

Premature greying of hair in children: A comprehensive review for pediatricians and primary care physicians

Venugopal Reddy. I *

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

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Abstract

Premature greying of hair (canities) in children is an increasingly recognized concern among parents and pediatricians. Although often benign, early hair depigmentation may be a clinical marker of nutritional deficiencies, autoimmune disorders, genetic predisposition, endocrine abnormalities, or oxidative stress related damage. The absence of standardized pediatrics guidelines leads to misdiagnosis, unnecessary anxiety, and missed opportunities for early intervention. This review synthesizes current evidence on epidemiology, pathophysiology, risk factors, diagnostic approach, and management of premature greying in children. A structured evaluation and targeted treatment of reversible causes especially vitamin B12, iron, vitamin D, zinc deficiency, and hypothyroidism can halt or partially reverse greying in many children. This article provides an evidence-based, clinically practical approach suitable for pediatricians, dermatologists, and general practitioners.

Keywords: Premature Canities; Pediatrics; Grey Hair in Children; Micronutrient Deficiency; Oxidative Stress; Thyroid Disorders; Trichology; Vitamin B12 Deficiency; Autoimmune Disorders

1. Introduction

Hair greying is classically associated with ageing. When it appears in children, however, it becomes a source of anxiety and confusion for parents, and clinicians often lack clarity on appropriate evaluation. In recent years, an increase in reported cases of premature greying among Indian school-aged children has been observed, driven by nutritional transitions, lifestyle changes, stress, and rising autoimmune disorders.

Despite being benign in most cases, early greying may serve as a clinical clue to underlying systemic disease. Early identification can facilitate timely intervention and prevent irreversible follicular melanocyte loss.

2. Definition

Premature greying is defined based on ethnicity due to genetic differences in melanocyte ageing.

Table 1 Age cut-off for Premature Greying based on Ethnicity

Population	Age cut-off for Premature Greying
Caucasian	<20 years
Asian / Indian	<25 years

* Corresponding author: Venugopal Reddy. I

African	<30 years
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For pediatrics practice, greying occurring before 18 years warrants clinical evaluation.

3. Epidemiology

Globally, premature greying affects 1–7% of children and adolescents, although true prevalence remains underestimated due to limited reporting.

Recent trends indicate

- Increasing cases in India, Middle East, South-East Asia
- Higher prevalence in children with
 - Atopic diathesis
 - Vitamin B12 deficiency
 - Vegetarian diet patterns
 - Autoimmune thyroid disease
 - Family history of premature greying

A school-based study from North India reported prevalence at 2.4%, with strong associations with micronutrient deficiencies.

4. Hair Pigmentation Physiology

Hair color depends on the production and distribution of melanin by follicular melanocytes. Two pigments contribute to hair color.

- Eumelanin – black/brown pigment
- Pheomelanin – yellow/red pigment

Melanocyte activity is active during the anagen (growth) phase and reduced during catagen and telogen.

Greying occurs due to

- Reduced melanocyte stem cells
- Decreased tyrosinase activity
- Accumulated free radical damage
- Melanocyte apoptosis

5. Pathophysiology of Premature Greying

5.1. Oxidative Stress (Central Mechanism)

- Recent research identifies oxidative stress as the key driver in premature greying.
- Mechanisms include
 - Accumulation of hydrogen peroxide (H₂O₂) in the hair shaft
 - Reduced catalase and methionine sulfoxide reductase activity
 - Mitochondrial dysfunction
 - Melanocyte DNA damage

Multiple studies show that children with premature greying exhibit elevated oxidative stress markers.

5.2. Nutritional Deficiencies

Strongest evidence associations

- Vitamin B12 deficiency

- Iron deficiency (low ferritin)
- Vitamin D deficiency
- Zinc deficiency
- Copper deficiency (rare but significant)
- Folate deficiency

These nutrients are directly involved in

- DNA synthesis
- Methylation pathways
- Antioxidant defense
- Melanin production

5.3. Autoimmune Mechanisms

Includes

- Alopecia areata overlap
- Vitiligo spectrum disorders
- Thyroid autoimmunity (Hashimoto thyroiditis)

Autoimmune activity may target melanocytes, leading to pigment loss.

5.4. Genetic Predisposition

Familial premature greying is common.

