Thyroid Profile in Premature Canities: A Study of 216 Patients

Sheikh Manzoor1, Samia Aleem2, Seema Qayoom3, Farah Sameem4, Suhail Raheem Rather4, Syed Shahab ud din Bukhari2

ABSTRACT

Introduction: Premature canities or premature graying of hair is an important cause of low self-esteem with significant adverse effects on the appearance and socio-cultural adjustment and acceptance, and thus is a common cause of referrals to the dermatology clinics. Despite extensive research being carried out, its pathogenesis is still poorly understood. Its association with certain organ specific autoimmune diseases including those of thyroid has been observed. The purpose of this study was to evaluate the role of thyroid gland in pathogenesis of premature canities.

Material and Methods: This study was a cross-sectional, observational, hospital based study conducted on patients of premature canities of age less than 25 years, over a period of eighteen months. Patients were evaluated fully using complete history and routine investigations including thyroid profile. Family history of thyroid disorders was also looked into.

Results: A total of 216 patients were enrolled, with the mean age of presentation being 17.2 ±2.3 years and that of onset being 15.4 ±1.8 years. Females predominated the study (1:1.45). Derangement of thyroid profile was observed in 70 (32.40%) of the cases themselves and first degree family members of 136 (62.96%) cases.

Conclusion: This study highlights the importance of periodic and regular thyroid profile screening in patients of premature canities. Whether, early detection and correction of its disorders can prevent progression of premature graying, needs further evaluation.

Keywords: Autoimmune, canities, premature Canities, thyroid stimulating hormone.

INTRODUCTION

Hair, an appendage of skin, has no vital function in humans, yet its psychological functions are extremely important.1 Hair graying or canities is a process of chronological ageing and occurs regardless of gender and race. However, the age of graying varies with race and ethnicity. Hair is said to gray prematurely (premature canities) only if graying occurs before the age of 20 years in whites, 25 years in Asians and 30 years in Africans.2 Pathogenesis of premature canities is poorly understood. It is mainly considered to be genetic, with interplay of various environmental factors.3,8 It may appear alone as an autosomal dominant condition or as part of various premature ageing syndromes and in association with certain organ specific autoimmune diseases including those of thyroid.3,9,10 The purpose of this study was to determine the role of thyroid in pathogenesis of premature canities.

MATERIAL AND METHODS

This study was a cross-sectional, observational, hospital based study conducted on patients of premature canities attending the OPD block of the department of dermatology, STD and leprosy of a tertiary care centre over a period of eighteen months from January 2014 to June 2015. After obtaining ethical clearance from the institutional review board and informed patient consent, all the patients with hair graying and age less than 25 years attending the OPD during the specified period were enrolled in the study. Patients above 25 years and those with premature aging syndromes were excluded from the study. Basic demographic information and complete history including age of onset, age of presentation, duration, family history and history of associated disorders was taken from each patient. All patients underwent general physical examination and mucocutaneous examination to rule out any other associated disorder. Patients were evaluated fully using all routine investigations. Two ml of venous blood was also collected at the time of presentation for evaluating thyroid profile. Patients immediate family members were also subjected to thyroid function test. Thyroid function test was done using semiquantitative assay based on chemiluminescence.

STATISTICAL ANALYSIS

Results were collected, tabulated and statistically analysed using statistical package SPSS version 19. Descriptive statistics were used to infer results.

RESULTS

Our study completed over a period of eighteen months involved 216 patients of premature canities. The youngest patient was 4 years old. The mean age of presentation was 17.2 ± 2.3 years and mean age of onset of premature graying was 15.4 ± 1.8 years. Females predominated the study with male/female ratio of 1:1.45 (88 males/ 128 females). Majority of the patients i.e 116 (53.70%) were of urban background as compared to 100 (46.29%) from rural background. Parental history of premature canities was present in 11 (5.09%) patients and siblings

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were involved in 4 (1.85%). (Table-1) 70 (32.40%) out of a total 216 patients had hypothyroid thyroid profile. Family history of altered thyroid hormone levels was present in a total of 136 (62.96%) patients, all of which were hypothyroid. Among them 100 (46.29%) patients had deranged thyroid hormone levels in their mothers. Sisters gave similar involvement in 20 (9.25%) patients and fathers and brothers in 8 (3.70%) and 12 (5.55%) patients respectively. 4 (1.85%) patients gave history of deranged thyroid function in their mothers as well as sisters. 44 (20.37%) patients had altered levels in themselves as well as in their mothers. (Table-2)

DISCUSSION

Our study involving patients of premature canities was a cross-sectional study completed over a period of eighteen months and involved 216 patients. The mean age of cases enrolled was 17.2 ± 2.3 years and mean age of onset was 15.4 ± 1.8 years which was comparable to a study done by Fatemi et al (17.8 ± 2 years and 15.5 ± 3.2 years) and Ramesh et al (16.8yrs and 15yrs) on school children with premature canities.

