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A STUDY ON THE CLINICAL AND HORMONAL PROFILE OF THE PATIENTS WITH HIRSUTISM

BADANIA NAD KLINICZNYM I HORMONALNYM PROFILEM PACJENTÓW Z HIRSUTYZMEM

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Abstract

Hirsutism is the presence of terminal hairs in a male-like pattern in females, due to elevated male hormone levels. Females with hirsutism are often overweight and have metabolism disturbances as insulin resistance and impaired glucose tolerance. We selected fifty patients of hirsutism from the department of dermatology for the study. A thorough physical examination with specific emphasis on signs of virilization (including frontal baldness, loss of female body contours, increased muscularity, acne, clitoromegaly, and atrophy of breast) was done in all the patients.

Streszczenie

Hirsutyzm to obecność u kobiet męskiego typu owłosienia, ze względu na podwyższony poziom hormonów męskich. Kobiety z hirsutyzmem mają często nadwagę i zaburzenia przemiany materii, takie jak oporność na insulinę i upośledzenie tolerancji glukozy. Wybraliśmy pięćdziesięciu chorych z hirsutyzmem z oddziału dermatologii do badania. U wszystkich pacjentów zostało wykonane dokładne badanie fizykalne ze szczególnym naciskiem na objawy wirylizacji (w tym przednie łysienie, utrata kobiecego konturu ciała, przyrost mięśni, przerost łechtaczki, trądzik i zanik piersi).

Key words: Ferriman-Gallwey scoring; hirsutism; polycystic ovaries; terminal hairs; androgen

Słowa klucze: punktacja Ferriman-Gallwey; hirsutyzm; zespół policystycznych jajników; włosy terminalne; androgen

Introduction

Hirsutism is defined as male-pattern growth of terminal body hair in women in androgen-stimulated locations such as face, chest, and areolae [1]. Hirsutism can be classified broadly into 2 groups viz. androgen induced and non-androgen induced [2,3]. Androgen induced can either be due to excessive endogenous androgen production (ovarian/adrenal) or exogenous due to drugs. Central over production of androgens, increased peripheral conversion of androgens, decreased metabolism and enhanced receptor binding are potential causes of hirsutism [4,5]. Non-androgen induced hirsutism can be idiopathic, familial or drug induced.

Other accompanying signs and symptoms of hyperandrogenism include acanthosis nigricans, obesity, pelvic mass, signs or symptoms of virilization, features of Cushing's syndrome, acne, increased sebaceous activity and alopecia [6]. The modified Ferriman-Gallwey (F-G) score is used to determine the severity of hirsutism by assessing the extent of hair growth in nine key anatomical sites [7]. Simple laboratory measurement of total and free testosterone, dehydroepiandrosterone sulfate, and androstenedione identifies about half of the patients with hyperandrogenism. The rate, pattern and distribution of hair growth at these sites is influenced by various factors including an individual's genetic makeup and hormonal status. A disturbance in the complex interaction between these factors can lead to a male pattern of hair growth in a female [8,9]. Hirsutism may be idiopathic, i.e. secondary to increased responsiveness of the hair follicles to normal circulating levels of androgens, or it may result from an excess of androgens and other hormones. The source of the excess androgens may be either the ovaries, the adrenals or increased peripheral conversion of weak androgenic hormones to more potent ones [10].

Materials and Methods

We selected fifty patients of hirsutism from the department of dermatology for the study. Specific points recorded in the history included: age of onset, duration, rate of progression of the disease, marital status of the patient and the presence of symptoms of virilization (i.e. deepening of voice, thinning of scalp hair, increased muscularity, increased sebum production, acne, decreased breast size, and oligomenorrhoea). A complete physical assessment including height, weight and body mass index was done in

all the patients. Family history of the disease and history of psychiatric illness and its treatment were also specifically sought for. The levels of serum testosterone, sex hormone binding globulins, luteinizing hormone (LH), follicle stimulating hormone (FSH), prolactin, cortisol and abdominal and pelvic ultrasound for adrenals and ovaries were carried out in all the patients. Thyroid function tests, growth hormone level, dehydroepiandrosterone sulfate. A thorough physical examination with specific emphasis on signs of virilization (including frontal baldness, loss of female body contours, increased muscularity, acne, clitoromegaly, and atrophy of breast) was done in all the patients. Ultrasound assessment was done in all the patients to rule out any polycystic ovarian disease.

Aims

1. To study the clinical, biochemical and hormonal profile of the patients with hirsutism.
2. To study the various associated features of patients with hirsutism.

Results (Tabl. I-III)

The data was collected and the results were analyzed statistically.

