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A CLINICOEPIDEMIOLOGICAL STUDY OF PREMATURE CANITIES OF DEGREE COLLEGE STUDENTS IN THE RURAL AREA

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ABSTRACT
Premature graying of hair, also called 'premature canities', refers to diffuse loss of hair colour, especially of scalp hair, at an age earlier than that generally accepted as physiological i.e, before the age of 20 years in whites, 25 years in Asians and 30 years in Africans. The beard and moustache areas commonly become gray before scalp or body hair. To study the clinico-epidemiological aspects of premature canities. To correlate serum hemoglobin levels in patients with premature canities. This was a cross-sectional observational study wherein 1400 degree college students between the age group of 18-30 years in the field practice area of B.G Nagara, Mandya district, Karnataka between November 2011 and May 2013 were screened for premature graying of hair and a clinicoepidemiological study was conducted in those found to have premature graying. Haemoglobin levels were determined and the data was analysed. Out of 1400 students screened, 532 (38%) were males and 868 (62%) were females, among which 371 (26%) had premature graying of hair. Among 371 students 173 (47%) were males and 198 (53%) were females. The commonest age of presentation was 18-21 years. Positive family history was found in 50.4%. Exacerbating factors included change/usage of hard water (67%) and emotional stress (22%). History of smoking was elicited in 4% of patients. Commonest site of distribution of gray hairs was vertex followed by parietal, occipital and frontal areas of scalp. Anemia was found in 11% of patients. In our study, the prevalence of premature canities was 26%. Genetic predisposition was present in half the number of patients. Change of water/hard water usage was the most common aggravating factor, followed by psychosocial stress and smoking. Anemia was found in 11% of patients with premature canities.

INTRODUCTION

In humans, hair pigmentation depends entirely on the presence of melanin from melanocytes in the hair bulb epithelium [1]. Melanogenic activity in the hair follicle is closely related to the hair cycle. Melanin production occurs only during the anagen phase where melanocytes are located in the hair bulb. Some melanocytes undergo apoptotic deletion during catagen, but amelanotic melanocytes located in the outer root sheath serve as stem cells that replace the melanogenic melanocytes in the hair bulb following their apoptosis during the telogen phase of melanogenesis [2, 3].

Melanocyte stem cells are located in the lower permanent portion of the hair follicle to replenish the pool of melanocytes and loss of melanocyte stem cells is responsible for hair graying [4]. Graying is an expression of a gradual decrease in the function of the melanocytes, the pigment producing cells located near each hair bulb in the lower region of the follicle.

Hair graying or canities is closely related to chronological age, and the age of its onset is largely controlled by genetics, regardless of gender and race, where age of onset varies from race to race and ethinicity; graying of hair is progressive and permanent [5].

Loss of pigmentation is due to decrease or eventual cessation of tyrosinase activity in the lower bulb. Melanocytes are absent from the bulbs of white hairs although non-melanized melanocytes are still present in the outer root sheath [6].

Graying usually appears at the temples first, then the vertex and finally occiput. Gray hairs are thicker and longer than normally pigmented hairs [7]

Premature graying of hair also called as 'premature canities', refers to varying degrees of admixture of white and black hairs, at an age earlier than that generally accepted as physiologic, before the age of 20 years in whites, 25 years in Asians and 30 years in Africans [1,2,6,7].

Premature hair graying has significant adverse effects on the appearance, self-esteem, and socio-cultural acceptance of the affected individual. It is often viewed as a sign of old age and loss of health and vigor. Affected individuals are often subjected to social stigma, discrimination, and difficulties in marriage [8].

According to free radical theory of graying, oxidative stress promotes premature apoptosis of melanogenic melanocytes in the hair bulb leading to premature graying [4,6]. Understanding the mechanism and pathophysiology behind the premature graying of hair followed by appropriate intervention leads to better aesthetic value and social interaction.

This study was undertaken to know the burden, incidence and prevalence of premature graying of hair and its relation to anemia and malnutrition along with triggering factors like family history, hard water, smoking, pollution, stress, lack of sleep, lifestyle and hair care practices.

AIMS AND OBJECTIVES

1) To study the clinico-epidemiological aspects of premature canities.
2) To correlate serum hemoglobin levels with premature canities.

