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Clinical Study of Premature Canities and Its Association with Hemoglobin, Serum Calcium, Vitamin B12, Vitamin D and Ferritin Levels

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ABSTRACT

Background: Premature canities (PHG), or early-onset hair graying, is a multifactorial condition influenced by genetic, environmental, and nutritional factors. Among these, biochemical deficiencies—particularly in vitamin B12 and vitamin D—have gained attention for their possible role in hair pigmentation. This study aims to assess the association between various biochemical markers and the severity of PHG.

Methods: A cross-sectional study was conducted over 1.5 years at KD Medical College Hospital, Mathura. Seventy individuals aged 15–30 years presenting with premature graying were enrolled. Data collection included detailed history, grading of PHG severity, and biochemical investigations, including hemoglobin, ferritin, calcium, vitamin B12, and vitamin D. Statistical analysis was performed to evaluate correlations between these markers and PHG severity.

Results: The mean age of participants was 21.47±5.94 years, with the highest prevalence in the 15–20 age group. A positive family history of PHG was found in 57.14% of cases, and 40% reported stress as a contributing factor. Notably, serum vitamin B12 (mean 212.34±179.00 pg/mL; r=-0.61; p=0.01) and vitamin D levels (mean 18.41±15.39 ng/mL; r=-0.52; p=0.01) showed a statistically significant inverse correlation with PHG severity. Hemoglobin, ferritin, and calcium levels did not show a significant association.

Conclusion: Vitamin B12 and vitamin D deficiencies are significantly associated with the severity of premature canities. Early nutritional assessment and supplementation may serve as effective strategies for managing PHG. Further studies are warranted to explore additional genetic and environmental contributors.

Key-words: Premature Canities, Hemoglobin, Serum Calcium, Vitamin B12, Vitamin D, Ferritin

INTRODUCTION

Premature canities, or premature graying of hair, is a condition characterized by the early onset of hair graying before the typical age range associated with chronological aging.

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It is a condition that not only alters physical appearance but also significantly impacts self-esteem and societal acceptance due to its implications on aesthetics and social perception [1]. Hair is one of the defining features of mammals and plays a vital role in shaping physical appearance and self-image. The onset of hair graying is influenced by multiple factors, including genetics, environmental exposures, and nutritional imbalances, all of which contribute to its complex etiology [2].

Hair color in humans varies widely and is primarily determined by the pigmentary unit of hair follicles. This unit functions most efficiently during post- adolescence and early adulthood, with the diversity in hair color attributed to the quantity and ratio of eumelanin (black-

brown pigment) and pheomelanin (reddish-brown pigment) [3]. These variations in hair pigmentation are especially notable among different ethnic groups, such as Asians, Africans, and Caucasians. However, irrespective of ethnicity or gender, the process of hair graying, medically termed "canities," occurs universally with age [4].

Graying of hair occurs due to two primary processes: weathering of the hair shaft and aging of the hair follicle. Weathering involves the progressive degeneration of hair fibers from root to tip, while aging of the follicle is associated with a decline in melanocyte activity, resulting in reduced pigmentation and diminished hair production [5].

Studies suggest that approximately 50% of individuals experience significant hair graying by the age of 50 years. The onset of graying varies by ethnicity, beginning in the mid-30s for Caucasians, late 30s for Asians, and mid-40s for individuals of African descent [6]. Premature graying of hair, or premature canities, is defined as the appearance of gray hair before the age of 20 in Caucasians, 25 in Asians, and 30 in individuals of African descent. Despite its prevalence, the exact pathogenesis of premature canities remains unclear [7]. However, it is widely accepted that the condition has a multifactorial etiology, involving genetic predisposition, oxidative stress, environmental factors, and nutritional deficiencies. Genetic factors are often considered the primary cause, but other contributors include autoimmune disorders such as vitiligo, pernicious anemia, and autoimmune thyroid diseases. Premature canities have also been associated with certain syndromic conditions like Werner syndrome, as well as environmental exposures such as ultraviolet light, smoking, and the use of certain medications.

MATERIALS AND METHODS

Study Design- This cross-sectional observational study was conducted at the KD Medical College Hospital and Research Center over 1.5 years. It included participants aged 15–30 who had premature canities. The subjects were selected based on strict inclusion and exclusion criteria, and their biochemical parameters were evaluated.

Inclusion Criteria

- Age between 15–30 years
- Presence of at least five gray hairs confirmed by clinical examination
- Ability to provide informed consent for participation

Exclusion Criteria

- Age above 30 years
- History of chronic scalp disorders such as alopecia areata or psoriasis
- Use of hair dyes or chemical treatments within the past six months
- Pregnancy or lactation
- Any pre-existing systemic illness known to influence hair pigmentation

Hemoglobin Levels- Evaluated through Complete Blood Count (CBC) to detect anemia, which can affect hair follicle health.

