Hirsutism and Homoeopathy

Research Paper On Hirsutism and Homoeopathy by Dr. Arindam Roy. B.S.C, M.D(HOMO) Medicine

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Definition

Excessive growth of facial or body hair in women is called hirsutism (Psora- Sycosis). It is defined as the presence of excessive terminal hair in androgen-dependent areas of a woman´s body.

It has to be differentiated from hypertrichosis, which is usually familial in nature and associated with an endocrine dysfunction - such as thyroid dysfunction - or with medications such as phenytoin or minoxidil (Psora).

Types of hirsutism

It may bea- **Primary or idiopathic hirsutism-** Idiopathic hirsutism is defined as hirsutism with no other clinical abnormalities, particularly no menstrual irregularity; and using the Rotterdam criteria, no evidence of a multifollicular ovary. It may well be a mild variant of PCOS. It is probably hereditary, because there is usually a family history of the disorder (Psora).

b- **Secondary hirsutism-** It is most often associated with polycystic ovary syndrome. This type of hirsutism may also be caused by:

- malfunctions of the pituitary or adrenal glands (Psora)
- use of male hormones or minoxidil (Loniten), a drug used to widen blood vessels (Causa occassionalis)
- adrenal or ovarian tumors. (Pseudopsora- Sycosis)

Other classification of hirsutism may be as below-

a- **Androgen-independent hirsutism–** entire body is covered with vellous hair evenly distributed over androgen-dependent and androgen-independent regions. It is caused by Congenital diseases –e.g. Cornelia de Lange and Seckel syndromes, drugs –e.g. androgen analogues, anticonvulsants, corticosteroids, cyclosporine, minoxidil, phenytoin and progesterone analogues, metabolic disorders –e.g. anorexia nervosa, porphyria cutanea tarda. (Psora- Sycosis- Syphilis)

b- **Androgen-dependent hirsutism–** Increased terminal hair over androgenic regions of the face and upper chest.(Psora- Sycosis)

Causes of Hirsutism

Hirsutism is a sign of increased androgen action on hair follicles (Psora), from increased circulating levels of androgens (Sycosis) or increased sensitivity of hair follicles to normal levels of circulating androgens (Psora). Any condition that increases the blood level of testosterone, male sex hormone or androgens (Sycosis) or decreases the sex hormone binding globulin (SHBG) levels (Psora- Syphilis) results in hirsutism.

Causes of Hirsutism

1- PITUITARY/ADRENAL
a- ACTH overproduction
▪ Pituitary adenomas
▪ Tumors that may secrete ACTH, the most common are-
□ Small-cell carcinoma of the lung
□ Tumors of the thymus
□ Pancreatic islet-cell carcinoma
b- Congenital adrenal hyperplasia, deficiencies of:
▪ 21-hydroxylase (most common, 90% of cases)
▪ 3 13-hydroxysteroid dehydrogenase
▪ 11-hydroxylase
c- Androgen-producing adrenal tumors
▪ Adrenal adenomas
▪ Adrenal carcinomas
2- OVARIAN
a- Polycystic ovarian syndrome
b- Virilizing ovarian tumors
▪ Granulosa- stromal cell tumors
▪ Sertoli- Leydig cell tumors
c- Insulin resistance
3- COMBINED OVARIAN AND ADRENAL
a- Idiopathic hirsutism (mostly ovarian)
b- Polycystic ovarian syndrome secondary to adrenal hyperandrogenism
4- EXOGENOUS MEDICATIONS
a- Androgens or anabolic steroids
b- Oral contraceptives (uncommon)
c- Medications that cause hyperprolactinemia
▪ Metoclopramide
▪ high-dose phenothiazines
▪ butyrophenones such as haloperidol
▪ thioxanthines such as thiothixene
▪ methyldopa
▪ reserpine
▪ estrogens
▪ opiates

Some of the main conditions causing hirsutism are-

- **Polycystic ovary syndrome (POS or PCOS)** –Also known as Stein-Leventhal syndrome, is an endocrine disorder which is the most common hormonal disorder among women of reproductive age, and is a leading cause of infertility (Psora- Sycosis). The principal features are weight problems, lack of regular ovulation and/or menstrual and excessive amounts or effects of androgenic (masculinizing)

hormones. The symptoms and severity of the syndrome vary greatly between women. While the causes are unknown, insulin resistance, diabetes and obesity are all strongly correlated with PCOS.

