(e) ISSN Online: 2321-9599

(p) ISSN Print: 2348-6805

ORIGINAL ARTICLE

# Assessment of relation of thyroid function with Alopecia areata

Apoorva Singh[1], Jayanti Singh[2]

[1]Assistant Professor, Department of Dermatology, Venereology And Leprosy, Saraswathi Institute of Medical Sciences, Hapur, U.P., India;
[2]Assistant Professor, Department of Physiology, Saraswathi Institute of Medical Sciences, Hapur, U.P., India

***ABSTRACT:***
**Background:** Alopecia areata is a common form of localized, non- scarring hair loss that occurs on any hair bearing skin. The present study was conducted to assess relation of thyroid function with AA. **Materials & Methods:** forty patients with AA without thyroid disorders were divided into 2 groups. Healthy subjects were selected as a control group. Venous blood samples were taken from all for measurement of thyroid stimulating hormone (TSH), free T3, freeT4. **Results:** Onset was sudden seen in12 in mild, 2 in moderate and 4 in severe, gradual in 6 in mild, 3 in moderate and 5 in severe, accidental in 2 in mild, 5 in moderate and 1 in severe. Course was stationery in 10 mild, 6 moderate and 1 severe, progressive in 2 mild, 1 moderate and 4 severe, intermittent in 5 mild, 2 moderate and 3 severe, recurrent in 3 mild, 1 moderate and 2 severe, site was scalp in 4, 2 and 3, chin in 6, 1 and 2, scalp+ chin in 5, 1 and 3 and all body in 5, 6 and 2 in mild, moderate and severe cases respectively. The mean TSH (ug/dl) was 3.46 and 2.84, free T3 (pg/dl) was 2.82 and 3.15 and free T4 (ng/dl) was 1.16 and 1.42 in group I and group II respectively. The difference was significant (P< 0.05). **Conclusion:** Patients with AA have thyroid abnormalities. So, patients with AA should be screened for thyroid functions.
**Key words:** Alopecia areata, thyroid abnormalities, T3.

**Corresponding author**: Dr. Jayanti Singh, Assistant Professor, Department of Physiology, Saraswathi Institute of Medical Sciences, Hapur, U.P., India

**This article may be cited as**: Singh A, Singh J. Assessment of relation of thyroid function with Alopecia areata. J Adv Med Dent Scie Res 2015;3(1):250-253.

## INTRODUCTION

Alopecia areata is a common form of localized, non-scarring hair loss that occurs on any hair bearing skin. The etiopathogenesis of the disease is still unclear. It is suspected to be an autoimmune disease, having a genetic predisposition and being influenced by environmental and ethnic factors.[1]

AA is an autoimmune condition that attacks the hair follicles, causing nonscarring hair loss. Population studies from the Rochester Epidemiology Project estimate a lifetime incidence of AA of 2.1%, in a population in Olmsted County, Minnesota, with no difference in incidence between genders. A systemic review of the epidemiology of AA indicated a similar worldwide lifetime incidence of around 2%. Some smaller studies indicate a slight female-to-male gender bias, but this may be due to higher female concern regarding hair loss and subsequent treatment.[2]

AA typically presents as smooth, sharply demarcated, round patches of hair loss without atrophy with "exclamation point hairs" observed on the periphery of the patches.[3] Special designations of the disease include alopecia universalis (AU) (total body hair loss), alopecia totalis (AT) (total scalp hair loss, or alopecia in an ophiasis pattern (band-like hair loss on the temporal and occipital scalp). Less common variants include the diffuse variant with widespread thinning of hair across the scalp or the reticular pattern with recurrent hair loss in one area and spontaneous hair regrowth in another. Ophiasis inversus causes band-like hair loss in the frontoparietotemporal area.[4]

Thyroid disorders and vitiligo have the strongest association with AA. Thyroid disorders that may be associated with AA include hypothyroidism, Hashimoto's thyroiditis, Graves' disease and simple goiter. Among these, hypothyroidism was the most frequent association.[5] The present study was conducted to assess relation of thyroid function with AA.

## MATERIALS & METHODS

The present study comprised of forty patients with AA without thyroid disorders of both genders. All were informed regarding the study and their written consent was obtained.