METHODS AND MATERIAL

This study was conducted between November 2011 to May 2013 among 1400 students in the age group of 18-30 years in the field practice area of B.G Nagara, Mandya, Karnataka. A written informed consent was taken from the students, before ensuing the study. Graying of hair was considered premature if its onset was before the age of 25 years. Diagnosis of premature graying was made based on history and the number of gray hairs appreciable on scalp during examination. A detailed history of onset, duration, aggravating factors, family history of graying were recorded on a predesigned proforma. Scalp examination was done for all cases and the number of gray hairs were counted. Severity was graded into mild (<20 gray hairs), moderate (20-50 gray hairs), severe (50–100 gray hairs) and very severe (>100 gray hairs). Patients' blood samples were collected to perform haemoglobin percentage. Results were tabulated and analysed.

RESULTS

The clinical data is shown in table 1.

In total, 1400 students screened, out of which 371 (26%) students had premature graying. Among 371 students with premature canities, 173 (47%) were males and 198 (53%) were females. The most common age of presentation was 18-21 years with 258 (69%) patients and the mean age of our patients was 20.87+/-2.67 years. Sudden onset of graying was reported in 28 (8%) patients and insidious onset in 343 (92%). The mean duration of premature canities in our patients was 2.16 +/- 3.45 years. Vertex (89%) was the most common initial site of premature graying, followed by parietal and occipital areas. Distribution of gray hairs was predominantly over the vertex (95%), followed by parietal (84%), occipital (66%), frontal (55%), temporal (27%) and was diffuse in 25% (table 2).

Among our patients, 4% gave a history of smoking. Majority of patients (67%) reported change of water/hard water to be the aggravating factor, followed by stress (22%). Positive family history was found in 187 (50.4%) patients (table 3), with a mean age of onset of 20.79 +/- 2.70 years and 184 (49.6%) patients had a negative family history with a mean age of onset of 20.96 +/- 2.64.

Graying of hair was mild in 55% of patients, moderate in 23%, severe in 16% and very severe in 6%. Blood investigation revealed that 39 (11%) patients had anemia (table 4), of which, 29 (8%) students had mild anemia, 6 (2%) students had moderate anemia and 4 (1%) students had severe anemia.

Table 1. clinical features in premature canities students

Features	N= 371 (%)
Gender	
Male	173(47)
Female	198(53)
Age distribution in years	
18-21	258 (69)
22-25	88 (24)

26-30	25 (7)
Duration of disease	
< 1 year	37 (10)
1 – 2 years	200 (54)
3 – 4 years	94 (25)
5 – 6 years	29 (8)
>7 years	11 (3)

Table 2. Distribution pattern in Premature canities

Features	**N= 371 (%)**
Distribution over scalp	
Temporal	102 (27)
Vertex	352 (95)
Parietal	313 (84)
Occipital	245 (66)
Frontal	203 (55)
Diffuse	92 (25)

Table 3. Family history

Family history	**N = 371 (%)**
Positive	187 (50.40)
Negative	184 (49.60)

Table 4. Haemoglobin levels in our patients

Haemoglobin % with grade of anemia	**N= 371 (%)**
<7mg/dl (severe)	04 (1)
>7 – 10mg/dl (moderate)	06 (2)
>10-11 mg/dl (mild)	29 (8)
>11 mg/dl	312 (84)

DISCUSSION

Greying of hair is a physiological phenomenon. Greying, occurring at an age earlier than that generally accepted as physiological i.e, before the age of 20 years in whites, 25 years in Asians and 30 years in Africans is called premature canities. Etiology of greying of hair is believed to be multifactorial including genetic, environmental factors, nutritional status and oxidative stress; but its exact pathogenesis has not yet been detected. Premature graying of hair is also seen in Werners syndrome, Rothmund-Thompson syndrome, Progeria, Books syndrome, Cri du chat syndrome, Ataxia telengectasia, HIV infection, Hypothyroidism, Vitamin B12 deficiency, Hodgkin's lymphoma, Pernicious anemia, drugs like chloroquine, mephensin, phenylthiourea, triparanol, fluorobutyrophenone, dixyrazine, imitinab, topical dithranol, chrysarobin, resorcin [5].

In our study comprising of 1400 students, 371 (26%) had premature graying including 173 males (47%) and 198 females (53%). Sixty eight percent were in the age group of 18-21 years, 29% were in the age group of 22-25 years and 3% were in the age group of 26-30 years. Females outnumbered males (M:F=1:1.14) and the mean age of students with premature graying was 20.87±3.45 years. This is comparable with the study done by Bhat [9], wherein male to female ratio was 1:1.1. In our study, the prevalence rate of premature graying was 26%. In a study done by Jo [10], which included 1002 cases in the age group between 12-91 years, the prevalence of premature gray hair was 51.5%.

