# An update on the assessment and management of hirsutism

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## Abstract

Hirsutism is the abnormal excessive growth of coarse terminal hair over androgen-sensitive body areas. It is a very common endocrine pathology, affecting up to 10% of young females and has been linked with multiple conditions. Polycystic ovary syndrome (PCOS) and Idiopathic hirsutism encompass 90% of the cases. Patients presenting with hirsutism may require additional investigations and a plan with options for treatment. Mechanical hair removal is first-line management, along with lifestyle changes. Laser and phototherapy have been gaining popularity. The combined contraceptive pill is the preferred initial medical treatment offered, if not contraindicated, followed by anti-androgen therapy. Other emerging therapeutic options are inositol and vitamin D, especially in patients with PCOS. Treatment options should be discussed with patients, irrespective of the clinical severity, as they could be addressing underlying psychosocial concerns.

**Keywords** Abnormal hair growth; hirsutism; hyperandrogenism; idiopathic; polycystic ovary syndrome

## Introduction

Hair is a fundamental component of the integumentary system. It has multiple functions controlling thermoregulation, offering barrier protection, participating in pheromone, sebum or sweat production, as well as influencing social interactions. Hair follicles can be divided into two categories: vellus and terminal hair. Vellus hair is fine, thin, lightly pigmented covering most of the body. Terminal hair is coarse, thick, evidently pigmented covering the scalp, forming the eyebrows and eyelashes, and after puberty developing on the underarms and pubic area.

Generalised excess hair growth on the body, affecting both females and males, is defined as hypertrichosis. Hirsutism is characterized by excessive, coarse, terminal hair over androgen-sensitive parts such as the chin, upper lip, chest, upper arms, lower back and thighs affecting females. It is a common endocrine pathology affecting women of reproductive age. Transfeminine patients may also present with hirsutism. The commonest presenting complaints are aesthetic concerns and irregular menses; however, the psychosocial burden of hirsutism should not be disregarded, and alternate pathologies of androgen excess should not be neglected.

Hirsutism is estimated to affect approximately 5–10% of the population with a greater prevalence amongst Afro-Caribbean, Hispanic, Mediterranean, Middle Eastern, and South Asian populations. Patients with hirsutism may attend a specialist like a dermatologist, endocrinologist or gynaecologist for advice. A holistic approach should be undertaken to identify pathologies as well as address other psychological concerns. This review covers the latest updates on the assessment and management of hirsutism.

## Pathophysiology

Hirsutism is considered to be a result either of increased testosterone production or increased follicular androgen sensitivity. Androgenic steroids, testosterone and dihydrotestosterone (DHT), are responsible for the transformation of vellus hair to terminal hair on the face, chest, lower abdomen, lower back, upper arms and thighs. Increased androgen levels during puberty transform vellus hair to terminal follicles of larger size and diameter, which have a longer lasting active growing phase. Over-terminalisation of hair defines hirsutism.

The ovaries and adrenal glands are estimated to produce 80% of total body's testosterone. Pregnenolone and progesterone are converted by 17α-hydroxylase to 17-hydroxypregnenolone and 17-hydroxyprogesterone (17-OHP), and are then converted into dehydroepiandrosterone (DHEA) and androstenedione respectively. DHEA is converted to androstenedione and then to testosterone (Figure 1). The estimated remaining 20% of circulating testosterone is derived from peripheral DHEA or androstenedione that originates from liver, skin, hair or adipocytes.

In a physiological state, only up to 2% of testosterone is free, unbound and functioning as an active androgen. The great majority of testosterone, the rest 98%, is biologically inactive and bound to different proteins; mainly to steroid hormone binding globulin (SHBG), followed by cortisol binding globulin or albumin. Free testosterone is released to peripheral tissues, reaching the follicles, and further being converted to DHT by 5α-reductase, which is a more potent biometabolite. Systemically raised levels of androgenic steroids ultimately will result in raised DHT levels at the hair follicles, inducing hirsutism.