5.4.1. Genes linked to early depigmentation

- IRF4 – regulates melanin production
- BCL2 – inhibits melanocyte apoptosis
- MITF – melanocyte survival gene

Children with a parent who developed greying before age 25 have a 4–7× higher risk.

5.5. Endocrine Causes

Premature greying is linked with

- Hypothyroidism
- Hyperthyroidism
- Type 1 Diabetes

Thyroid hormones strongly influence hair follicle cycling and melanin expression.

5.6. Lifestyle and Environmental Factors

- Air pollution and heavy metal exposure
- Chronic stress and poor sleep
- Ultra-processed foods
- Overuse of chemical hair products, SLS shampoos, heat styling

Children today face higher levels of oxidative burden due to modern lifestyle factors.

6. Clinical Presentation

Common patterns include

- Isolated grey strands
- Patchy clusters

- Diffuse greying
- Grey regrowth in alopecia areata patches
- Associated scalp issues (seborrhea, dandruff, atopy)

7. Differential Diagnosis

Table 2 Differential Diagnosis of Premature Greying in Children

Condition	Key Features
Nutritional deficiencies	Reversible, associated with poor diet
Autoimmune disorders	Vitiligo, alopecia areata
Thyroid disorders	Altered growth, fatigue, weight issues
Genetic premature canities	Strong family history
Post-inflammatory greying	Following dermatitis or trauma
Systemic diseases	Progeroid syndromes, mitochondrial disorders
Drug-induced	Chemotherapy, valproate, antimalarials

8. Diagnostic Evaluation

8.1. History

- Age of onset
- Progression speed
- Family history
- Vegetarian/vegan diet
- Fatigue, weight issues (thyroid)
- Autoimmune disease history
- Stress, sleep hygiene
- Hair care practices

8.2. Physical Examination

- Pattern and distribution of grey hair
- Scalp Dermoscopy
- Skin examination for vitiligo
- Nails (pitting suggestive of alopecia areata)
- Growth charting
- Pubertal staging

8.3. Recommended Investigations

Table 3 Recommended Laboratory Tests and Purpose

Investigation	Purpose
CBC	Anaemia
Ferritin, Iron, TIBC	Iron deficiency
Vitamin B12	Strong association with greying
Folate	Nutritional evaluation
Vitamin D	Hair follicle health
Serum Zinc	Oxidative balance
Serum Copper	Melanin synthesis
TSH, Free T4	Thyroid disorders

Anti-TPO	Autoimmune thyroiditis
ANA	Autoimmune screening
Dermoscopy	Follicular melanin evaluation

9. Management

9.1. Treat Reversible Causes

9.1.1. Nutritional Deficiencies

Table 4 Treatment of Nutritional Deficiencies

Deficiency	Treatment
Vitamin B12	IM/Oral B12 1000 mcg weekly × 4 weeks
Iron	3–6 mg/kg/day elemental iron
Vitamin D	60,000 IU weekly × 6 weeks
Zinc	0.5–1 mg/kg/day
Copper	Only if confirmed; avoid unnecessary supplementation

9.2. Antioxidant Therapy

9.2.1. Helpful agents

- Vitamin C
- Vitamin E
- Omega-3 fatty acids
- Calcium pantothenate
- PABA (limited evidence)

9.2.2. Natural antioxidants

- Amla
- Curry leaves
- Black sesame seeds
- Almonds, walnuts

9.2.3. Topical Therapies

- Melitane™ peptide – stimulates melanogenesis
- Topical minoxidil – if associated with hair thinning (not pigment regeneration)
- Avoid: sulphates, parabens, artificial dyes

9.3. Lifestyle Modification

- Reduce junk food
- Increase antioxidants and proteins
- Adequate sleep
- Stress management
- Avoid heat styling
- Use mild shampoos

10. Prognosis

- Nutritional causes: high reversibility within 3–6 months
- Genetic causes: low reversibility

- Autoimmune causes: variable outcomes
- Early diagnosis improves results significantly

11. Prevention

- Balanced diet with adequate micronutrients
- Screening for vitamin B12 and vitamin D in high-risk children
- Healthy sleep routine
- Avoid early chemical exposure to hair

