endocrine disturbances if exhaustive investigations are performed.One possible explanation could be that women with idiopathic hirsutism have subclinical hormonal imbalance which are not responsible to cause menstrual irregularities or other signs of hyperandrogenism.

Therefore, patients with the so-called "idiopathic hirsutism" may experience several metabolic disturbances and need careful evaluation and long term follow-up.

A positive family history of hirsutism was found in 35 (33.3 %) of this study and this was the second group . This indicates familial bases of some of the underlying disease or genetic role.

CONCLUSION

This study shows that Hirsutism is a common cosmetic problem in Iraqis women and most patients were considered to be idiopathic followed by familial cases.

RECOMMENDATION

A large proportion of hirsute females need to be studied and followed up for long time to find other causes and show the long term response of various treatment modalities.

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Appendix 1

Definition of Hair Gradings at Each of 19 Sites (Ferriman-Gallwey Scale)*

Site Grade Definition

1 .Upper lip : 1 A few hairs at outer margin
2 A small mustache at outer margin
3 A mustache extending halfway from outer margin
4 A mustache extending to midline

2. Chin : 1 A few scattered hairs
2 Scattered hairs with small concentrations
3 & 4 Complete cover, light and heavy, respectively

3 .Sideburns: 1 Few nonterminal hairs
2 More nonterminal hairs
3 Terminal hair on side of face
4 Terminal hair extending to mandible

4. Neck: 1 Few hairs on neck
2 More hairs on neck
3 & 4 Complete cover, light and heavy, respectively

5. Chest : 1 Circumareolar hairs
2 With midline hair in addition
3 Fusion of these areas, with three-quarter cover
4 Complete cover

6 .Upper back : 1 A few scattered hairs
2 Rather more, still scattered
3 & 4 Complete cover, light and heavy, respectively

7. Lower back : 1 A sacral tuft of hair
2 With some lateral extension
3 & 4 Three-quarter cover or complete cover, respectively

8 .Buttocks : 1 & 2 Few or many hairs, respectively, over lower buttocks
3 & 4 Hair extending to upper buttocks, light and heavy

9. Upper abdomen: 1 A few midline hairs
2 Rather more, still midline involvement
3 & 4 Half and full cover, respectively

10. Lower abdomen: 1 A few midline hairs
2 A midline streak of hair
3 A midline band of hair
4 An inverted V-shaped growth

11. Inguinal area 1 Pubic hair extending to inguinal area
2 A few hairs below inguinal area
3 & 4 Complete cover below inguinal area, light& heavy,respectively.

12. Perianal area: 1 Hair encircling introitus and anus
2 Hair extending to inner thigh
3 & 4 Hair on inner thigh and buttocks, light and heavy, respectively

13 .Arm: 1 Sparse growth affecting no more than a quarter of the limb surface
2 More than this; cover still incomplete
3 & 4 Complete cover, light and heavy, respectively

14. Forearm 1, 2, 3, 4 As for arm

15. Thigh 1, 2, 3, 4 As for arm

16. Leg 1, 2, 3, 4 As for arm

17. Foot 1 A few hairs on dorsum of foot
2 More hair on dorsum of foot
3 & 4 Hair over one-half or three-quarters or more, respectively, of dorsum

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18. Toes	1 & 2 Few hairs or many hairs, respectively, on big toe 3 & 4 Few hairs or many hairs, respectively, on other toes
19 Fingers	1 Few hairs on proximal phalanx—dorsal surface 2 Many hairs on proximal phalanx—dorsal surface 3 Few hairs on 2nd phalanx—dorsal surface 4 Many hairs on 2nd phalanx—dorsal surface

Source of support: Nil; **Conflict of interest:** Nil.

Cite this article as:
Mohammed, B.M., Abdulatef , W.B. and Khalaf, A.F. "Hirsutism among Women Attending Al-Yarmouk Teaching Hospital, (Clinical and Investigative Study)." *Sarcouncil journal of Medical sciences* 2.01 (2023): pp 1-12.

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