- **Congenital adrenal hyperplasia-** It refers to any of several autosomal recessive conditions resulting from biochemical paths of the steroidogenesis of cortisol from cholesterol by the adrenal glands (Psora-Syphilis). Most of these conditions involve greater or lesser production of sex steroids and can alter development of primary or secondary sex characteristics in affected infants, children, and adults.
- **Delayed adrenal hyperplasia-** It is due to deficiency of Classical 3ß-hydroxysteroid dehydrogenase (3ß-HSD) (Psora- Syphilis).
- **Cushing syndrome-** Also known as hypercortisolism, it is a hormonal disorder caused by an abnormally high circulating level of corticosteroid hormones causing hirsutism (Sycosis).
- **Hyperinsulinemia-** Growing evidence implicates high circulating levels of insulin (sycosis) in women to the development of hirsutism. This theory is consistent with the observation that obese (and thus presumably insulin resistant hyperinsulinemic) women are at high risk of becoming hirsute. Further, treatments that lower insulin levels will lead to a reduction in hirsutism. It is speculated that insulin, at high enough concentration, stimulates the ovarian theca cells to produce androgens (sycosis). There may also be an effect of high levels of insulin to activate the insulin-like growth factor-I (IGF-1) receptor in those same cells. Again, the result is increased androgen production (sycosis).
- **Insulin resistance-** It is the condition in which normal amounts of insulin are inadequate (psora) to produce a normal insulin response from fat, muscle and liver cells. Insulin resistance in fat cells results in hydrolysis of stored triglycerides, which elevates free fatty acids in the blood plasma. Insulin resistance in muscle reduces glucose uptake, whereas insulin resistance in liver reduces glucose storage, with both effects serving to elevate blood glucose (Psora- Sycosis). High plasma levels of insulin and glucose due to insulin resistance often lead to metabolic syndrome and type- II diabetes (Sycosis).
- **Hyperprolactinemia-** PRL modulates 5- alpha- RA and peripheral androgen metabolism and is involved in evolution of hirsutism in state of hyperprolactinemia (Psora- Sycosis).
- **Hypothyroidism-** Hypothyroidism can lead to a reduction of sex hormone binding globulin (PsoraSyphilis) and increase in free testosterone (Sycosis). Free testosterone is one of the factors contributing to PCOS symptoms- infertility, polycystic ovaries, hirsutism, male pattern hair loss, and acne.
- **Ovarian tumors-** Androgen-secreting tumors of the ovary (Psora- Syphilis- Sycosis) are usually heralded by virilization (i.e., development of male characteristics in women) and rapid progression of hirsutism and cessation of menses.
- **Drug induced-** Anabolic steroids, Danazol, Metoclopramide, Methyldopa, Phenothiazines, Progestins, Reserpine, Testosterone etc. **Idiopathic**–Most common.

Pathophysiology of Hirsutism

Hair growth cycle

The hair growth cycle is comprised of three phases-

1- Anagen (growth phase)
2- Catagen (involution phase) 3- Telogen (rest phase)

Depending upon the body site, hormonal regulation plays an important role in the hair growth cycle. Androgens increase hair follicle size, hair fiber diameter, and the proportion of time terminal hairs spend in the anagen phase. Androgen excess in women leads to increased hair growth in most androgen sensitive sites, but will manifest with loss of hair in the scalp region, in part by reducing the time scalp hairs spend in anagen phase.

Hair can be categorized as either-

 Vellus (fine, soft, and not pigmented) or
Terminal (long, coarse, and pigmented).

The number of hair follicles does not change over an individual's lifetime, but the follicle size and type of hair can change in response to numerous factors, particularly androgens. Androgens are necessary for terminal hair and sebaceous gland development and mediate differentiation of pilosebaceous units (PSU's) into either a terminal hair follicle or a sebaceous gland. In the former case, androgens transform the vellus hair into a terminal hair; in the latter, the sebaceous component proliferates and the hair remains vellus.

Male-pattern hair growth occurs in sites where relatively high levels of androgen are necessary for pilosebaceous unit differentiation. Although androgen excess underlies most cases of hirsutism, there is only a modest correlation between the quantity of hair growth and androgen levels. This is thought to result from the fact that stimulation of hair growth from the follicle does not depend solely on circulating androgen concentrations, but also depends upon local factors and variability in end-organ sensitivity to circulating androgens. Hypertrichosis is excessive growth of thin vellus (nonpigmented) hair at any body site, and is not androgen dependent. Hypertrichosis results either from persistence of the lanugo-vellus hairs covering the body of the fetus or from augmented development of hair follicles.

