Review Article

Hirsutism in adolescent: A review

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Abstract:

Hirsutism is an embarrassing condition of females characterized by excessive and unwarranted growth of terminal hair on the face and other androgen-dependent areas of sexual hair growth (male pattern). It is a common clinical problem for females and a sign of increased androgenic activities. Polycystic ovarian syndrome (PCOS) is the most common (70-80%) cause followed by idiopathic hirsutism. Other causes are congenital adrenal hyperplasia, androgen-secreting tumours, Cushing's syndrome, acromegaly, hypothyroidism, (rarely), hyperprolactinemia and medications. Hirsutism should be differentiated from hypertrichosis where excessive hair grows on non-selective and nonsexual areas of females without androgenic influence. Hirsutism imposes an extra psychological burden on adolescents when they are adapting to physical, endocrine and environmental changes. A hirsute adolescent girl may lose significant self-esteem and suffers from serious psychological comorbidities. Diagnosis and management of underlying causes of hirsutism especially PCOS in adolescent girls are controversial and challenging. Disease severity can be measured by using modified Ferriman–Gallwey scoring system. Clinical and laboratory evaluation for hyperandrogenism including testosterone is mandatory for females with significant hirsutism scores. A serum testosterone level > 200 ng/dL strongly indicates an adrenal or ovarian tumour and further evaluation is essential. For the management of hirsutism, a multimodal approach is necessary where hair removal and management of underlying causes should be continued at the same time.

Keywords: Hirsutism, PCOS, Adolescent.

Introduction:

Body hair is characteristic of both men and women, but its distribution is different in some specific parts of males and females. Women of most cultures have for countless years been concerned with the occurrence of hair on their bodies.[1] But in hirsutism excessive unwanted terminal hair grow in regions characteristic of masculine hair growth, i.e. upper lip, chin, side-burns, chest, upper pubic triangle and abdominal regions where terminal hair is normally not found in female.[2] Hirsutism is a common disorder and usually of benign aetiology affecting between 5% and 10% of women of reproductive age in the general population.[3] It causes a significant negative impact on the psychological well-being and quality of life of patient.[4] Adolescence is a very sensitive and important transitional time of life when teenagers start to notice their physical, mental and emotional changes. They start to establish self-image and adjust to the changing environment. Any disfigurement like hirsutism seriously hampers their self-esteem and confidence. Hirsutism in adolescents has some special issues regarding

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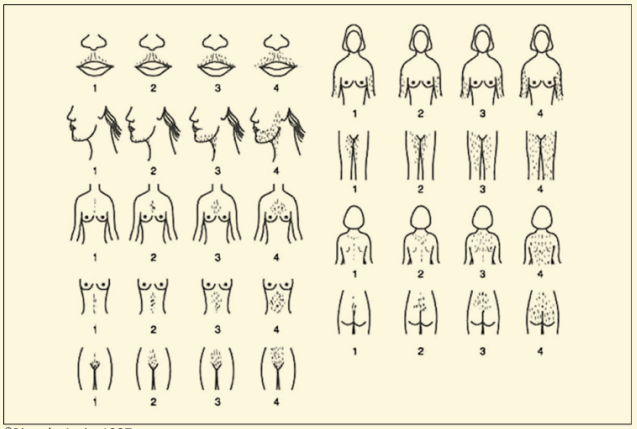
aetiology, clinical features, diagnosis and management.