Sudden onset of graying was reported in 28 (8%) of our patients and insidious onset in 343 (92%). The mean duration of premature canities in our patients was 2.16 +/- 3.45 years. In a similar study done by Daulatabad D [11] they reported a mean duration of 39.8 ± 37.3 months (range: 4 months–15 years) at the time of presentation.

Vertex (89%) was the most common initial site of premature graying of hair, followed by parietal and occipital areas. In contrast, a study done by Daulatabad D [11], found that frontal region was the first to be affected (48.1% cases), followed by the vertex in 34.6%, occiput in 13.5% and temporal region in 3.8%. In another study done by Jo [10] in Korean patients, the most common initial site was parietal followed by occipital region which is in contrast to our finding, possibly due to racial variation. Also this finding is in contrast to what is seen in physiological greying of hair where the initial site of onset is in the temporal area and side burns in men and scalp margins in women. This shows that premature canities is a separate entity in itself and not a part of chronological aging.

Vertex was the predominantly affected area in our study. However in a study done by Daulatabad the frontal and vertex areas were the most affected areas of the scalp.

Change of water/hard water (67%), was the most common aggravating factor in our study. In this part of Karnataka (Mandya district), hardness of ground water is due to higher calcium levels than the permissible limit [12]. Hardness of water can cause damage to the cuticle there by exposing the cortex. However there is no direct evidence suggesting hardness of water leading to premature graying of hair.

Emotional/psychosocial stress (22%) was the second most common aggravating factor in our study and only 4 % of patients gave a history of smoking. Smoking leads to oxidative stress and so does psychosocial stress. Daulatabad D [11]. Evaluated oxidative stress parameters in the sera of patients with premature canities and found that patients with premature canities had a higher level of pro-oxidants and lower levels of antioxidants than controls. Studies by Jo and Mosley [10, 13], found a significant association between smoking and premature canities.

Positive family history was found in 50.4% of patients. In a similar study done by Bhat RM positive family history of premature graying was present in 42.6% of patients and siblings were involved in 14.2% of patients. A questionnaire – based study in men younger than 30 years of age by Shin H [14] also found a positive family history in patients with premature canities. This suggests that genetic factors play an important role in premature canities.

Fifty five percentage of our study group had mild, 23% had moderate, 16% had severe and 6% had very severe premature graying of hair. In a study done by Bhat RM to assess the severity of graying, they found that among 35 children, 11.5% had mild, 65.7% had moderate and 23% had severe graying of hair. The scale used by them was mild (up to 50 gray hairs), moderate (50‑100) and severe (more than 100 gray hairs).

Anemia was found in 11% of students with premature gray hairs. Among them, 8% had mild (Hb% >10-11mg/dl), 2% had moderate (>7-10mg/dl) and 1% had severe (>6-7mg/dl) anemia. Iron deficiency has been reported to cause pigmentation abnormality of scalp hair [15, 16]. It has been shown that iron affects melanogenesis by the rearrangement of dopachrome to 5, 6 – dihydroxyindoles and oxidative polymerization of 5, 6 – dihydroxyindoles to melanin pigments. Also, studies provided evidence for the role of iron in the modulation of the activity of tyrosinase. It is reported that in a tautomerization reaction by dopachrome tautomerase, which is one of the later stages of melanin biosynthesis, the isomerization of dopachrome to dihydroxyindole ‑2‑carboxylic acid DHICA occurs. This enzyme is a metalloenzyme with ferrous in at its active site [17].

CONCLUSION

To conclude, our study revealed that the prevalence of premature canities was 26% with a slight female preponderance. Genetic predisposition was found in half the number of patients with premature canities. Majority of them had insidious onset. Vertex was the most common initial site of onset and also the predominantly affected area of the scalp. Change of water / hard water was the most common aggravating factor followed by psychosocial stress and smoking. Anemia was present in 11% of our patients. Elaborate case-control studies with a large population are required to substantiate these facts.

LIMITATIONS OF OUR STUDY

There were a few limitations in our study. It was not a case control study, therefore we couldn't compare our findings with the control population and elaborate investigations like total iron binding capacity, serum iron, calcium, ferritin, vitamin D3 were not performed.

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Nil

CONFLICT OF INTEREST

No interest

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