Data such as name, age, gender etc. was recorded. They were divided into 2 groups. Healthy subjects were selected as a control group. A thorough history, complete general and dermatological examination was performed. Venous blood samples were taken from all for measurement of thyroid stimulating hormone (TSH), free T3, freeT4 and detection of Anti-thyroglobulin Antibody (Tg-Ab) and Anti-thyroid Peroxidase Antibody (TPO-Ab). Results thus obtained were statistically analyzed. P value less than 0.05 was considered significant.

## RESULTS

**Table I Distribution of subjects**

| Groups | Group I | Group II |
|---|---|---|
| Status | AA | Control |
| M:F | 28:12 | 24:16 |

Table I shows that group I had 28 males and 12 females and group II had 24 males and 16 females.

**Table II Assessment of parameters in study group**

| Parameters | Variables | Mild (20) | Moderate (10) | Severe (10) | P value |
|---|---|---|---|---|---|
| **Onset** | Sudden | 12 | 2 | 4 | 0.02 |
| | Gradual | 6 | 3 | 5 | |
| | Accidental | 2 | 5 | 1 | |
| **Course** | Stationery | 10 | 6 | 1 | 0.01 |
| | Progressive | 2 | 1 | 4 | |
| | Intermittent | 5 | 2 | 3 | |
| | Recurrent | 3 | 1 | 2 | |
| **Site** | Scalp | 4 | 2 | 3 | 0.05 |
| | Chin | 6 | 1 | 2 | |
| | Scalp+ Chin | 5 | 1 | 3 | |
| | All body | 5 | 6 | 2 | |

Table II, graph I shows that onset was sudden seen in12 in mild, 2 in moderate and 4 in severe, gradual in 6 in mild, 3 in moderate and 5 in severe, accidental in 2 in mild, 5 in moderate and 1 in severe. Course was stationery in 10 mild, 6 moderate and 1 severe, progressive in 2 mild, 1 moderate and 4 severe, intermittent in 5 mild, 2 moderate and 3 severe, recurrent in 3 mild, 1 moderate and 2 severe, site was scalp in 4, 2 and 3, chin in 6, 1 and 2, scalp+ chin in 5, 1 and 3 and all body in 5, 6 and 2 in mild, moderate and severe cases respectively. The difference was significant (P< 0.05).