Definition: Hirsutism Vs. Hypertrichosis

Hirsutism is the increased unwarranted hair growth of females with a selective male pattern distribution. Defining an individual woman as a hirsute needs consideration of her ethnic background and interpretation of normal. Hair growth varies widely among women, and distinguishing normal variations of hair growth from hirsutism is important.[5]

It also must be differentiated from hypertrichosis. In hypertrichosis excessive hair grow in generalized or localized areas, on non-selective and nonsexual areas (predominantly on forearms or lower legs) of the female body and does not depend on androgens. In hirsutism, hairs are of a terminal type and in hypertrichosis vellus type (non-terminal).[6] Hypertrichosis is generally genetically determined and not physiological.[6] It can be caused by the use of medication, hereditary factors, porphyria, thyroid dysfunction, anorexia nervosa, malnutrition, juvenile dermatomyositis, tuberculosis, endocrine disorders and even paraneoplastic syndrome but, not induced by hyperandrogenism.[6] Many systemic and topical medicines can cause hypertrichosis including acetazolamide, citalopram, corticotrophin, cyclosporine, diazoxide, glucocorticoids, metoclopramide, methyldopa, mercury poisoning, minoxidil, penicillamine, phenothiazines, phenytoin, reserpine, streptomycin, valproic acid, and heavy metals.[7]

Physicians define hirsutism clinically by using a qualitative tool Ferriman–Gallwey (F-G) scoring system.[8] It evaluates and quantifies hair in nine androgen-dependent areas in women. This scoring system evaluates nine different body parts (upper lip, chin, chest, upper back, lower back, upper abdomen, lower abdomen, arm, and thigh), with scores ranging from 0 (no excessive terminal hair growth) to 4 (extensive hair growth) for each body part evaluated. A maximum score of 36 is possible, but a score of ≥ 8 typically indicates hirsutism, a score above the 95th percentile for the population defines Hirsutism.[8]



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The method was modified in 2001, including 19 areas, with 10 additional fields: sideburn, the neck, the inside of the thighs, perianal region, forearm, foot, thumb and toes of the foot.[9]

Adolescence:

Adolescence is the developmental phase between childhood and adulthood when individual faces significant physical and psychosocial change. At this stage of hypothalamo-pituitary-gonadal and the hypothalamo-pituitary-adrenal axis starts to function. When these two physiological hormonal axes start their function the gonads and adrenal glands produce sex steroids. These sex steroids (estrogen and testosterone) are responsible for the physiosexual and psychosexual changes seen in the adolescence.[10]

Around this period increased circulating adrenal androgens induces a site-specific response on the hair follicles. The hairs of the scalp miniaturize, while the hair of the body, axilla, pubic, chest, and beard (boys) area change from vellus to terminal hair, contributing to the development of the secondary sex characteristics.[11] But in some situations, abnormally high level of androgen in female causes excessive growth of unwanted terminal hair in some specific areas (masculine hair growth, i.e. upper lip, chin, side-burns, chest, upper pubic triangle and abdomen). Hirsutism in adolescent girls is a more complex entity to manage.

Epidemiology:

Hirsutism affects more or less 10% of women of reproductive age except women of Far-East Asia.[12] Hirsutism is also more common among adolescent girls affecting 10.8% to 22.8%.[13-14]

Ethnicity: Body and facial hair growth in women significantly varies among different racial and ethnic

groups.Mediterranean, Hispanic, and Middle Eastern women have higher hair growth tendency and Asian has least. Upper normal limit of F-G score is 9 to 10 for Mediterranean, Hispanic, and Middle Eastern women and 8 for American women. An F-G score just 2 or above in a Chinese Han woman can be considered as hirsutism.[15]

Etiology

Androgen plays the key role in the pathogenesis of Hirsutism though there is some non-androgen etiologic factor.

Table I: Causes of hirsutism.

Major causes of hirsutism	
Excess androgen (≥80%)	
	Polycystic ovary syndrome (70%-80%)
	Other rare causes of androgen overproduction: 1.Non-classic congenital adrenal hyperplasia (NCCAH) (4.2%) 2.Androgen-secreting tumours 3.HAIR-AN (hyperandrogenism, insulin resistance, acne, obesity and acanthosis nigricans), 4.SAHA (seborrhea, acne, hirsutism and acanthosis nigricans)
	Cushing syndrome, Acromegaly, hypothyroidism, (rarely) hyperprolactinemia
Idiopathic (5%-20%)	
Drugs: Topical androgen use by a partner, exogenous androgens or anabolic steroids or valproic acid	

