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Hirsutism

D. Nikolaou[a,*], C. Gilling-Smith[b]

[a]*Department of Obstetrics, Gynaecology, Chelsea and Westminster Hospital, 369 Fulham Road, London SW10 9NH, UK*
[b]*Assisted Conception Unit, Chelsea and Westminster Hospital, 369 Fulham Road, London SW10 9NH, UK*

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Summary Hirsutism is defined as the excessive growth of terminal hair on the face and body of a female in a typical male pattern distribution. Untreated, it can be associated with considerable loss of self-esteem and psychological morbidity. Hyperandrogenaemia is the key trigger for excess hair growth, but the expression and severity are modified by genetic factors. Polycystic ovary syndrome, resulting in excess ovarian androgen production, is the most common cause of hirsutism. A raised serum testosterone level of greater than 5 nmol/l should prompt further investigations to exclude adrenal pathology or an underlying androgen-secreting tumour. The treatment of hirsutism is most effective using combination therapy, including androgen suppression, peripheral androgen blockade and mechanical/cosmetic amelioration and destruction of the unwanted hairs.

Introduction

Hair follicles are found on the whole body, except the palms of the hands, soles of the feet, lips and mucosal surfaces of the external genitalia. Prior to puberty, most of the body is covered with vellus hair, which is fine and downy except on the scalp and eyebrows, these being covered with terminal hair, which is thick and pigmented. During puberty, androgens stimulate the conversion of vellus hair to sexual hair in sex-specific areas. Sexual hair develops in males on the face (beard area), chest, lower back and anterior thighs, and in both sexes in the genital area and lower abdomen.

Hirsutism is caused by increased androgen production and/or bioavailability in a woman, leading to the appearance of sexual hair in areas normally associated with a male appearance. The development of coarse dark hair on the upper lip and chin is particularly distressing and a common reason for women to seek medical advice. If the excess hair growth occurs in areas typical of men, the hair growth is androgen induced, even if serum androgen levels are normal. Conversely, a diffuse increase in long fine hairs, including areas such as the forehead and flanks, is not androgen dependent.

Hirsutism is usually of benign aetiology and must be differentiated from virilisation, in which, in

*Corresponding author. Tel.: +44 208 746 8922; fax: +44 208 746 8921.
E-mail address: dnikolaou@talk21.com (D. Nikolaou).

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addition to excess hair growth, symptoms include increased muscle mass and libido, breast atrophy, clitoromegaly and deepening of the voice. Virilisation is usually more sinister and associated with a sudden rise in circulating androgens, for example a rapidly growing androgen-secreting tumour or exposure to androgenic drugs.

Pathophysiology of abnormal hair growth

The key modulators of sexual hair growth are androgens, promoting growth, increased thickness and pigmentation of the facial, genital and axillary hair. Paradoxically, on the scalp, they have the opposite effect in genetically predisposed individuals, producing regression of scalp hair to vellus hair (balding). Oestrogens reduce the rate of hair growth, resulting in thinner, less pigmented hair. Progestogens have a variable effect, depending on their androgenic potency. Racial and genetic factors affect the expression and perception of abnormal hair growth in women.

5α-Dihydrotestosterone is the most potent androgen in terms of abnormal hair growth. It is derived from the conversion of circulating testosterone and androstenedione within the hair follicle, a reaction catalysed by the enzyme 5α-reductase. In normal circumstances, the ovary and adrenal contribute equally to circulating testosterone and androstenedione levels. Luteinizing hormone (LH) and insulin stimulate ovarian, while adrenocorticotrophic hormone (ACTH) stimulates adrenal androgen, secretion. The biological effect of androgens depends on the level of free hormone in the circulation. Both testosterone and dihydrotestosterone are bound to albumin and sex hormone-binding globulin (SHBG). SHBG production falls as circulating insulin levels and body mass index rise, producing an increase in biologically active androgen. This explains the impact of obesity, insulin resistance and hyperinsulinaemia on the development and severity of hirsutism. Oestrogens have the opposite effect, i.e. they increase SHBG production and reduce free testosterone levels, which is why oral contraceptives are an effective treatment for mild hirsutism. Table 1 summarises the key factors that can lead to the development of hirsutism.

Table 1 Principal factors that can contribute to hirsutism.

- Increased ovarian androgen production
- Increased adrenal androgen production
- Reduced sex hormone-binding globulin levels—increased free androgen
- Increased body mass index
- Hyperinsulinaemia
- Increased 5α-reductase activity in the hair follicle
- Racial and genetic predisposition

Causes of hirsutism

The causes of hirsutism are summarised in Table 2.

Table 2 Causes of hirsutism.

- Polycystic ovary syndrome
- Idiopathic
- Late-onset congenital adrenal hyperplasia
- Cushing's syndrome
  - Cushing's disease (ACTH-secreting pituitary tumour)
  - Ectopic ACTH secretion by non-pituitary tumour (bronchus Table, thyroid)
  - Autonomous cortisol secretion by adrenal or ovarian tumour
  - Ectopic corticotrophin secretion by tumour (very rare)
- Androgen-secreting tumours of the ovary
  - Sex-cord stromal cell tumours
  - Adrenal-like tumours of the ovary
- Androgen-secreting tumours of the adrenal
  - Adenomas
  - Adenocarcinomas
- Iatrogenic
  - Testosterone
  - Danazol
  - Glucocorticoids

ACTH, adrenocorticotrophic hormone.

Polycystic ovary syndrome

This is by far the most common cause of hirsutism. Excess body and facial hair may occur alone or be accompanied by cycle irregularity, acne and/or male-pattern alopecia. Obesity and hyperinsulinaemia are common and exacerbate the clinical symptoms. Diagnosis rests on the finding of polycystic ovaries on pelvic ultrasound, which are