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(REVIEW ARTICLE)

Hirsutism in Children: A Comprehensive Review of Etiology, Clinical Evaluation and Contemporary Management

Venugopal Reddy Iragamreddy *

Department of Paediatrics, Ovum Woman and Child Speciality Hospital, Bangalore, India.

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Abstract

Hirsutism in children is an uncommon yet clinically significant manifestation characterised by excessive terminal hair growth in androgen-dependent areas. Unlike adults, paediatric hirsutism frequently indicates an underlying endocrine, metabolic, genetic, or neoplastic disorder and therefore requires systematic evaluation. Recent advances have improved understanding of androgen biosynthesis, peripheral androgen sensitivity, and the complex interaction between insulin resistance and hyperandrogenism. Conditions such as premature adrenarche, non-classical congenital adrenal hyperplasia, adolescent polycystic ovary syndrome, and androgen-secreting tumours represent key etiologies. Early identification is crucial to prevent long-term metabolic, reproductive, and psychosocial complications. This review integrates current evidence on pathophysiology, evolving diagnostic strategies, updated laboratory markers, imaging modalities, and contemporary management approaches, offering a practical framework for clinicians managing hirsutism in children.

Keywords: Hirsutism; Pediatric Hyperandrogenism; Premature Adrenarche; Polycystic Ovary Syndrome; Congenital Adrenal Hyperplasia; Androgen Excess; Pediatric Endocrinology; Diagnostic Algorithm

1. Introduction

Hirsutism refers to excessive growth of coarse, pigmented terminal hair in androgen-dependent regions, including the face, chest, abdomen, and back. While common in adult women, its presence in children particularly in the prepubertal age group is unusual and often pathological. Childhood hirsutism may be the earliest clinical sign of endocrine disorders such as premature adrenarche, congenital adrenal hyperplasia, or evolving polycystic ovary syndrome (PCOS). Beyond cosmetic concerns, hirsutism has profound psychosocial implications, contributing to low self-esteem, social withdrawal, and emotional distress. Advances in paediatric endocrinology have emphasised early recognition, risk stratification, and long-term follow-up to mitigate future metabolic and reproductive consequences.

2. Definition and Pathophysiology

2.1. Definition

Hirsutism is defined as excessive terminal hair growth following a male-pattern distribution due to androgen action on hair follicles. It differs from hypertrichosis, which is androgen-independent and characterised by generalised excessive hair growth.

2.2. Pathophysiological Mechanisms

Recent research highlights that hirsutism results from a combination of:

* Corresponding author: Venugopal Reddy Iragamreddy

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- Elevated circulating androgens (testosterone, DHEA-S, androstenedione)
- Increased peripheral conversion of testosterone to dihydrotestosterone via enhanced 5-alpha reductase activity
- Increased sensitivity or density of androgen receptors in hair follicles
- Reduced sex hormone-binding globulin (SHBG), particularly in insulin-resistant states

Emerging evidence also implicates **hyperinsulinemia** as a key driver of ovarian and adrenal androgen excess, linking childhood obesity and early metabolic dysfunction to later hyperandrogenic states.

3. Epidemiology and Emerging Trends

The true prevalence of paediatric hirsutism remains unclear due to underreporting and ethnic variation. However, recent global trends indicate a rise in cases attributable to:

- Increasing childhood obesity
- Earlier pubertal onset
- Greater recognition of adolescent PCOS
- Environmental endocrine-disrupting chemicals

Girls are more commonly affected, particularly during early adolescence, while significant hirsutism in boys often signals pathological androgen excess.

4. Etiology of Hirsutism in Children

4.1. Physiological Causes

4.1.1. Premature Adrenarche

Premature adrenarche is characterised by early activation of adrenal androgen production, leading to pubic or axillary hair before 8 years in girls and 9 years in boys. Recent longitudinal studies suggest that while often benign, premature adrenarche may represent an early marker of future insulin resistance, PCOS, or metabolic syndrome, warranting long-term surveillance.

4.1.2. Pubertal Transition

Mild hirsutism may occur during early puberty due to physiological androgen rise, provided there is no virilisation or rapid progression.

4.2. Endocrine and Metabolic Causes

4.2.1. Polycystic Ovary Syndrome (Adolescent PCOS)

PCOS is the most common cause of hirsutism in post-menarchal adolescents. Current consensus emphasises cautious diagnosis during adolescence, requiring persistent hyperandrogenism with menstrual irregularity beyond two years post-menarche. Associated features include insulin resistance, obesity, dyslipidemia, and increased long-term cardiometabolic risk.