Androgenic causes account for more than 80% of hirsute patients including polycystic ovary syndrome (PCOS), which affects about 70-80% of androgenic hirsute women.[16] PCOS is the most common endocrine condition affecting between 8 and 13% of women of reproductive age and 6–18% of adolescent girls.[17] Hirsutism not only reflects circulating androgen levels, but it is also influenced by the peripheral metabolism of androgens, by the sensitivity of the target tissues to androgens, and by other hormonal variables, such as insulin resistance.[19]

Polycystic ovary syndrome (PCOS)

Polycystic ovary syndrome (PCOS), is a varied disease condition, the diagnosis of which is difficult and often delayed. Its diagnosis is based on oligo-anovulation (OA), biochemical or clinical hyperandrogenism (HA), and polycystic ovary morphology (PCOM) on ultrasound. The widely agreed Rotterdam criteria proposed 4 PCOS phenotypes in adult women: (A) OA + HA + PCOM, (B) OA + HA, (C) HA + PCOM, and (D) OA + PCOM. The Rotterdom criteria are recommended and endorsed by the 2018 international PCOS evidence-based guideline.[20-21] For adolescent girls diagnosis of PCOS is much tough as irregular character of menstruation, acne, polymorphic ovarian morphology and facial hair growth as normal physiological variation and there is more chance of over diagnosis if adult criteria are used in adolescent.[22] There are only very few recommendation for the diagnosis of adolescent PCOS. The 2018 international PCOS guideline updated the Rotterdam criteria and now recommends applying OA and HA while avoiding PCOM for PCOS diagnosis in adolescents.[21] A justified and well accepted definition of menstrual irregularities is also important (Table -II).

Table II: Definition of irregular menstrual cycles in adolescents according to time post menarche.[18]

Time post menarche	Definition of irregular menstrual cycles
Less than 1-year post menarche	Irregular menstrual cycles are normal pubertal transition
> 1 to < 3 years post menarche	< 21 or > 45 days
> 3 years post menarche	< 21 or > 35 days or < 8 cycles per year
More than 1-year post menarche	> 90 days for any one cycle
Primary amenorrhoea	No menarche by age 15 years or > 3 years post thelarche (breast development)

Rare causes:

Congenital adrenal hyperplasia (CAH) and non-classical congenital adrenal hyperplasia (NCAH) interfere with the biosynthesis of cortisol and aldosterone due to deficiency of enzyme 21-hydroxylase (21-OH) leading to hyperandrogenism and hirsutism. HAIR-AN (hyperandrogenism, insulin resistance, acne, obesity and acanthosis nigricans) and SAHA (seborrhea, acne, hirsutism and acanthosis nigricans) are also

two rare causes of hirsutism.[22] Cushing syndrome, acromegaly, hypothyroidism, hyperprolactinemia are other causes. Adrenal and ovarian androgen-secreting tumours can be presented with rapid and progressive hirsutism and virilization. These are rare but very important to diagnose.[22]

In some cases of hirsutism girls presented with a normal menstrual cycle, sonographically normal ovarian morphology, normal androgen level and no other clinical evidence for PCOS or other causes of hyperandrogenism. The situation is called "idiopathic hirsutism".[23] Among eumenorrheic women with mild hirsutism (a Ferriman–Gallwey hirsutism score of 8 to 15 in the United States), approximately half have idiopathic Hirsutism. [24]

Physio-Pathology:

The human body is covered by approximately 5 million hair follicles. The hair follicle population is mostly static for a lifetime but follicular size and type of hair can be varied depending on different factors, especially androgens. The number, thickness, and types of hair reflect the interaction between circulating androgen concentrations, local androgen concentrations, and the sensitivity of the hair follicle to androgens.[25]

Hairs are grouped into three categories according to their structure i. lanugo – fine, soft and silky prenatal hair; i. vellus – fine, soft and unmedullated post-natal hair occasionally pigmented, and seldom exceeding 2 cm but larger than lanugo hair and less than 0.03 mm in diameter iii. Terminal- longer, coarser, often medullated, at least 0.06 mm in diameter and often pigmented hair.[26]

