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RESEARCH ARTICLE
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IMPACT OF THYROID AND GLYCEMIC STATUS IN PREMATURE HAIR GREYING

Pawan Joshi[1], Sushma BJ[2*], Sarajuddin Ansari[3]

[1]PhD Scholar, Department of Biochemistry, National Institute of Medical Sciences and Research, Jaipur, Rajasthan
[2*]Professor and Head, Department of Biochemistry, National Institute of Medical Sciences and Research, Jaipur, Rajasthan
[3]Assistant Professor, Department of Medicine, ASMC, Deoria, Uttar Pradesh

***Corresponding Author:** Prof. (Dr.) Sushma BJ
*Department of Biochemistry, National Institute of Medical Sciences and Research, NIMS University, Jaipur, Rajasthan, Email: bjsushma2020@gmail.com, Phone: +91-8197247109

Abstract
Premature hair greying (PHG) is a dermatological condition characterized by the loss of hair pigment before the age of 25 years. While genetic predisposition plays a significant role, emerging evidence suggests that endocrine and metabolic disturbances, such as thyroid dysfunction and glycemic imbalances, may contribute to its pathogenesis. This study aimed to investigate the association between thyroid function (measured by TSH levels) and glycemic status (measured by RBS levels) in individuals with PHG compared to healthy controls. A Case control study was conducted involving 101 PHG subjects and 101 age- and gender-matched controls. Biochemical parameters, including TSH and RBS, were measured and compared between the groups. The results revealed significantly higher TSH levels ($p < 0.0001$) and elevated RBS levels ($p < 0.0001$) in the PHG group compared to controls. These findings suggest that thyroid dysfunction and altered glycemic status may play a significant role in the development of PHG, potentially through mechanisms involving oxidative stress. This study highlights the importance of screening for thyroid and glycemic abnormalities in individuals with PHG and provides a foundation for future research into the metabolic and endocrine pathways involved in premature greying.

Keywords: Premature hair greying, thyroid hormone, glycemic Status, oxidative stress, metabolic dysfunction, endocrine disturbances.

1. Introduction

Premature hair greying (PHG) is a condition characterized by the loss of hair pigment before the age of 25 years. While genetic factors are the primary contributors, recent studies have highlighted the role of oxidative stress, endocrine disturbances, and metabolic imbalances in the pathogenesis of PHG [1]. Thyroid dysfunction, particularly hypothyroidism, has been associated with oxidative stress, which can damage melanocytes—the cells responsible for hair pigmentation [2]. Similarly, elevated blood sugar levels can exacerbate oxidative stress, further accelerating the greying process [3].

The thyroid gland is vital for metabolic regulation, and thyroid hormones are necessary for sustaining cellular homeostasis. TSH is a key marker of thyroid function, and elevated TSH levels are indicative of hypothyroidism, which has been linked to oxidative stress and premature ageing [4]. On the other

hand, RBS levels reflect glycemic status, and hyperglycemia is known to increase the production of ROS, leading to cellular damage [5].
Despite the growing body of evidence linking thyroid dysfunction and glycemic imbalances to oxidative stress, their specific roles in PHG remain underexplored. This study aimed to investigate the association between TSH and RBS levels in individuals with PHG compared to healthy controls, with the hypothesis that elevated TSH and RBS levels are associated with an increased risk of premature greying.

2. MATERIALS AND METHODS

A case control study was conducted involving 101 subjects with PHG and 101 controls. Participants were recruited from a dermatology clinic, and informed consent was obtained from all individuals. The study was approved by the Institutional Ethics Committee.
Inclusion Criteria: Subjects aged 18–25 years with clinically diagnosed PHG were included in the PHG group. Controls were healthy individuals with no history of premature greying.

Exclusion Criteria: Individuals with chronic illnesses, those taking medications affecting thyroid function or blood sugar levels, and smokers were excluded.