Serum calcium levels were examined to understand their role in keratinocyte function and melanin production.

Vitamin B12- Assessed owing to its essential role in DNA synthesis and hair pigmentation.

Vitamin D- Measured for its impact on hair follicle cycling and melanin production.

Ferritin Levels- Evaluated as a key indicator of iron storage, influencing hair growth and color maintenance.

Data Collection- The study used a detailed questionnaire that captured demographic data, family history, lifestyle habits, and dietary patterns. Each participant underwent a thorough clinical assessment, including scalp examination and severity grading of premature canities. Laboratory investigations included the following.

Statistical Analysis- Statistical analysis was performed using SPSS software, and the Pearson correlation test was used to determine the associations between premature canities and biochemical markers.

RESULTS

Demographic characteristics of 70 patients with premature hair greying (PHG), such as age, gender, BMI, severity, site of greying, family history, and stress factors, are tabulated in Table 1.

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Table 1: Demographic variables

Variables		N	%
Age groups	15-20 years	33	47.14
	21-25 years	16	22.85
	26-30 years	21	30.01
Gender	Male	34	48.57
	Female	36	51.42
BMI	Obese	23	32.86
	Normal	20	28.57
	Underweight	16	22.87
	Overweight	11	15.71
Severity	Mild	40	57.14
	Moderate	20	28.57
	Severe	10	14.29
Site of Progressive Hair Graying (PHG)	Vertex	24	34.29
	Frontal	19	27.14
	Diffuse	15	21.43
	Temporal	12	17.14
Family history	Yes	40	57.14
	No	30	42.86
Stress Factor	Yes	28	40
	No	42	60

Haemoglobin (p=0.12), ferritin (p=0.08), and calcium (p=0.09) show weak to moderate negative correlations with premature greying. In contrast, vitamin B12 (p=0.01) and vitamin D (p=0.01) exhibit moderate to strong negative correlations with premature greying. This implies that lower levels of vitamin B12 and vitamin D are significantly associated with an increased likelihood of premature greying (Table 2).

Table 2. Correlation analysis between biochemical parameter and premature greying of hair

Parameters	Mild (Mean±SD)	Moderate (Mean±SD)	Severe (Mean±SD)	p-value
Haemoglobin (g/dL)	14.69±1.31	13.59±1.73	12.87±2.59	0.12
Ferritin (ng/mL)	183.63±123.06	99.54±98.79	85.28±81.58	0.08
Calcium (mg/dL)	9.67±0.48	9.46±0.81	47.13±80.30	0.09
Vitamin B12 (pg/mL)	259.16±191.46	201.58±147.03	45.12±9.39	0.01*
Vitamin D ((ng/mL)	30.80±10.02	14.20±20.78	9.27±2.78	0.01*

**Statistically significant*

The bar graph illustrates a progressive decline in Vitamin B12 and Vitamin D levels with increasing severity of premature canities. Other parameters like hemoglobin, ferritin, and calcium show relatively less variation across severity levels (Fig. 1).

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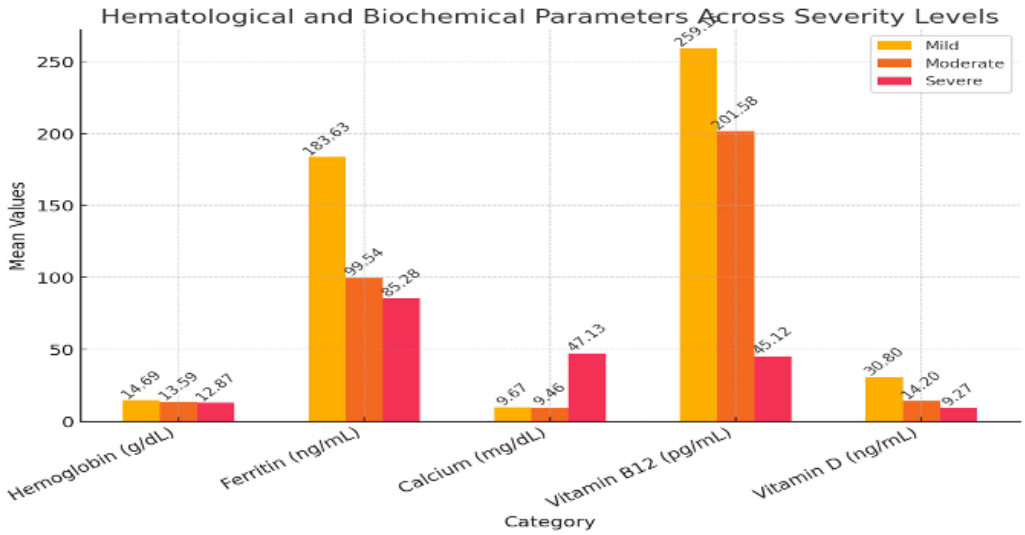

Fig. 1: Hematological and Biochemical Parameters Across Severity Levels

Fig. 2 illustrates the distribution of symptom duration among the study population. The mean duration was 31.73±18.07 days, indicating considerable variability in individual symptom persistence.