The role of testosterone

Testosterone stimulates hair growth and increases hair size and pigmentation. Testosterone acts on the hair follicles by converting in to its active form called dihydrotestosterone. 5- alpha reductase, the substance which is responsible for this conversion is found in these hair follicles. Testosterone travels in the blood attached to a protein called sex hormone binding globulin or SHBG. It is taken to the target areas where the protein releases the hormone. In the target areas, it gets converted into the active form - dihydrotestosterone.

Clinical Picture of Hirsutism

Hair follicles usually become enlarged, and the hairs themselves become larger and darker. A woman whose hirsutism is caused by an increase in male hormones has a pattern of hair growth similar to that of a man. A woman whose hirsutism is not hormone-related has long, fine hairs on her face, arms, chest, and back.
Other symptoms associated with a high level of male hormones include acne and deepening of the voice and increased muscle mass. Main points in evaluating hirsutism are Abdominal symptoms

- Family history
- Breast discharge
- Extent of hair growth
- Menstrual, reproductive, and medication history
- Skin changes (i.e., acne, striae, acanthosis nigricans)
- Symptoms of virilization
- Use of hair removal methods
- Weight gain
- Height, weight, blood pressure (may suggest condition of androgen excess related to adrenal enzyme deficiencies)
- Documentation of hair amount, distribution, and characteristics
- Galactorrhea
- Abdominal and pelvic examination/palpation for masses
- Physical features of Cushing's syndrome ○ Striae ○ Acne
    - proximal muscle weakness
    - moon facies ○ central obesity
- Signs of virilization

- Acne
- Clitoromegaly ○ Deepening of voice ○ Hirsutism
- Increased libido ○ Increased muscle mass (primarily shoulder girdle) ○ Infrequent or absent menses ○ Loss of breast tissue or normal female body contour
- Malodorous perspiration ○ Temporal hair recession and balding

Diagnosis

Given that most women with hirsutism have idiopathic hirsutism or PCOS, the problem is how best to identify the small number of women who have other causes for their hirsutism. The basic approach to the differential diagnosis should be:

- Documentation of the degree of androgen excess
- Exclusion of the serious but rare causes of hirsutism such as ovarian and adrenal androgen-secreting tumors

There are several clinical findings that suggest one of the rare and more serious causes of hirsutism-

- Abrupt onset, short duration (typically less than 1 year), or progressive worsening of hirsutism.
- Onset in the third decade of life or later, rather than near puberty.
- Symptoms or signs of virilization.
- Moderately elevated (or higher) serum androgen concentrations, eg, in young women raise the possibility of an androgen-secreting tumor.

Laboratory Testing

- Serum androgens ○ serum testosterone values above 150 ng/dL (5.2 nmol/L) ○ serum free testosterone values above 2 ng/dL (0.07 nmol/L) and
  - serum dehydroepiandrosterone sulfate (DHEA-S) values above 700 mcg/dL (13.6 µmol/L)
- Serum prolactin above 20 g/L (0–20 ng/mL)
- Serum luteinizing hormone (LH) above normal values- ○ Follicular phase 2.0–15.0 U/L or 2.0–15.0 U/L ○ Ovulatory phase 22.0–105.0 U/L or 22.0–105.0 U/L ○ Luteal phase 0.6–19.0 U/L or 0.6–19.0 U/L ○ Postmenopausal 16.0–64.0 U/L or 16.0–64.0 U/L
- 17 OH Progesterone above normal○ Follicular <3.18 nmol/L or <1.0 ng/mL ○ Midluteal 9.54–63.6 nmol/L or 3–20 ng/mL
- Pelvic ultrasonography
- Testing for Cushing's syndrome
- Abdominal CT or MRI
- Laparoscopy or laparotomy
- Ovarian and adrenal vein sampling
- Dexamethasone suppression testing
- Clinical investigation tools
- GnRH agonist testing

Hirsutism profile-

It is a battery of tests performed on a female to ID the cause of increased hair growth. It includes the estimation of 3-alpha-androstanediol, androstenedione, DHEA-sulfate, 17-hydroxyprogesterone, total & free testosterone.

Ferriman-Gallwey score-

One method of evaluating hirsutism is the Ferriman-Gallwey score which gives a score based on the amount and location of hair growth on a woman. It is the semiquantitative system for the clinical assessment of the presence and severity of hirsutism in the premenopausal woman. Each of nine body areas is graded separately from no hirsutism (grade 0) to minimal hirsutism (grade 1) to marked hirsutism (grade 4). Then the grades of all areas are summed. A normal hirsutism score is less than 8.

Ferriman-Gallwey scoring

Prognosis

Most cases of hirsutism can be treated successfully with medication and cosmetic attention. It may require time and persistent use of these therapies, but most cases will eventually will respond to a combined approach.