The rate of appearance of terminal hair provides crucial clinical information. Gradual and progressive growth indicates a functional aetiology, whereas rapid, new onset of terminal hair may imply a neoplastic source of androgen secreting tumour of ovarian or adrenal origin. Temporal and male pattern balding can be associated with progressive hyperandrogenism along with irregular cycles. Virilisation may occur due to prolonged hyperandrogenism, where besides hirsutism, patients may experience signs of masculinisation such as deepening of the voice, increased muscle mass, increased libido or enlargement of the clitoris.

Obesity, hyperprolactinaemia, acromegaly and thyroid pathologies have also been associated with lower SHBG levels, increasing the bioavailability of free testosterone. Therefore,

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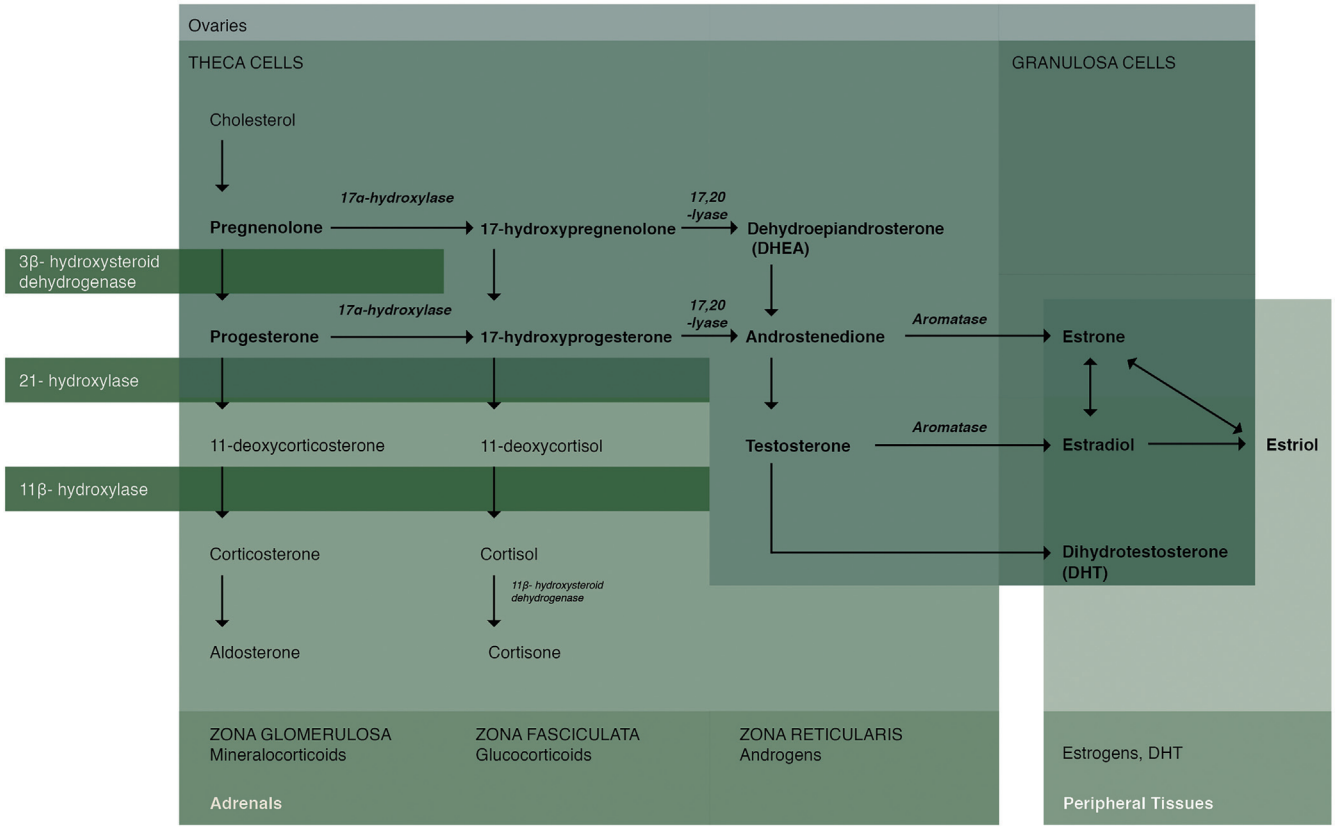


**Figure 1** Androgen synthesis in the ovaries and adrenal glands.

unexplained hirsutism ought to be investigated for other endocrine underlying possible pathologies.