In childhood terminal hair is present in certain body parts e.g. scalp, eyelashes and eyebrows, serving protective roles but some human hairs are secondary sexual characteristics. Some hairs are a sign of puberty for both sexes and are present in the axillae and the lower pubic triangle but some are features of sexual maturity of males that appear in the sexual specific areas (beard area, the chest and the upper pubic diamond). Hair follicles in those areas are totally androgen-dependent; under the influence of androgen, these hair follicles start to produce thick, dark-coloured hair which was previously tiny, pale, vellus hairs which were hardly visible.[26]

A hair follicle has a cyclical lifetime passes through three stages: growth (anagen), involution (catagen), and rest (telogen).[26]

The maximum duration of hair growth in different body areas depends on the length of the anagen phase of a hair cycle. The androgen induces the widening of the anagen phase, abnormal enlargement of the hair follicles and the transformation of vellus hairs into terminal hairs leading to hirsutism.[22]

PCOS is the most common cause of a consistent rise in androgen beyond early puberty in adolescent girls and women. Here persistently raised luteinizing hormone and insulin levels lead to increased androgen production within the ovarian theca. Moreover, raised insulin level suppresses hepatic production of sex hormone binding globulin, which results in increased levels of free testosterone.[27] Hirsutism not only reflects circulating androgen levels, but it is also influenced by the peripheral metabolism of androgens, by the sensitivity of the target tissues to androgens, and by other hormonal variables, such as insulin resistance.[27]

Quality of life of an adolescent with hirsutism:

Adolescence is the period of life when body and self-image are formed, a time of concern with physical appearance when the opinion of peers on body image is strong. A negative body image carries consequences that may adversely affect an adolescent's overall HRQoL such as psychosocial problems, low self-esteem, depression and eating disorders.[28] The disfiguring nature of the disease affects the quality of life of girl and the impairment is more in hirsutism patients with PCOS than those without PCOS.

Clinical evaluation and diagnosis:

Excessive unwanted hair imposes an emotional and psychological burden on adolescent girls and reasonable deserves a complete evaluation. Hirsutism is a clinical diagnosis and most women are concerned about their appearance due to excessive hair growth on the face, chin, upper lips and other areas. They seek help to remove their unwanted hair. But the multifactorial and multiorgan nature of this disorder makes it difficult to identify a single etiologic cause which necessitates a detailed clinical and diagnostic evaluation of the involved organs. A complete medical history including the age of thelarche, adrenarche, and menarche; menstrual history, including frequency and duration; rate of onset of symptoms (gradual or sudden), any signs or symptoms of virilisation (acne, deepening of the voice, infrequent menstruation, loss of breast tissue

or loss of normal female body contour, clitoromegaly, increased libido, increased muscle mass as in shoulder girdle, malodorous perspiration etc), history of weight gain or diabetes and drug history before onset should be taken. Family association and complete general physical and systemic examination should be done including palpation of the abdomen for any ovarian mass.[29] Insulin resistance can be clinically evident as acanthosis nigricans. A sudden start and fast advancing Hirsutism predicts androgen-secreting tumours. On the other hand, mild hirsutism around the time of menarche suggests androgen excess from a non-tumour ovarian origin, as in polycystic ovary syndrome.

Laboratory investigation:

Endocrine evaluation for hyperandrogenism is essential for all adolescent girls with an abnormal hirsutism score and/or clinical evidence of hyperandrogenism. Initially, serum testosterone (on days four to ten of the menstrual cycle) and DHEAS should be done to exclude a serious underlying disease which may cover for detection of most androgen-producing tumours.[7] A serum testosterone level > 200 ng/dL strongly indicates an adrenal or ovarian tumour. If serum testosterone is elevated despite a normal DHEAS level, an ovarian source is more likely. A high DHEAS level (> 700 µ g/dL) suggests benign or malignant adrenal pathology should be suspected as the cause of Hirsutism.[7]