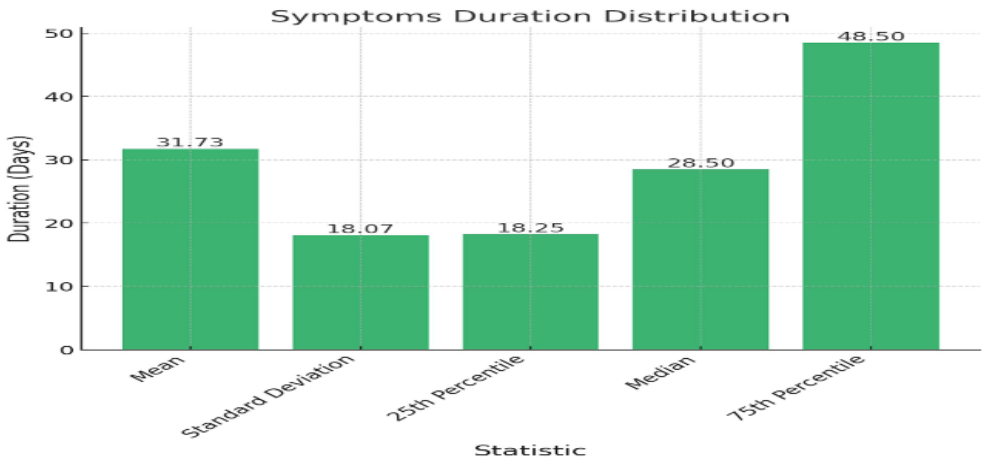

Fig. 2: Symptoms Duration distribution in study population

Individuals with low B12 levels had a higher prevalence of severe graying. A strong inverse correlation was observed between Vitamin D deficiency and premature canities, suggesting that supplementation could be beneficial. Genetic predisposition remained a dominant factor, with over 57% of cases reporting a family history of premature graying.

DISCUSSION

In this cohort of 70 young adults (mean age 21.5±5.9 years), the highest prevalence of premature canities (PHG) occurred in the 15–20-year bracket, and the majority (57.1%) exhibited mild hair graying. Notably, 57.1% reported a family history, reaffirming the strong genetic predisposition described by Panhard *et al.* [8].

The acute awareness of graying—mean symptom duration 31.7±18.1 days—suggests that even minor pigmentary changes spur early clinical presentation.

Our key biochemical findings show a stepwise decline in serum vitamin B12 (259 → 45 pg/mL) and vitamin D (30.8 → 9.3 ng/mL) from mild to severe PHG with both correlations reaching statistical significance (p=0.01). This dose–response pattern supports a causal link: vitamin B12 deficiency impairs melanocyte DNA synthesis and melanin production [9,10], while vitamin D receptor signaling governs keratinocyte differentiation and melanogenesis [11,12].

Although hemoglobin, ferritin, and calcium exhibited inverse trends (hemoglobin 14.7 → 12.9 g/dL; ferritin 183 → 85 ng/mL; calcium 9.67 → 9.46 mg/dL), none

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reached significance (p=0.08–0.12). These near-significant associations hint that chronic low iron or calcium stores may subtly compromise follicle matrix proliferation and keratinocyte signaling, warranting larger or longitudinal studies [13,14].

Oxidative stress emerges as a unifying mechanism: accumulation of H_2O_2 in aging follicles damages melanocytes and blunts methionine sulfoxide repair, driving pigment loss [15-18]. Trueb's work further underscores ROS as a central aging driver in hair [19]. Psychosocial factors also appear relevant: 40% of participants cited stress, which accelerates telomere attrition and oxidative DNA damage in melanocytes [20,21], potentially compounding nutritional and genetic susceptibilities.

Taken together, our data reinforce a multifactorial model for PHG—genetic predisposition [8], nutritional deficits in B12 and D [10,14], oxidative damage [16,18], and stress-mediated cellular aging [20,21]. Clinically, early screening for these modifiable factors and targeted supplementation (±antioxidants) could delay or ameliorate PHG progression. Future work should adopt longitudinal designs to clarify causality, explore gene–nutrient interactions, and test interventional efficacy across diverse ethnic groups [22-24].

CONCLUSIONS

This study established a strong correlation between premature canities and deficiencies in vitamin B12 and vitamin D levels, emphasizing the need for routine nutritional screening in affected individuals. Although hemoglobin, ferritin, and calcium levels were not significantly associated, their role in hair health requires further investigation. Dietary modifications, supplementation, and lifestyle changes should be considered as preventive and therapeutic measures against premature graying.

Future research should focus on understanding the mechanistic pathways linking these deficiencies to premature canities and exploring interventions that could potentially delay or reverse the progression of hair graying.

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