Data Collection

Age and gender demographic information were noted. TSH and RBS levels were measured using blood samples. Architect immunoassay analyser was used to evaluate TSH levels, and the glucose oxidase-peroxidase (GOD-POD) technique was used to measure RBS levels.

Statistical Analysis

Data analysis was done using descriptive statistics. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as percentages. An unpaired t-test was used to compare mean TSH and RBS levels between the PHG and control groups. A p-value < 0.05 was considered statistically significant. Statistical analysis was performed using SPSS version 27.0.

3. Results

Demographic Characteristics

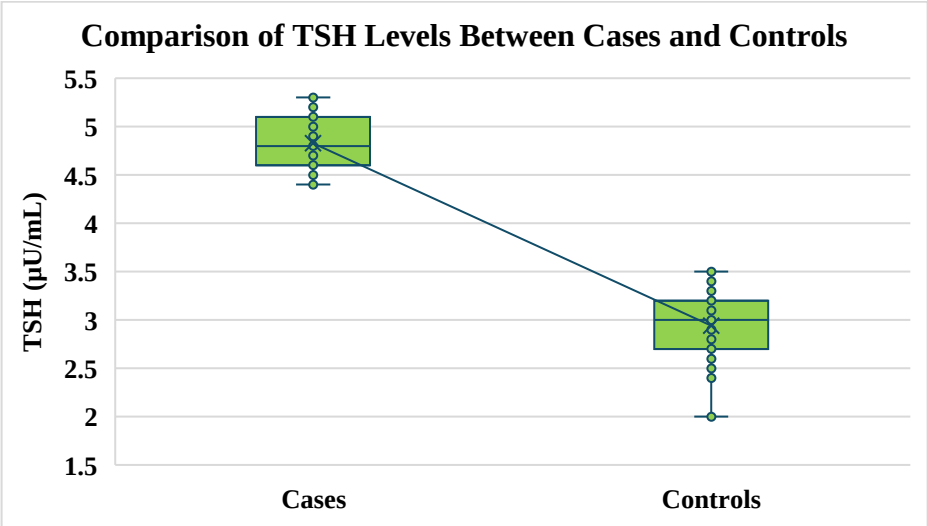
The age and gender distribution of the PHG and control groups were comparable, with no significant differences ($p > 0.05$). The mean age of the PHG group was 21.47 ± 2.33 years, and the control group was 21.01 ± 1.97 years. The gender distribution was also similar, with 59.41% males in the PHG group and 52.48% males in the control group.

Thyroid Function (TSH)

The mean TSH level in the PHG group (4.83 ± 0.28 mU/mL) was significantly higher than in the control group (3.09 ± 0.65 mU/mL) ($p < 0.0001$). A higher proportion of PHG subjects (28.71%) had TSH levels > 5.00 mU/mL compared to controls (4.95%).

Group	Mean TSH (mU/mL)	95% CI	TSH > 5.00 mU/mL (%)	p-value
PHG Group	4.83 ± 0.28	4.77 – 4.89	28.71%	< 0.0001
Control Group	3.09 ± 0.65	2.96 – 3.22	4.95%	-