## Aetiology

Hyperandrogenism in hirsutism could have a variety of aetiologies. It could be due to ovarian or adrenal pathologies, androgen secreting tumours, or idiopathic. The main ovarian cause for hyperandrogenism is polycystic ovary syndrome (PCOS) and the main adrenal pathologies are Cushing's' syndrome and congenital adrenal hyperplasia (CAH). Furthermore, it may relate to obesity and insulin resistance, or other hormonal imbalances or sensitivities (Table 1).

### Polycystic ovary syndrome (PCOS)

PCOS represents almost 80% of all hirsutism cases. The presence of hirsutism alone should be considered predictive of biochemical hyperandrogenism and PCOS in adults. Hyperandrogenism in PCOS is driven by altered thecal steroidogenesis. Elevated androstenedione augments oestrogen and ovarian androgen derivatives which negatively feed back to the pituitary resulting into reduced FSH and higher LH secretions. This contributes further to ovarian stimulation with hyperplastic theca cells and stroma, and a thick cortex, which over time culminate in an inflamed microenvironment with impaired ovarian function. From another point of this causal-nexus, high testosterone levels, increase insulin secretion, which further stimulates the theca cells to secrete greater oestrogen and testosterone levels. Consequently, SHBG levels decrease, inducing the clinical symptoms of hyperandrogenism, while hyperinsulinaemia evolves to insulin resistance, overall growth, weight gain and further ovarian overstimulation.

### Cushing syndrome

Primary hypercortisolism or Cushing syndrome refers to raised cortisol levels due to pituitary overproduction of adrenocorticotropic hormone (ACTH). Raised ACTH levels have a secondary stimulant effect on the zona reticularis of the adrenals, resulting in excessive androgen production that clinically presents with hirsutism.

### Congenital adrenal hyperplasia (CAH)

CAH encompasses the spectrum of defective adrenal steroidogenesis due to autosomal recessive enzyme dysfunction leading to hypocortisolism. There are three specific enzyme deficiencies associated with virilization and hirsutism in females. Deficiency of 21-hydroxylase is the commonest form of CAH, and impairs aldosterone production. Therefore, patients have low blood pressure because of low sodium and high potassium levels (salt wasting). In response, renin levels rise, and following the diverted metabolic pathways, sex hormones increase, along with 17-OHP, which is the pathognomonic marker often investigated. Deficiency of 11β-hydroxylase and 3β-hydroxysteroid dehydrogenase have also been reported to be associated with hirsutism in females but are less common (Figure 1).

## Summary of differential diagnoses

| Differentials summary | Diagnostic criteria | Associated features |
|---|---|---|
| Polycystic ovary syndrome | Rotterdam criteria<br>↑Testosterone, LH<br>↓FSH | Menstrual irregularities<br>Insulin resistance<br>Acne |
| Cushing syndrome | 24-h urine free cortisol<br>Low-dose dexamethasone suppression test | Central obesity<br>Moon face<br>Buffalo hump<br>Purple striae<br>Subfertility |
| Congenital adrenal hyperplasia (CAH) | ↑17 OHP<br>↓Cortisol and aldosterone | Early puberty<br>Family history<br>Menstrual irregularities<br>Subfertility<br>Ambiguous genitalia at birth in females |
| Obesity and insulin resistance | ↑Insulin/insulin resistance<br>BMI > 30 | Acanthosis nigricans<br>Central obesity |
| Androgen-secreting tumours | ↑Testosterone (>6.94 nmol/L)<br>↑DHEAS, Androstenedione<br>↑Testosterone: epitestosterone ratio | Rapid new onset of terminalisation<br>Virilization<br>Abdominal or pelvic mass<br>Increased libido<br>Clitoromegaly |
| Idiopathic hirsutism<br>Transgender feminine patients | Normal androgen levels | Regular menstrual cycle<br>Gender dysphoria<br>Body image dissatisfaction<br>Mental health issues |
| Hyperprolactinemia | ↑Prolactin levels | Galactorrhoea<br>Amenorrhoea<br>Subfertility |
| Thyroid dysfunction | Abnormal TFTs | Fatigue<br>Subfertility<br>Weight and skin changes<br>Menstrual irregularities |
| Medications | Reviewing patient medication history | Androgens, oestrogen antagonists, glucocorticosteroids, progestins, cyclosporine, danazole, phenytoin, D-penicillamine, interferons |