If serum total testosterone level is normal but sexual hair growth is moderate/severe or sexual hair growth is mild but there is clinical evidence of a hyperandrogenic endocrine disorder (such as menstrual disturbance or progression despite therapy), measuring an early morning serum total and free testosterone by a reliable speciality assay is recommended. Serum testosterone may be normal to higher in case of benign pathologies such as PCOS and CAH.[30]

Females with hyperandrogenism or hirsute women with normal androgen levels having a family history and belonged to a high-risk ethnic group need screening of serum 17-hydroxyprogesterone levels for diagnosis of non-classic congenital adrenal hyperplasia (NCCAH).[30] Samples should be collected in the early morning (0700-0900 hours) in the early follicular phase. Levels less than 200 ng/dl exclude the CAH. Mildly increased levels between 300 and 1,000 ng/dl require an ACTH stimulation test.[30]

Patients with PCOS often have elevated free serum testosterone with increased luteinizing hormone (LH) and lowered follicle-stimulated hormone (FSH) (FSH/LH = 1:2 or 1:3). If lady presents with both amenorrhea and hirsutism, serum prolactin levels and thyroid function tests should be done to differentiate hyperprolactinemia and hypothyroidism.[30] Girls who have features of Cushing's syndrome 24 hours urinary free cortisol should be measured.[7] Pelvic ultrasonography is indicated for suspected ovarian neoplasm or a PCOS. Ultrasonography, MRI or CT scan of the adrenal gland may also be helpful.[29]

Identification of serious underlying disorders is the primary purpose of laboratory testing for hirsutism.

Gene study:

Searching for a causative gene is needed when congenital adrenal hyperplasia is taken under consideration as the cause of hirsutism or virilization.[31]

Management:

For hirsutism in adolescents, a multifaceted management approach should be taken including lifestyle modification, physical hair removal, and androgen suppression or blockade to slow or prevent new hair growth. The affected girl should be counselled about the life cycle of hair, explain the cause of slow response to treatment and to wait at least six months to decide on changing one treatment modality. They should also be informed about the importance of weight reduction.[32] As circulating androgen and follicular response to it remains at the centre of the pathogenesis of hirsutism pharmacotherapy for changing either the endocrine milieu or follicular responsiveness is crucial in the management of hirsutism. Considering the psycho-social impact of hirsutism, the hair removal procedure should be considered at the same time as the clinical and biochemical evaluation of the disease. So cases of hirsutism should be managed in a multimodal approach including i. androgen suppression, ii. peripheral androgen blockade iii. mechanical/cosmetic amelioration, and iv. destruction of unwanted hairs.

Lifestyle therapies:

Control of body weight by diet and lifestyle change is beneficial and considered a first-line treatment for obese women with hirsutism and PCOS. In obese women production of Sex Hormone Binding Globulin

(SHBG) decreases leading to raise free testosterone levels as compared to non-obese women.[33] Reduction of body weight by more than five percent has a significant improvement in their biochemical profile, including a reduction of testosterone, an increase in sex hormone-binding globulin, and an improvement in their Ferriman-Gallway scores.[34]

Medical therapy: The aim of medical treatment in hirsutism is to correct the hormonal imbalance and thereby stop further progress of hairiness and to reduce the amount of hair to improve the aesthetic appearance of the woman.

Lifestyle therapies:

Combined oral contraceptives (COC) are inexpensive and promote regular uterine bleeding. COCs are preferred as the first-line pharmacotherapy for young women with hirsutism. It acts by i. reducing androgen production (suppress gonadotropins production from anterior pituitary gland leading to reduction of luteinizing hormone (LH) induced ovarian androgen production), reducing androgen availability (stimulating the hepatic synthesis of SHBG which binds access free androgens) and reducing androgen action (direct receptor antagonist). Combined estrogen-progestin COCs contain the lowest effective dose of Ethinyl estradiol (EE) (usually 20 mcg) and progestin.[35] The progestins in COCs are attenuated derivatives of testosterone and may have mild androgenic activity. But the third (Nor-gestimate, gestodene, desogestrel) and fourth (Drospirenone, cyproterone) generation pills have progestins with no androgenic activity and may appear to be the preferred choice in the treatment of hirsutism.[36] Drospirenone and cyproterone) are structurally unrelated to testosterone and function as weak androgen receptor antagonists.[32] Drospirenone is a derivative of spironolactone having both anti-androgenic and mineralocorticoid properties. It blocks ovarian steroid production, reducing adrenal androgen synthesis and blocking peripheral androgen receptors in the dermis and pilosebaceous units.[37] But practically hair reduction with OCP monotherapy is not satisfactory and they do not remove existing hair.[38] One important message is, that hormonal therapy should not be started before menarche. An anti-androgen is suggested to add if the clinical response is suboptimal after six months of therapy. But for moderate-to-severe hirsutism, hormonal therapy and antiandrogens can be started concomitantly.[32] Antiandrogen should not be given initially as single therapy (considering the teratogenic potentials of these agents) unless ensuring reliable contraceptive measures. For adolescents and those who are not sexually active COCs or antiandrogen either can be given.

Spironolactone (SPA) is an aldosterone and androgen receptor antagonist that is structurally similar to progestins. It competes with dihydrotestosterone (DHT) for binding to the androgen receptor and inhibits enzymes involved in androgen biosynthesis. The starting dose is 50 mg twice daily and may be increased to a total daily dose of 200 mg. It takes at least six months to have any beneficial effect. Spironolactone is more effective in treating hirsutism when combined with COC, because, together, these drugs have complementary anti-androgenic actions, and COC ensures pregnancy prevention and menstrual cycle regulation.39 SPA is generally well tolerated, but contraindicated in renal impairment. It has a dose-dependent association with menstrual irregularity unless the patient uses a COC concomitantly. It rarely can cause hyperkalemia, diuresis and occasionally postural hypotension and dizziness early in treatment.40 For women engaged in sexual activity and at risk of being pregnant use of contraception is a must as SPA can affect the genital development in a male fetus.[40]

Flutamide has the same level of efficacy as spironolactone, but is not recommended due to the potential for hepatoxicity and finasteride has not had enough supportive data to use in adolescents.

Biological modifiers of hair follicular growth:

Topical eflornithine hydrochloride cream (13.9%) is a potent, irreversible inhibitor of the enzyme ornithine decarboxylase, which is necessary for the production of the polyamines that mediate cell migration, proliferation, and differentiation. The cream is applied to the face twice a day. Gradual improvement is seen in six to eight weeks. It can also be used in combination with laser treatments for better effects.[41]

Current options for hair removal:

Hormonal therapy can stop further progress of the disorder, but it has no effects on the removal of existing hair. Hair removal is the main patient expectation and ultimate treatment goal in hirsutism. Different temporary hair removal modalities including plucking, shaving, waxing, using depilatory creams, and bleaching are usually used

for localized mild hirsutism. Electrolysis and laser hair removal are the two permanent hair removal techniques. A particular procedure is selected according to the severity of hirsutism, adverse effects, past treatment cost, and the availability of treatment modality.[42-43]

Temporary self-care methods

These temporary methods can be convenient for the patients and they can do it as a self-care method at home; although multiple treatments are often needed and individual responsiveness must be taken into consideration.

Plucking-Plucking is a painful and time taking procedure for removing only a few hairs present in a small area where a total hair with the root is removed.[42-43]

Shaving- Manual and electrical shaving is simple, convenient, cheaper, and painless process for hair removal although many women do not accept it for some misconception. There is a common belief that shaving increases pigmentation, thickness and growth rate of hair but shaving influences none of these. Shaving can cause irritation, folliculitis, pseudofolliculitis, and infection.[42-43] Shaving requires continuous treatment; however if added to pharmacological therapies it may no longer be needed.