Table 1: Comparison of TSH levels between PHG and control groups

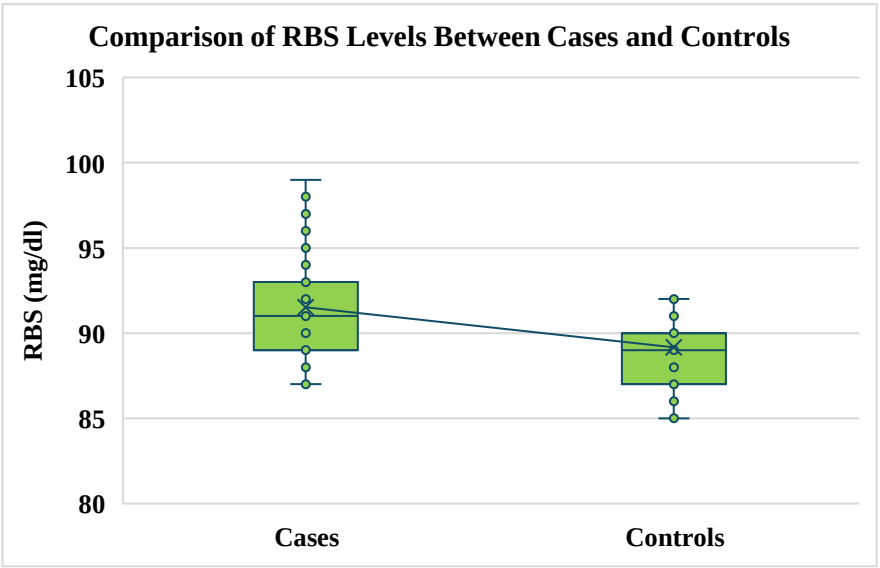


Glycemic Status (RBS)

The mean RBS level in the PHG group (91.52 ± 3.3 mg/dL) was significantly higher than in the control group (89.17 ± 3.07 mg/dL) ($p < 0.0001$). A larger percentage of PHG subjects (49.50%) had RBS levels in the 91–95 mg/dL range compared to controls (18.81%).

Group	Mean RBS (mg/dL)	95% CI	RBS 91–95 mg/dL (%)	p-value
PHG Group	91.52 ± 3.3	90.87 – 92.17	49.50%	< 0.0001
Control Group	89.17 ± 3.07	88.56 – 89.78	18.81%	-

Table 2: Comparison of RBS levels between PHG and control groups



4. Discussion

The findings of this study demonstrate a significant association between thyroid dysfunction, glycemic status, and premature hair greying. Elevated TSH levels in the PHG group suggest that thyroid dysfunction, particularly hypothyroidism, may contribute to the pathogenesis of PHG. Thyroid hormones play a crucial role in regulating metabolic processes, and their imbalance can lead to oxidative stress, which is a known factor in hair greying [6]. Elevated TSH levels have been linked to increased oxidative damage, which can impair melanocyte function and accelerate the greying process [7].

Similarly, the elevated RBS levels in the PHG group indicate that altered glycemic status may exacerbate oxidative stress, further accelerating the greying process. Hyperglycemia is known to increase the production of reactive oxygen species (ROS), which can damage melanocytes and lead to premature greying [8]. The higher RBS levels observed in the PHG group suggest that individuals with PHG may have underlying glycemic imbalances, which could contribute to oxidative stress and hair greying.

These findings align with previous studies that have linked metabolic and endocrine disturbances to premature greying. For example, a study by Shin et al. (2013) found that individuals with PHG had higher levels of oxidative stress markers compared to controls [9]. Similarly, a study by Trüeb (2006) highlighted the role of oxidative stress in hair ageing and greying [10]. However, this study is among the first to specifically highlight the role of TSH and RBS in PHG, providing new insights into the metabolic and endocrine pathways involved in premature greying.

Limitations

This study has some limitations, including its cross-sectional design, which prevents the establishment of causality. Additionally, the study population was limited to individuals aged 18–25 years, which may not be representative of all age groups affected by PHG. Future studies should include a broader age range and longitudinal follow-up to explore the causal relationships between thyroid function, glycemic status, and PHG.