**Table 1**

### Androgen-secreting tumours

Androgen-producing tumours may arise in the ovaries or adrenals. Rapid, new onset of terminal hair growth may imply a neoplastic source of androgen producing tumours or ovarian hyperthecosis, especially for postmenopausal patients. Further investigations are required, even if their incidence is rare.

### High BMI and insulin resistance

Adipose tissue produces a multitude of peptide and steroid hormones, as well as enzymes involved in steroid metabolism. Adipose tissue can contribute up to 50% of the circulating testosterone in overweight premenopausal women, often resulting in hyperandrogenism and hirsutism. Additionally, hyperinsulinaemia further induces LH secretion which stimulates thecal androgen and oestrogen production, with insulin functioning as a co-gonadotropin. Raised insulin levels suppress hepatic SHBG production, augmenting the free serum testosterone levels. Obesity and hirsutism share a close link.

### Idiopathic

Idiopathic hirsutism is associated with normal androgen levels. It is speculated to be due to exaggerated peripheral 5α-reductase activity, androgen receptor polymorphism or altered androgen metabolism. It is currently a diagnosis of exclusion; new markers are being evaluated but are yet to be established.

### Transgender feminine patients

Hirsutism and facial hair in transfeminine patients depict a prominent male secondary sex characteristic, that can often result to body image dissatisfaction, gender dysphoria and mental health problems. Health care professionals need to be mindful of these, as it can cause extensive psychological implications.

Gender-affirming surgical and hormonal treatments do not interfere with hair growth. Complete hair removal is considered medically necessary before genital gender affirming surgery, as active follicles on inverted skin flaps can lead to postoperative intra-vaginal or intra-urethral hair growth increasing the risk of infection and discomfort for some patients. Transgender patients face greater barriers to care; therefore, it is imperative for clinicians to review patients holistically and address all underlying issues with every opportunity.

### Other

Hyperprolactinaemia increases adrenal DHEA-S production, which is suspected to consequently lead to hirsutism. Acromegaly, hyperthyroidism or hypothyroidism have also been reported as rare causes of isolated hirsutism. Lastly medications' side effects can result in hirsutism.

## Assessment

A comprehensive history and physical examination should be undertaken when assessing symptoms and signs of hirsutism and clinical hyperandrogenism (Table 1). Healthcare professionals should be aware of the potential negative psychosocial impact of hirsutism and empathise without prejudice over the unwanted hair overgrowth, regardless of the apparent clinical severity.

### History taking

Important points to be addressed during history:

- Age of onset of symptoms: Idiopathic hirsutism or hirsutism caused by PCOS starts during puberty and worsens over time. If the onset of hirsutism is over the age of 40, this may indicate ovarian hyperthecosis or malignancy.
- Onset of symptoms: A more rapid onset of symptoms may indicate Cushing's syndrome or an androgen-secreting tumour.
- Presence of virilisation: Virilisation may be associated with an androgen-secreting tumour. Signs include male pattern alopecia, deepening of the voice, breast atrophy, increased muscle bulk and clitoromegaly.
- Family history: Hirsutism in other family members suggests a hereditary or idiopathic cause.
- Drug history: Screening and discontinuing medication associated with hirsutism or hypertrichosis may lead to resolution of symptoms. Phenytoin, streptomycin, diazoxide, minoxidil, acetazolamide and latanoprost are also associated with hypertrichosis.
- Menstrual cycle, Reproductive history: Irregular anovulatory cycles and subfertility occur with PCOS, Cushing syndrome, CAH, thyroid abnormalities and hyperprolactinaemia.
- Other signs and symptoms: Weight changes may occur in thyroid disorders. Galactorrhoea occurs in hyperprolactinaemia. Patients with hirsutism should have a height, weight, BMI and blood pressure measured.
- Mental health: Hirsutism can be linked to anxiety and depression, eating disorders, body image concerns with deeper psychosocial and psychosexual implications. It is important to recognise this impact in order to understand and address patients' concerns. Physicians involved in the care of transgender patients should be particularly aware as treatment may have a gender-affirming effect.

### Physical examination

The focus of the physical examination of a patient presenting with excessive growth hair is to assess the type of hair (terminal vs vellus) and the distribution to distinguish hirsutism from hypertrichosis. Examination should evaluate terminal hair of varying shape and texture that measure more than 5 mm. Standardised visual scales are recommended when assessing hirsutism in combination with a photographic atlas. It is important to be reminded that the severity of hirsutism may appear to vary by ethnicity, and that self-treatment, which often precedes the referral to the specialist, can limit clinical assessment.

The modified Ferriman Gallwey score (m FG) is a common visual scale. It assesses terminalization of hair in the nine androgen-sensitive areas: lip, chin, chest, back, upper arms, lower abdomen, and upper thighs. The nine areas are graded between 0 (no terminal hair) to 4 (excess terminal hair), with hirsutism defined by a score of 8 or more. Scores classify hirsutism as mild (8−15), moderate or severe (>15) and monitor progress or response to treatment.

New onset, severe, or worsening hirsutism, requires further investigation to rule out androgen-secreting tumours and ovarian hyperthecosis across all ages. Complete physical examination should include palpation of the abdomen with bimanual examination, if indicated, for initial identification of ovarian masses. A methodical, systemic review may reveal other signs of hyperandrogenaemia or virilisation.

### Investigations

Patients with mild hirsutism and regular menses do not require testing for hyperandogenism before treatment. Further investigations however are recommended for patients with moderate or severe hirsutism or patients with suspected PCOS and a mFG score of 4−6. Blood tests and biomarkers should be performed in the early follicular phase. Assessment of the hormonal profile includes most of the hormones secreted by the anterior pituitary (FSH, LH, TSH, PRL), as well as other relevant hormones and markers of hyperandrogenism released by the ovaries and the adrenals (Table 2).

**Bloods and biomarkers:**

- FSH and LH: Examining the function of the Hypothalamus-Pituitary-Ovarian axis with FSH and LH levels can provide valuable information on the hormonal feedback.
- Testosterone: Free testosterone levels are considered the "gold standard" to assess hyperandrogenism. If levels are above 6.94 nmol/L or 200 ng/dl an urgent referral to endocrinology is required.
- 17 Hydroxyprogesterone: A unique marker for CAH because of 21-hydroxylase deficiency
- DHEA: DHEA and androstenedione are secreted by the ovaries and the adrenals, with values fluctuating throughout the menstrual cycle. However, Dehydroepiandrosterone-sulphate (DHEA-S) is secreted only from the zona reticularis of the adrenals, formulating an objective marker of adrenal pathology. DHEA-S levels can rise because of different conditions, like PCOS, or because of the influence of prolactin or insulin-like growth factor, being linked to other endocrine pathologies. Levels >700 μg/dl indicate an adrenal cause and require further testing to exclude malignancy.