Waxing -Waxing is a mild painful slow process of hair removal taking weeks. It has some risk of burn, irritation, folliculitis, scarring, and postinflammatory dyspigmentation.[42-43]

The chemical depilation-Another painless inexpensive process is chemical depilation by using chemical depilatory agents thioglycolates. These agents dissolve the hair by disrupting disulfide bonds in the hair. It is remaining as one of the most common procedures but can cause the emission of an unpleasant sulfurous odour and irritant dermatitis (especially on the face), which may be followed by hyperpigmentation.[32]

Bleaching -Bleaching with products containing hydrogen peroxide and sulfates are not a method of hair removal but it can mask the appearance of pigmented hair. Burn, irritation, pruritus, and dyspigmentation are potential adverse effects.

Permanent methods:

Women with hirsutism who expect a permanent reduction of unwanted hair can choose one of the three recommended hair removal modalities: electrolysis, thermolysis and laser treatment. These techniques have more sustainable outcomes than depilatory techniques, as they can cause permanent damage to the follicle. They include electrolysis, laser and other light-based therapies including intense pulse light. These modalities destroy the base of the hair follicle without causing a scar on the surface. However, it is operator-dependent and may be associated with local side effects.

Electrolysis:

An epilation probe is inserted into the hair follicle and destroys the follicle by galvanic electrolysis (direct current) or by thermolysis (high-frequency alternating current). Each particular follicle needs to be treated individually and the results are very operator-dependent. The regrowth rate is about 40% and the best result is expected in dark and grey hair.[42] It is painful and can cause erythema, folliculitis, pseudofolliculitis, infection, scarring, and postinflammatory dyspigmentation.[43]

Laser hair removal:

Mostly used modalities include long-pulsed Ruby laser (694), long-pulsed Alexandrite (755 nm), long-pulsed Nd:YAG (1064 nm) and long-pulsed Diode (800-810 nm). Long pulsed ruby lasers (694) were the first devices on the market for hair removals and resulted in long-term hair reduction. Intense Pulse Light (IPL) emits polychromatic non-coherent light with wave-lengths from 400-1400 nm. For hair removal, a filter that filters the wave-lengths below 525-550 nm is often used.[44] All these photo epilation procedures interrupt hair growth temporarily, but permanent results depend on the number of sessions, fluence, and hair colour intensity. The optimal target is dark hair on fair skin, whereas blond, red, and white hairs are not suitable for laser.[45] The anagen hairs are the most suitable target for laser and IPL.[46]

Selecting a perfect device:

No device can give true permanent hair removal, however, long-term reduction is possible to achieve. Hair re-growth is usually finer and lighter, and a reduction of 10 to 40 percent can be achieved each session. So, multiple sessions should be scheduled with an appropriate treatment interval.[47] All FDA-approved photoepilation lasers including diode, alexandrite, ruby, and Nd:YAG lasers have the same range of hair reducing capacity.[48] All photoepilation lasers and IPL devices have been found effective for long-term reduction of pigmented terminal hair.[49] Hair removals with lasers or IPL are much less

effective in female patients suffering from Polycystic Ovarian Syndrome (PCOS) without hormonal thereby and require more treatment sessions.[50]

Patient education:

Patient counselling on the aetiology, petrophysical aspect of hirsutism, the life span of hair, achievable treatment expectations, and emotional support are crucial in the management of hirsutism. Regular follow-up to measure and document the response to treatment is also important; including repeating Ferriman-Gallwey scoring, taking photographs of affected areas, and retesting androgen levels after 3 to 6 months.[51]

Conclusion:

Hirsutism is commonly associated with underlying PCOS and endocrine diseases specially hyperandrogenism. Excessive unwanted hair imposes a significant psychological burden on women and it is more intense in adolescent girls. It is not only a cosmetic issue, hirsutism should be taken seriously. It needs multimodal approach including evaluation and management of underlying disease and hair removal. Hormonal evaluation (serum testosterone) is mandatory for all women with significant hirsutism.

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