SR NO	Causes of hirsutism	Number	Percentage
1	Idiopathic hirsutism	25	50
2	Polycystic ovarian disease	20	40
3	Congenital adrenal hyperplasia	1	2
4	Hypothyroidism	4	8

Table I. Various causes of hirsutism

SR NO	Associations	Number	Percentage
1	obesity	10	20
2	acne	19	38
3	striae	9	18
4	acanthosis nigricans	7	14
5	androgenetic alopecia	8	16
6	menstrual irregularities	11	22

Table II. Various associations of hirsutism

SR NO	Ultrasonographic findings	Number	Percentage
1	normal	30	60
2	unilateral cysts	12	24
3	bilateral cysts	6	12
4	bilaterally enlarged ovaries with multiple cysts	2	4
5	adrenal pathology on ultrasound	1	2

Table III. Ultrasonographic findings of the patients with hirsutism

Discussion

The mean age of presentation was 22.4= 2.48 years. Total and free testosterone and 17 hydroxy progesterone was higher in the patients with PCOD (p value < 0.05). Average Ferriman Gallway score was 11.8. Family history of hirsutism was present in 14% patients. The face was the most common site, while the chest and abdomen were the next most common sites. Serum testosterone levels were raised in two patients, one of whom had PCOD and the other had Idiopathic hirsutism. The LH/FSH ratio was elevated (>2) in four patients. All of them had PCOD. Serum Prolactin was increased in eight patients. Of these, two patients had hypothyroidism, three patients had PCOD, and three had idiopathic hirsutism. One patient had elevated 17-hydroxyprogesterone levels and DHEAS. This patient was diagnosed as having congenital adrenal hyperplasia.

Regarding the causes of hirsutism, idiopathic hirsutism was seen in 50% patients, PCOD was seen in 40% patients, hypothyroidism was seen in 8% patients and congenital adrenal hyperplasia was seen in 2% patients. The commonest associated abnormality seen with hirsutism was acne seen in 38% patients, menstrual irregularities were seen in 22% patients, obesity was seen in 20% patients, striae in 18% patients, androgenetic alopecia was seen in 16% patients and acanthosis nigricans was seen in 14% patients. The ultrasonographic findings were normal in 60% patients, PCOD was seen in 40% patients with 24 % patients showing unilateral cysts, 12% patients showing bilateral cysts, bilaterally enlarged ovaries with multiple cysts were seen in 4 % patients and adrenal pathology on ultrasound was seen in 2% patients.

About five percent of women in the reproductive age group in the general population are hirsute, while about 25% of normal young women have

some terminal hair in the face, areola or lower abdomen. The growth of terminal hair in male pattern is determined by the androgens and the intrinsic potential of the hair follicles to respond to the hormonal changes [11,12].

The sensitivity of the hair follicle to androgens is largely governed by the alpha reductase activity in the skin, which is responsible for the conversion of testosterone to dihydrotestosterone. The severity of hirsutism does not correlate well with the level of androgens, because the response of the androgen dependent hair follicle varies considerably within and between individuals. The source of testosterone in a female is the ovaries and the adrenals. Androgen dependent hirsutism may be caused by disorders affecting the adrenals or ovaries, exogenous administration of androgens or a combination of these factors [13,14]. Approximately half the women with mild hirsutism (FG score of 8-15) have idiopathic hirsutism. The most common identifiable cause of hyperandrogenic hirsutism appears to be polycystic ovarian syndrome [15,16]. Insulin resistance with compensatory hyperinsulinemia has been associated with PCOD and is thought to contribute to other features of the metabolic syndrome. Hyperandrogenism has been found to manifest clinically by frontal balding, acne, hirsutism, and clitoromegaly [17].

FG used a scoring system loosely based on that of Garn, evaluating 11 body areas, including the upper lip, chin, chest, upper back, lower back, upper arm, forearm, upper and lower abdomen, thighs and lower legs. A score of 0–4 was assigned to each area examined, based on the visual density of terminal hairs, such that a score of 0 represented the absence of terminal hairs, a score of 1 minimally evident terminal hair growth, and a score of 4 extensive terminal hair growth [18]. Terminal hair hairs can be distinguished clinically from vellus hairs primarily by their length (i.e. >0.5 cm), coarseness, and pigmentation. In contrast, vellus hairs generally measure <0.5 cm in length, are soft and non-pigmented. Biologically active free testosterone is responsible for hair growth and is regulated by sex hormone-binding globulin. The causes of androgenic hirsutism can be exogenous due to drugs (testosterone, dehydroepiandrosterone sulfate, danazol, corticotropin, high-dose corticosteroids, metyrapone, phenothiazine derivatives, anabolic steroids, androgenic progestin, and acetazolamide) or excess endogenous androgen of adrenal or ovarian origin [19,20]. Various causes of ovarian hyperandrogenism are PCOD and virilizing ovarian neoplasia (Luteoma of pregnancy, arrhenoblastomas, leydig cell tumors, hilar cell tumors, thecal cell tumors, etc.). However, PCOD alone accounts for 75-80% cases of hyperandrogenism [21]. Clinically the most common sign of hyperandrogenism in PCOD is hirsutism. The prevalence of hirsutism in PCOD varies between 17% and 83% [22]. Since gonadotrophins are released in a pulsatile manner their concentration varies over the menstrual cycle and a single measurement of LH and/or FSH may not be a sensitive method for diagnosis. Adrenal hyperandrogenism is uncommon and seen in congenital adrenal hyperplasia, late-onset adrenal hyperplasia, Cushing's syndrome, pituitary adenomas that produce excess corticotropin or prolactin and acromegaly. Other less common causes include anorexia nervosa, hypothyroidism and porphyria. Idiopathic hirsutism, also called simple or peripheral hirsutism, is diagnosable in women who have normal ovulatory function and normal androgen profile. Only 5-15% of hirsute women qualify for this diagnosis by these criteria [23].