## Summary of investigations

| Laboratory investigations | Indication |
|---|---|
| FSH and LH | Assessment of the hypothalamus-pituitary-ovarian axis |
| Testosterone | Hyperandrogenism assessment<br>If T is normal, consider idiopathic hirsutism<br>If T >6.94 nmol/L, urgent specialist referral |
| DHEA | Hyperandrogenism assessment<br>↑DHEA-S indicative of adrenal origin |
| SHBG | Sever hyperandrogenism, monitoring treatment |
| 17-OHP | Suspected CAH |
| 24-h free urine cortisol | Cushing syndrome |
| PRL | Hyperprolactinaemia |
| TSH | Thyroid dysfunction |
| Ca-125, AFP, HCG, LDH | Suspicious ovarian mass |
| **Imaging investigations** | **Indication** |
| US Scan | Pelvis USS for PCOS/ovarian mass<br>Adrenal gland USS if suspected tumour |
| MRI brain/CT head | Pituitary adenoma |

**Table 2**

- SHBG: SHBG levels can be particularly useful in assessing severity or hyperandrogenism and monitor response to treatment

Additional blood test can be requested depending on clinical concerns. For instance, 24 h urine cortisol and a short dexamethasone suppression test can be requested for Cushing Syndrome, Prolactin and TSH for oligomenorrhea, or tumour markers (Ca-125, AFP, HCG, LDH) for suspicious ovarian masses. Idiopathic hirsutism is characterized by normal androgen levels and is considered to be a diagnosis of exclusion.

## Management

Management options depend on the severity of symptoms, the psychological morbidity perceived by the patient and timing with regards to family planning. Lifestyle changes on diet, exercise and weight loss should be considered primary interventions, as applicable to individual needs. Medical professionals need to be aware and acknowledge the psychological burden of hirsutism. Therefore, options for treatment should be provided without prejudice despite the clinical picture (Table 3).

### Mechanical treatment, laser and light therapies for hair reduction

Mild hirsutism can be treated with epilation methods such as threading, waxing, plucking or shaving. More permanent options include electrolysis or professional photolysis, but laser therapy is more effective. They are easily accessible and have been gaining popularity. Can offer quick results and provide relief to associated mental health concerns, improving patients' quality of life. A greater number of treatment sessions may be required in women with PCOS or other pathologies in comparison to patients with idiopathic hirsutism. The 2023 International Evidence-Based Guideline for the Assessment and Management of PCOS urges policy makers to consider funding mechanical laser and light therapies for women with PCOS as this treatment provides significant psychological benefits.

### Combined oral contraceptive pills

First line medical treatment for hirsutism is the combined oral contraceptive pill (COCP) for patients that satisfy the general population prescription guidelines. COCPs offer control of the ovarian function by suppressing FSH and LH, increasing SHBG levels, and ultimately reducing androgen production. Some of the considerable benefits are that they can provide regular withdrawal bleeds, inhibit ovulation and protect against future endometrial hyperplasia. Strong evidence supports that regulation of the menstrual cycle can control hyperandrogenism, and is therefore associated with improvement of hirsutism.

Shared decision making is recommended and considered to improve adherence. Guidelines recommend the use of natural Oestrogen preparations, such as ethinyl estradiol (EE), at the lowest effective doses of 20–30 μg. Oestrogenic COCPs are preferred, as they have been proven particularly effective at increasing SHBG levels and thus treating hyperandrogenism. Vaginal rings (15 μg EE) have also been reported effective treating acne and hirsutism, but evidence is limited. Drospirenone/Ethinyl estradiol (DRSP/EE) is the newer generation of COCPs which includes progestin derived from spironolactone instead of testosterone, having antiandrogenic, anti-mineralocorticoid and progestogenic effects against hirsutism.

### Anti-androgen pharmacological agents

Anti-androgen pharmacological agents are not superior to COCPs in hirsutism treatment. However, they can be very effective when COCPs are contraindicated, not well tolerated, or additional treatment is required in refractory cases. The commonest anti-androgens used in the treatment of hirsutism are finasteride, flutamide, spironolactone and bicalutamide. The advice is to consider anti-androgen treatment when COCPs provide an inadequate response after at least 6 months of treatment. These pharmacologic agents function either by impairing the androgen receptors, decreasing androgen production or inhibiting 5α-reductase activity.