**Graph I Assessment of parameters in study group**

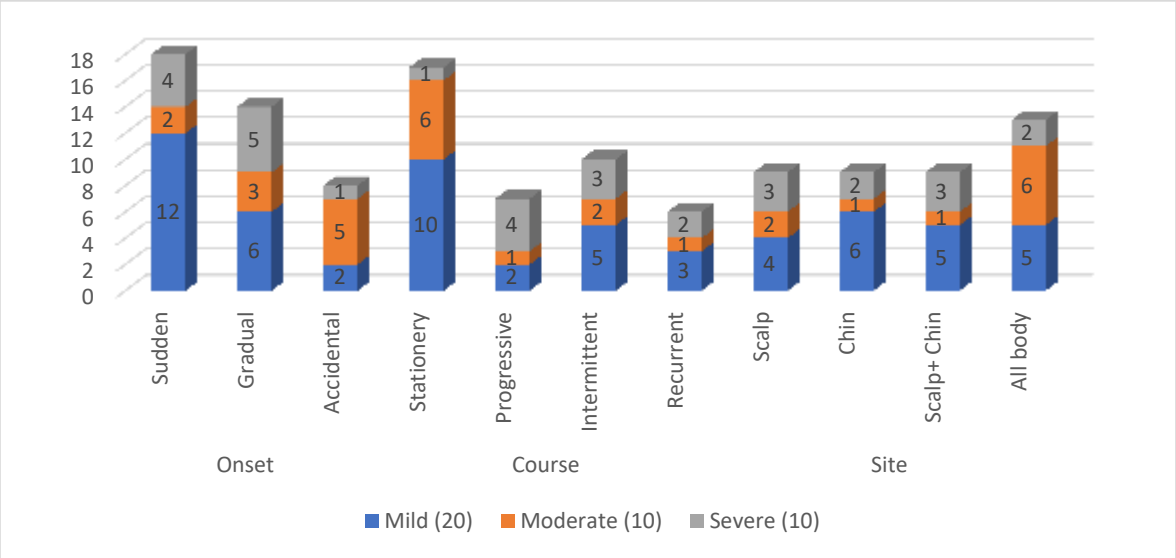
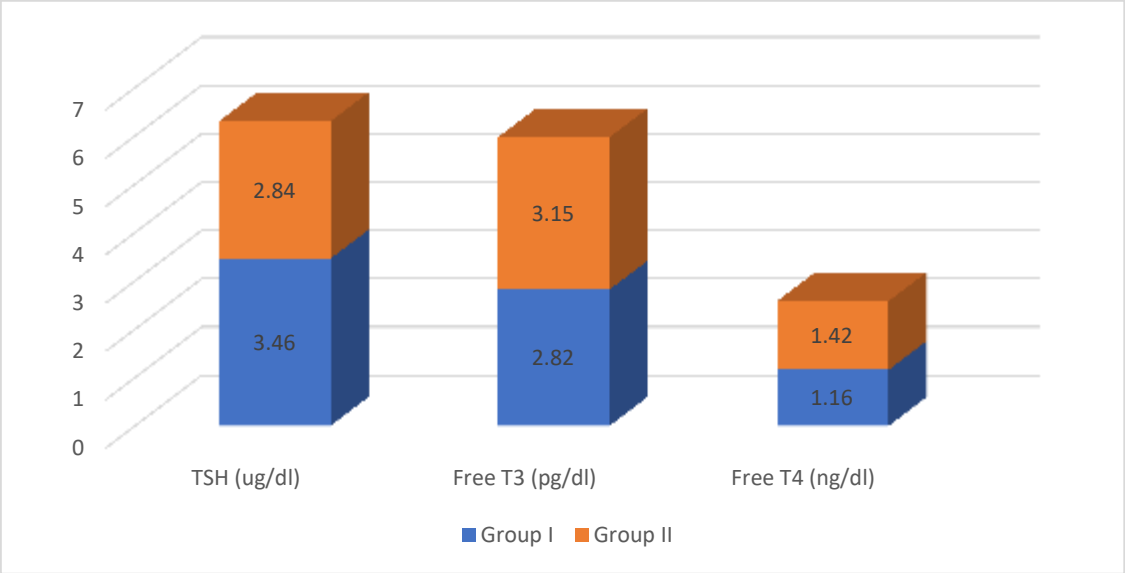



**Table III Comparison of thyroid function on both groups**

| Groups | Group I | Group II | P value |
|---|---|---|---|
| TSH (ug/dl) | 3.46 | 2.84 | 0.04 |
| Free T3 (pg/dl) | 2.82 | 3.15 | 0.02 |
| Free T4 (ng/dl) | 1.16 | 1.42 | 0.05 |

Table III, graph II shows that mean TSH (ug/dl) was 3.46 and 2.84, free T3 (pg/dl) was 2.82 and 3.15 and free T4 (ng/dl) was 1.16 and 1.42 in group I and group II respectively. The difference was significant (P< 0.05).

**Graph II Comparison of thyroid function on both groups**




## DISCUSSION

The exact pathophysiology of the disease is currently unknown. However, evidence suggests that AA is caused by an autoimmune reaction to the hair follicles due to both genetic and environmental factors.[6] Observational studies show a high correlation (10%–42%) between AA and family history.[7] Genome-wide association studies have identified numerous single-nucleotide polymorphisms (SNPs) associated with AA.[8] In a recent meta-analysis, human leukocyte antigen-DR (HLA-DR) on chromosome 6 appears to be the largest risk factor for AA.[9] These HLA class II genes are highly linked to CD4+ and CD8+ T-cells, which are important effector cells in AA. In addition, this study implicated BCL2-like protein 11, also known as BIM, which helps to regulate autophagy in the disease pathogenesis. Environmental factors likely exacerbate or induce AA. Stress is an often-cited cause of AA, but the literature from human studies is inconclusive.[10] The present study was conducted to assess relation of thyroid function with AA.