12. Clinical Algorithm for Paediatricians

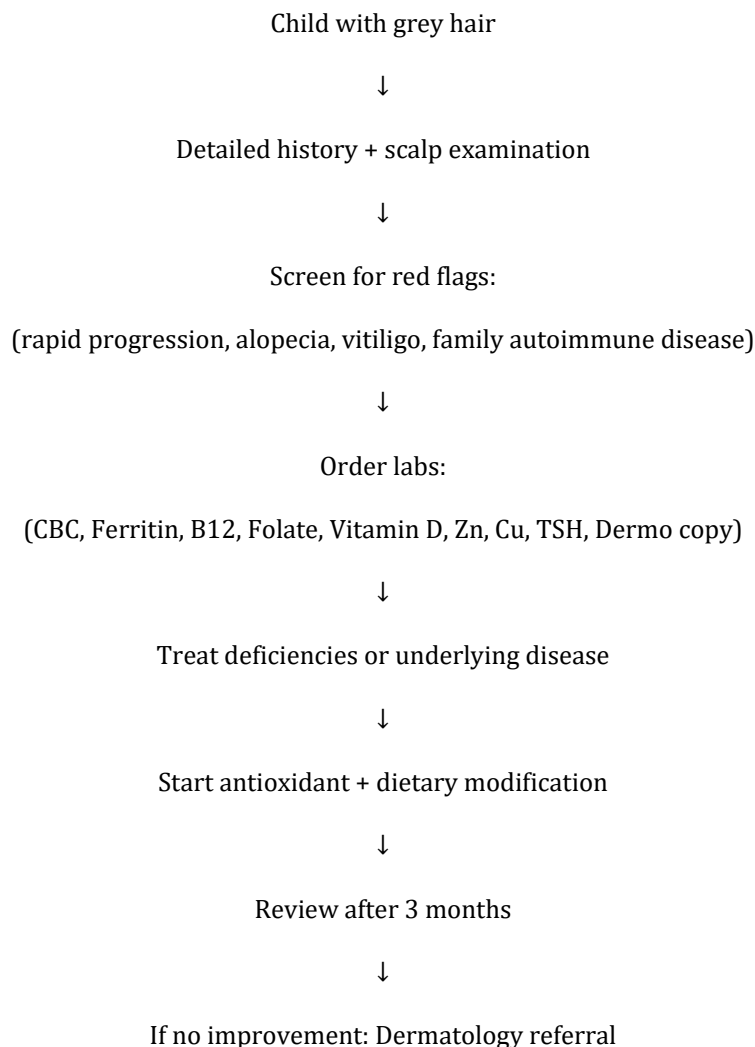


Table 5 Causes of Premature Greying in Children

Category	Examples
Nutritional	B12, Iron, Vitamin D, Zinc, Copper deficiency
Autoimmune	Alopecia areata, Vitiligo, Thyroiditis
Endocrine	Hypothyroidism
Genetic	IRF4, BCL2, MITF mutations
Environmental	Pollution, stress, food habits

Drug-induced	Chemotherapy, valproate
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Table 6 Investigation–Based Approach

Laboratory Test	Interpretation	Action
Low ferritin	Iron deficiency	Iron therapy
Low B12	Megaloblastic risk	B12 supplementation
Low Vitamin D	Follicle health	Vit D therapy
Low Zinc	Oxidative stress	Zinc supplementation
High TSH	Hypothyroid	Levothyroxine
Positive ANA	Autoimmune	Dermatology/Rheumatology referral

13. Discussion

Premature greying in children requires a balanced approach that avoids over-investigation yet ensures identification of reversible causes. Micronutrient deficiencies, particularly vitamin B12, iron, zinc, and vitamin D, are prevalent in Indian children due to vegetarian dietary patterns and limited sun exposure. Autoimmune thyroid disease and alopecia areata are important considerations in children with rapid progression of greying or associated hair loss.

Current evidence highlights the central role of oxidative stress in premature canities. This underscores the importance of antioxidant-rich diets, minimizing environmental exposures, and early correction of micronutrient deficiencies. Long-term studies are needed to clarify the extent of reversibility of pediatric greying and evaluate newer topical depigmentation agents.

14. Conclusion

Premature greying in children is a clinically relevant condition with significant parental concern. Early greying may be the first visible sign of underlying nutritional or autoimmune disease. A structured approach involving history, targeted investigations, nutritional correction, antioxidants, and lifestyle modifications forms the foundation of management. Most nutritional causes are reversible, especially when addressed early. Pediatricians should adopt an evidence-based evaluation strategy to optimize outcomes.

Compliance with ethical standards

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Disclosure of conflict of interest

The author declares no conflict of interest.

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