**Androgen receptor blockers:** androgen receptor blockers are considered to control hyperandrogenism. The commonest agents used are Spironolactone, Flutamide, Bicalutamide and Cyproterone acetate. Androgen receptors inhibitors are most effective when used in combination with COCPs, synergistically improving hirsutism symptoms and eliminating common anti-androgen side effects. In combination, the risk for teratogenicity or feminisation of male fetus is diminished by the contraceptive action, and equally Spironolactone's dysfunctional uterine bleeding and Cyproterone acetate's bone loss are eliminated by the hormonal effects of COCPs. Nevertheless, the combination of anti-androgens and COCPs demands appropriate hepatic and lipid follow up.

Spironolactone and Finasteride have a dual function; are competitive, dose-sensitive inhibitors, with a parallel inhibition of

**Medical treatment synopsis for hirsutism**

| Medication family | Subtype | Commercial name | Important side effects |
|---|---|---|---|
| Contraceptives | COCP | Oestrogenic pills: Ovysmen, Marvelon, Brevinor<br>COCPs with DSRP/EE: Yasmin, Petibelle | Headache, nausea, breast tenderness, metrorrhagia, acne |
| | Vaginal ring | Nuva Ring | |
| Anti-androgens | Androgen receptor blockers | Spironolactone<br>Flutamide, bicalutamide<br>Cyproterone acetate | Electrolyte abnormalities<br>Severe liver toxicity<br>Thromboembolism<br>Meningioma |
| Enzyme inhibitors | 5a-reductase inhibitors<br>Ornithine decarboxylase | Finasteride<br>Eflornithine | Liver toxicity<br>Skin irritation |
| GnRH analogues | | Leuprolide | Menopausal symptoms<br>Osteoporosis |
| Insulin sensitising agents | | Metformin | Gastrointestinal symptoms<br>Metabolic acidosis |
| Other agents | | Inositol<br>Vitamin D | Gastrointestinal symptoms<br>Abdominal pain |

**Table 3**

the 5α-reductase. Spironolactone is mainly used as a potent diuretic, therefore, electrolytes and blood pressure monitoring is required 3 weeks post initiation, and then annually. Common side effects include polyuria, hypotension, hyperkalaemia, fatigue, headache, dizziness and breast tenderness. Spironolactone at 25 −100 mg/day has lower risk profile but clinical improvement is noted over at least 6 months. Flutamide is a non-steroidal anti-androgen with equal efficacy to Spironolactone. Cyproterone acetate, being a progesterone derivative, competes with testosterone and DHT to bind onto the androgen receptors, inhibiting androgen production and eventually suppressing LH secretion. Having strong anti-androgenic properties, it increases androgen clearance from the liver. Side effects include irregular bleeding patterns, decreased libido, nausea and depression. Cyproterone acetate of more than 10 mg per day is associated with increased risk of meningioma and a four times risk of thromboembolism. Flutamide and Bicalutamide are significantly hepatotoxic and dyslipidaemic; thus, used only in selected cases with resistant hirsutism.

**Enzyme inhibitors:** finasteride is a 5α-reductase inhibitor impeding the conversion of testosterone to DHT. It is particularly effective in idiopathic hirsutism; it improves hyperandrogenism and hirsutism, but increases DHEA-S levels. Finasteride has been found to be less effective than Spironolactone, but has similar efficacy to Cyproterone acetate. It has a low side effect profile, mainly associated with liver toxicity, and can be used in combination with the COCP to avoid the risk of reduced virilisation of male fetuses. Finasteride 2.5 mg daily is proven more effective than taken every three days. Topical facial Eflornithine cream is applied twice per day and irreversibly inhibits ornithine decarboxylase at hair follicles, thus reducing hair growth.

Eflornithine can be used in conjunction with laser or microneedle treatment to be more effective; however, it tends to provide impermanent benefits, occasionally sensitise the skin and cause pruritus or worsen acne or pseudofolliculitis barbae. Improvement is visible within 8 weeks of treatment initiation, but if no change is noted within 4 months of treatment Eflornithine should be reviewed and abandoned.