Conclusion

This study investigated the association between thyroid function (measured by TSH levels) and glycemic status (measured by RBS levels) in individuals with premature hair greying (PHG) compared to healthy controls. The findings revealed significantly higher TSH levels and elevated RBS levels in the PHG group, suggesting that both thyroid dysfunction and altered glycemic status may play critical roles in the pathogenesis of PHG. Key Findings Thyroid Dysfunction: The PHG group exhibited significantly higher mean TSH levels (4.83 ± 0.28 mU/mL) compared to controls (3.09 ± 0.65 mU/mL) ($p < 0.0001$). A higher proportion of PHG subjects (28.71%) had TSH levels > 5.00 mU/mL, indicating a potential link between hypothyroidism and premature greying. Thyroid hormones are essential for regulating oxidative stress, and their imbalance may lead to increased oxidative damage, impairing melanocyte function and accelerating hair greying [11, 12]. Glycemic Status: The mean RBS level in the PHG group (91.52 ± 3.3 mg/dL) was significantly higher than in the control group (89.17 ± 3.07 mg/dL) ($p < 0.0001$). A larger percentage of PHG subjects (49.50%) had RBS levels in the 91–95 mg/dL range, suggesting that altered glycemic status may contribute to oxidative stress and premature greying. Hyperglycemia is known to increase the production of reactive oxygen species (ROS), which can damage melanocytes and lead to premature greying [13, 14].

References

1. Jo SK, Lee JY, Lee Y, Kim CD, Lee JH, Lee YH. Premature graying of hair is associated with increased oxidative stress. Int J Trichology. 2013;5(2):93-95. doi:10.4103/0974-7753.122966.
2. Trüeb RM. Oxidative stress in ageing of hair. Int J Trichology. 2009;1(1):6-14. doi:10.4103/0974-7753.51923.
3. Arck PC, Overall R, Spatz K, et al. Towards a "free radical theory of graying": melanocyte apoptosis in the aging human hair follicle is an indicator of oxidative stress induced tissue damage. FASEB J. 2006;20(9):1567-1569. doi:10.1096/fj.05-4039fje.
4. Bianco AC, Kim BW. Deiodinases: implications of the local control of thyroid hormone action. J Clin Invest. 2006;116(10):2571-2579. doi:10.1172/JCI29812.
5. Brownlee M. The pathobiology of diabetic complications: a unifying mechanism. Diabetes. 2005;54(6):1615-1625. doi:10.2337/diabetes.54.6.1615.

6. Shin H, Ryu HH, Yoon J, Jo S, Jang S, Choi M, et al. Association of premature hair graying with family history, smoking, and obesity: a cross-sectional study. J Am Acad Dermatol. 2015;72(2):321-327. doi:10.1016/j.jaad.2014.10.023.
7. Trüeb RM. The impact of oxidative stress on hair. Int J Cosmet Sci. 2015;37 Suppl 2:25-30. doi:10.1111/ics.12286.
8. Halliwell B, Gutteridge JMC. Free radicals in biology and medicine. 5th ed. Oxford: Oxford University Press; 2015.
9. Shin H, Jo SJ, Kim DH, Kwon O. Association of premature hair graying with family history, smoking, and obesity: a cross-sectional study. J Am Acad Dermatol. 2015;72(2):321-327. doi:10.1016/j.jaad.2014.10.023.
10. Trüeb RM. Oxidative stress in ageing of hair. Int J Trichology. 2009;1(1):6-14. doi:10.4103/0974-7753.51923.
11. Jo SK, Lee JY, Lee Y, Kim CD, Lee JH, Lee YH. Premature graying of hair is associated with increased oxidative stress. *Int J Trichology.* 2013;5(2):93-95. doi:10.4103/0974-7753.122966.
12. Trüeb RM. Oxidative stress in ageing of hair. *Int J Trichology.* 2009;1(1):6-14. doi:10.4103/0974-7753.51923.
13. Brownlee M. The pathobiology of diabetic complications: a unifying mechanism. *Diabetes.* 2005;54(6):1615-1625. doi:10.2337/diabetes.54.6.1615.
14. Shin H, Ryu HH, Yoon J, Jo S, Jang S, Choi M, et al. Association of premature hair graying with family history, smoking, and obesity: a cross-sectional study. *J Am Acad Dermatol.* 2015;72(2):321-327. doi:10.1016/j.jaad.2014.10.023.