4.2.2. Non-Classical Congenital Adrenal Hyperplasia (NCAH)

NCAH due to partial 21-hydroxylase deficiency often presents in late childhood or adolescence with hirsutism, acne, and advanced bone age. Advances in biochemical testing and genetic analysis have improved diagnostic accuracy, allowing early intervention.

4.2.3. Cushing Syndrome

Although rare, endogenous cortisol excess should be suspected in children with hirsutism accompanied by growth failure, truncal obesity, and violaceous striae.

4.2.4. Thyroid and Prolactin Disorders

Hypothyroidism and hyperprolactinemia may exacerbate hirsutism by altering gonadotropin secretion and androgen metabolism.

4.3. Androgen-Secreting Tumours

Rapidly progressive hirsutism with virilisation strongly suggests an androgen-secreting tumour of adrenal, ovarian, or testicular origin. Recent guidelines emphasise early biochemical localisation followed by targeted imaging to prevent diagnostic delay.

4.4. Genetic and Syndromic Causes

HAIR-AN syndrome, severe insulin resistance syndromes, and rare androgen receptor abnormalities represent important yet under-recognised causes of severe paediatric hirsutism.

4.5. Drug-Induced Hirsutism

Exposure to exogenous androgens, anabolic steroids, danazol, valproate, or inadvertent transfer of topical testosterone preparations remains a preventable cause.

5. Differential Diagnosis

5.1. Hypertrichosis

Distinguished by diffuse hair growth unrelated to androgen action, commonly associated with medications, systemic illness, or genetic conditions.

5.2. Ethnic and Familial Variants

Certain populations exhibit higher baseline hair density without underlying pathology.

6. Clinical Evaluation

6.1. History

A structured history should assess age of onset, progression rate, pubertal development, growth pattern, menstrual history, family history, medication exposure, and symptoms of virilisation.

6.2. Physical Examination

Assessment includes anthropometry, blood pressure, acne, acanthosis nigricans, Tanner staging, and signs of virilisation. The modified Ferriman Gallwey score may be cautiously applied in post-pubertal adolescents.

7. Diagnostic Investigations

7.1. Laboratory Evaluation

Recent recommendations support an expanded hormonal panel including:

- Total and free testosterone
- DHEA-S
- Androstenedione
- Early-morning 17-hydroxyprogesterone
- LH, FSH
- SHBG
- Prolactin and thyroid profile
- Fasting glucose, insulin, HbA1c, lipid profile

Anti-Müllerian hormone (AMH) is emerging as a supportive marker in adolescent PCOS but should not be used in isolation.

7.2. Imaging and Bone Assessment

Pelvic ultrasound remains useful for ovarian assessment in adolescents. Adrenal imaging is reserved for suspected tumours. Bone age evaluation aids differentiation between benign and pathological androgen excess.

8. Management Strategies

8.1. General Principles

Management should be individualised, addressing the underlying cause, cosmetic concerns, metabolic health, and psychological well-being.

8.2. Lifestyle Interventions

Recent evidence confirms that weight reduction and physical activity significantly reduce androgen levels and improve insulin sensitivity, particularly in PCOS and premature adrenarche.

8.3. Pharmacological Therapy

- **Combined oral contraceptives** remain first-line for adolescent PCOS
- **Anti-androgens** (spironolactone, finasteride) are effective adjuncts
- **Metformin** improves metabolic parameters and reduces hyperandrogenism
- **Low-dose glucocorticoids** are reserved for confirmed NCAH

8.4. Cosmetic Therapies

Laser hair removal and electrolysis offer effective long-term cosmetic improvement, while topical eflornithine serves as an adjunct.

8.5. Tumour Management

Surgical excision followed by endocrine surveillance remains definitive.

9. Psychosocial Impact

Recent studies highlight significant psychological morbidity in children with hirsutism. Early counselling, family education, and mental health support are essential components of care.

10. Prognosis and Long-Term Follow-Up

While many cases are benign, conditions such as PCOS and premature adrenarche require long-term follow-up due to associated metabolic risks. Early intervention improves reproductive and cardiometabolic outcomes.

11. Conclusion

Hirsutism in children represents a clinical sign with diverse etiologies and important long-term implications. Advances in understanding androgen physiology, insulin resistance, and genetic contributions have refined diagnostic and therapeutic approaches. A structured, multidisciplinary strategy focusing on early identification, targeted investigations, and holistic management ensures optimal outcomes and improved quality of life.

Compliance with ethical standards

Disclosure of conflict of interest

The author declares that there is no conflict of interest regarding the publication of this manuscript. The author has no financial, personal, academic, or professional relationships that could be perceived to influence the content of this review.

Author Contributions

Dr. Venugopal Reddy solely conceptualised the study, performed the literature search, analysed and interpreted the available evidence, and drafted the manuscript. The author also critically reviewed the final version and approved it for submission.

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