### Inositol

Inositol is considered to regulate hyperandrogenism and hyperinsulinaemia. Recent studies indicate that inositol may be associated with mild gastrointestinal side effects like nausea, flatulence or diarrhea; however, it appears to provide improvement in metabolic measures, weight loss, hirsutism and increased ovulation rates. More research needs to be conducted to identify the appropriate form, dose or combination of Inositol that is recommended for optimal outcomes.

### Bariatric and metabolic surgery

First line management options for overweight patients are lifestyle changes including exercise, diet and weight loss. Upper body and abdominal obesity are associated with increased testosterone and reduced SHBG levels. Bariatric and metabolic surgery can be considered to improve hirsutism and other comorbidities such as obesity, hypertension, diabetes, irregular menstrual cycles, ovulation and pregnancy rates in women with PCOS at lower BMI thresholds compared to other conditions.

### GnRH analogues

GnRH analogues induce temporary ovarian suppression and can help treat severe hirsutism caused by hyperinsulinaemia or hyperandrogenism. They require time to become effective and are reserved for refractory cases. They induce menopause symptoms and prolonged use is associated with relevant long term side effects; therefore, some added oestrogen is recommended for bone protection.

### Insulin sensitising agents

Insulin sensitizing agents are thought to reduce hyperinsulinaemia, facilitate reduction of ovarian and adrenal

androgen synthesis with respective increase in SHBG and optimise gonadotropin secretion, but more extensive evidence is needed. Medications that have been used are Metformin and Thiazolidinediones. Metformin increases insulin sensitivity and reduces insulin resistance, particularly evident in PCOS patients regardless of their BMI, and patients with hyperinsulinaemia. The latest guidelines report that metformin could be considered, if other options are contraindicated, for management of hirsutism in irregular cycles, but overall anti-androgens are proven to be superior. It should be acknowledged that Metformin has recognised additional anthropometric and metabolic benefits and therefore at present is recommended over Inositol in cases of central adiposity, despite Metformin's significant gastrointestinal side effects. Thiazolidinediones (TZD) improve peripheral and liver insulin action. A few studies have yielded positive outcomes with TZDs over placebo, but the effect of TZDs compared to other treatments has not been studied. TZDs are associated with significant side effects therefore they would not be routinely recommended to the general population. These medications require further research to support efficacy, and therefore should not be used before the recommended options.

### Vitamin D

Vitamin D is a new emerging treatment, proven to be effective in patients with raised BMI and PCOS. High doses supplementation of 50,000 IU per week are noted to improve 25(OH)vitamin D levels and decrease androgen levels, becoming clinically effective in treating hirsutism. Thus, Vitamin D therapy could offer promising results for overweight patients, improving their reproductive health and increasing their fertility potential.

## Summary

Hirsutism is the excessive growth of coarse terminal hair over androgen-sensitive body areas usually affecting patients of reproductive age. The commonest cause of hirsutism is hyperandrogenism because of PCOS; however, many more conditions are linked to this pathology. Hirsutism can be classified into mild, moderate or severe based on clinical assessment; however, this may not necessarily correlate with the psychosocial and psychosexual impact that patients experience. Mild hirsutism does not require any additional investigations; moderate or severe states should be investigated further. Recurrent, persisting, or cases of new rapid onset of symptoms should be reviewed by specialists. Treatment options include mechanical destruction of hair follicles, correction of hyperandrogenaemia or blockade of peripheral androgen receptors. ◆

## FURTHER READING

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### Practice points

- Hirsutism is a result of hyperandrogenaemia, increased follicular androgen receptor sensitivity or because of its idiopathic nature
- Hirsutism in adults can be evidence of hyperandrogenism and PCOS
- Hirsutism assessment utilises the modified Ferriman-Gallwey score and photographic atlases, while clinicians should be mindful of history of previous epilation treatments
- Medical treatment outcomes are best observed at least 6 months after initiation of a new intervention or after combination therapies
- Hirsutism can impose significant psychological implications which should be addressed in a sensitive manner