Male: female ratio in our study was 1: 1.45 which was more as compared to a study done by Ramesh et al who observed a M:F ratio of 1:1.1. Fatemi et al observed no sex difference.

Parental history of premature canities was present in 5.09% of patients as compared to 42.6% in a study by Ramesh et al and siblings were involved in 1.85% as compared to 14.2% in a study by Ramesh et al.

70 (32.40%) out of a total 216 patients had deranged thyroid profile but family history of deranged thyroid hormone levels was present in 140 (64.81%) patients.

We in our study observed increased prevalence of premature canities in females. Also involvement of thyroid was more in female relatives as compared to male ones. This can be explained by increased prevalence of both autoimmune and thyroid diseases in females.

This study highlights the role of thyroid gland in pathogenesis of premature canities. This could represent a deleterious effect of autoimmunity or altered levels of thyroid hormone, on the melanocytes of hair follicles.

Association of premature canities with autoimmune diseases has been observed by Dawber et al also. He noted premature and early graying of hair to be a significant integumentary association of pernicious anaemia. 55% of patients with pernicious anaemia were found to develop graying of hair before 50 years as compared to only 30% in the control group.

Leary et al, in a study also observed association of premature canities with disorders of thyroid. He noted higher percentage (36%) of patients with Graves' disease were affected by premature graying than control patients (25%); this finding however did not reach statistical significance (\(P=0.14\)). Higher incidence of deranged thyroid profile in first degree relatives as compared to cases themselves could represent inherited but hidden thyroid anomalies which will be diagnosed sooner or later. Researches have also shown genetic-environmental networks and mechanisms underlying both thyroid abnormalities and premature canities, further explaining variable age of manifestation of thyroid abnormalities in them. Therefore patients complaining of premature graying should be screened for thyroid dysfunction as a rule and at regular intervals. On the other hand, studies have shown that the presence of premature graying in patients with known thyroid disorder, may warrant special consideration of risk factors for osteoporosis, necessitating a lower threshold for bone mineral density screening. The limitation of our study was lack of controls in the study and high prevalence of hypothyroidism in our population.

### Table-1: Demographic profile

<table>
<thead>
<tr>
<th>Relation</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Self</td>
<td>70</td>
<td>32.40%</td>
</tr>
<tr>
<td>Mother</td>
<td>100</td>
<td>46.29%</td>
</tr>
<tr>
<td>Father</td>
<td>8</td>
<td>3.70%</td>
</tr>
<tr>
<td>Sister</td>
<td>20</td>
<td>9.25%</td>
</tr>
<tr>
<td>Brother</td>
<td>12</td>
<td>5.55%</td>
</tr>
<tr>
<td>Total first degree family</td>
<td>136</td>
<td>62.96%</td>
</tr>
</tbody>
</table>

### Table-2: Involvement of thyroid gland in patient and his relations

<table>
<thead>
<tr>
<th>Involvement of thyroid gland</th>
<th>Total patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thyroid profile in cases</td>
<td>216</td>
</tr>
<tr>
<td>Mean age ±SD*</td>
<td>17.2±2.3 years</td>
</tr>
<tr>
<td>Range</td>
<td>4-25 years</td>
</tr>
<tr>
<td>Male/Female</td>
<td>1: 1.45</td>
</tr>
<tr>
<td>Rural/Urban</td>
<td>1: 1.16</td>
</tr>
<tr>
<td>Family History</td>
<td>15 (6.94%)</td>
</tr>
</tbody>
</table>

### Figure-1: Thyroid profile in cases

![Thyroid profile in cases](image1)

### Figure-2: Thyroid profile in first degree family

![Thyroid profile in first degree family](image2)
from which the study group was derived. Anti-thyroid peroxidase (anti-TPO) if also done could authenticate role of autoimmunity as well as our findings.

CONCLUSION

Our study highlights the importance of screening for thyroid dysfunction in premature canities. Detection and thus replacement or correction of these disorders at an early age may prevent further progression of the disease. Further studies on a larger scale involving control group also, are required to confirm the findings noted.

REFERENCES