Conclusions

Thus, our data shows that PCOS is the commonest cause of hirsutism in our clinical practice and that it is prominent among young obese females, which reflects the worldwide pattern. Our findings call for an early intervention strategy to prevent or reduce metabolic syndrome in this subgroup of the population. Further prospective studies on a larger scale are needed, however, to verify our findings.

REFERENCES

1. Erkkola R, Ruutianen K: Hirsutism: definition and etiology. Ann Med 1990; 22: 99-103.
2. Hatch R, Rosenfield RL, Kim MH, Tredway D: Hirsutism: implications, etiology and management. Am J Obstet Gynecol 1981; 140: 815-830.
3. Brooksbank BW: Endocrine aspects of Hirsutism. Physiol Rev 1961; 41: 623-676.
4. Conway GS, Honour JW, Jacobs HS: Heterogeneity of the polycystic ovary syndrome: Clinical, endocrine and ultrasound features in 556 patients. Clin Endocrinol (Oxf) 1989; 30: 459-70.
5. Mckenna TJ: Hirsutism and polycystic ovary syndrome. In: Grossman A, editor. Clinical Endocrinology. London: Blackwell Scientific; 1992. p. 691-712.
6. Moran C, Tapia MC, Hernandez E, Vazquez G, Garcia-Hernandez E, Bermudez JA: Etiological review of hirsutism in 250 patients. Arch Med Res 1994; 25: 311-314.
7. Ferriman D, Gallwey JD: Clinical assessment of body hair growth in women. J Clin Endocrinol Metab 1961; 21: 1440-1447.
8. Barth JH, Catalan J, Cherry CA, Day A: Psychological morbidity in women referred for treatment of hirsutism. J Psychosom Res 1993; 37: 615-619.
9. The Rotterdam ESHRE /ASRM -sponsored PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long term health risks related to polycystic ovary syndrome. Feril Steril 2004; 81: 19-25.
10. Gatee OB, Al-Attia HM, Salama IA: Hirsutism in the United Arab Emirates: a hospital study. Postgrad Med J 1996; 72: 168-71.
11. Carmina E, Rosato F, Janni A, Risso M, Longo RA: Relative prevalence of different androgen excess disorders in 950 women referred because of clinical hyperandrogenism. J Clin Endocrinol Metab 2006; 91: 2-6.
12. Azziz R, Sanchez LA, Knochenhauer ES, Moran C, Lazenby J, Stephens KC, et al: Androgen excess in women: Experience with over 1000 consecutive patients. J Clin Endocrinol Metab 2004; 89: 453-462.
13. Ehrmann DA: Polycystic ovary syndrome. N Engl J Med 2005; 32: 773-777.
14. Azziz R, Carmina E, Sawaya ME: Idiopathic hirsutism. Endocr Rev 2000; 21: 347-362.
15. Azziz R, Woods KS, Reyna R, Key TJ, Knochenhhauer ES, Yildiz BO: The prevalence and features of the polycystic ovary syndrome in an unselected population J Clin Endocrinol Metab 2004; 89: 2745-2749.
16. Balen AH, Coway GS, Kaltsas G, Techatrasak K, Manning PJ, West C, et al:. Polycystic ovary syndrome: the spectrum of the disorder in 1741 patients. Hum Reprod 1995; 10: 2107-2111.

17. Barbieri RL, Ryan KJ: Hyperandrogenism, insulin resistance, and acanthosis nigricans syndrome: a common endocrinopathy with distinct pathophysiologic features. Am J Obstet Gynecol 1983; 147: 90-101.
18. Ferriman D, Gallwey JD: Clinical assessment of body hair growth in women. J Clin Endocrinol Metab 1961; 21: 1440-1447.
19. Carmina E: Prevalence of idiopathic hirsutism. Eur J Endocrinol 1998; 139: 421-423.
20. Taponen S, Martikainen H, Jarvelin M, Laitinen J, Pouta A, Hartikainen A, et al: Hormonal profile of women with self-reported symptoms of oligomenorrhea and/or hirsutism: Northern Finland Birth Cohort 1966 Study. J Clin Endocrinol Metab 2003; 88: 141-147.
21. Balen AH, Laven JS, Tan SL, Dewally D: Ultrasound assessment of the polycystic ovary: International consensus definition. Hum Reprod Update 2003; 9: 505-514.
22. Rotterdam ESHRE/ASRM sponsored PCOS consensus workshop group 2004 revised 2003 consensus on diagnostic criteria and long term health risk related to polycystic ovary syndrome. Hum Reprod 2004; 19: 41-47.
23. Jahanfar S, Eden JA: Idiopathic hirsutism or polycystic ovary syndrome? Aust NZ J Obstet Gynnecol 1993; 33: 414-416.