In present study, group I had 28 males and 12 females and group II had 24 males and 16 females. Bakry et al[11] in their study fifty subjects with AA (37 males and 13 females) without clinical evidence of thyroid disorders were selected and were divided into 3 groups according to severity of AA. Fifty age and sex-matched healthy volunteers (35 males and 15 females) were selected as a control group. Every case and control were subjected to history taking, complete general and dermatological examination. Venous blood samples were taken from cases and controls after taking their consents for measurement of thyroid stimulating hormone (TSH), free T3, freeT4 and detection of Anti-thyroglobulin Antibody (Tg-Ab) and Anti-thyroid Peroxidase Antibody (TPO-Ab). Subclinical hypothyroidism was detected in 16% of cases. There were statistically significant differences between cases and controls regarding levels of TSH, free T3 and free T4. There were significant differences between cases and controls regarding the presence of Tg-Ab and TPO-Ab.

We found that onset was sudden seen in12 in mild, 2 in moderate and 4 in severe, gradual in 6 in mild, 3 in moderate and 5 in severe, accidental in 2 in mild, 5 in moderate and 1 in severe. Course was stationery in 10 mild, 6 moderate and 1 severe, progressive in 2 mild, 1 moderate and 4 severe, intermittent in 5 mild, 2 moderate and 3 severe, recurrent in 3 mild, 1 moderate and 2 severe, site was scalp in 4, 2 and 3, chin in 6, 1 and 2, scalp+ chin in 5, 1 and 3 and all body in 5, 6 and 2 in mild, moderate and severe cases respectively. Kurtiv and Lliev[12] assumed that, the histologic studies have shown CD3+HLA-DR+lymphocytic infiltration in the epithelial cells of hair follicles, epidermal keratinocytes, and thyrocytes. These activated T lymphocytes play a role in the pathogenesis of AA and autoimmune thyroid dysfunction, as the aberrant HLA-DR antigen statement on the surface of the epithelial cells is realized through HLA-DR T lymphocytes. The increased number of activated T lymphocytes in the peripheral blood of patients with AA and autoimmune thyroid dysfunction, points not only to the participation of these lymphocytes in the pathogenesis of these diseases, but also to their inter-relationship.

## CONCLUSION

Authors found that patients with AA have thyroid abnormalities. So, patients with AA should be screened for thyroid functions.

## REFERENCES

1. Gilhar A, Ullmann Y, Berkutzki T, Assay B, Kalish RS. Autoimmune hair loss (alopecia areata) transferred by T lymphocytes to human scalp explants on SCID mice. J Clin Invest 1998;101:62-7.

2. Dilas LT, Icin T, Paro JN, Bajkin I. Autoimmune thyroid disease and other non-endocrine autoimmune diseases. Med Pregl 2011;64:183-7.
3. McElwee KJ, Boggess D, Olivry T, Oliver RF, Whiting D, Tobin DJ, et al. Comparison of alopecia areata in human and non human mammalian species. J Immunopathol Mol Cell Biol 1998;66:90-107.
4. Petukhova L, Cabral RM, Mackay-Wiggan J, Clynes R, Christiano AM. The genetics of alopecia areata. What's new and how it will help our Patients. Dermatol Ther 2011;24:326-36.
5. Wasserman D, Guzman-Sanchez DA, Scott K, McMichael A. Alopecia areata review. Int J Dermatol 2007;46:121-31.
6. Puavilai S, Puavilai G, Charuwichitratana S, Sakuntabhai A, Sriprachya-Anunt S. Prevalence of thyroid diseases in patients with alopecia areata. Int J Dermatol 1994;33:632-3.
7. Kavak A, Baykal C, Ozarmağan G, Akar U. HLA in alopecia areata. Int J Dermatol 2000;39:589-92.
8. Zulewski H, Müller B, Exer P, Miserez AR, Staub JJ. Estimation of tissue hypothyroidism by a new clinical score: Evaluation of patients with various grades of hypothyroidism and controls. J Clin Endocrinol Metab 1997;82:771-6.
9. Hay ID, Bayer MF. American Thyroid Association assessment of current free thyroid hormone and thyrotropin measurements and guidelines for future clinical assays. Clin Chem 1991;37:2002-8.
10. Freinkel RK. Hair growth and alopecia in hypothyroidism. Arch Dermatol 1972;106:349-52.
11. Bakry OA, Basha MA, El Shafiee MK, Shehata WA. Thyroid disorders associated with alopecia areata in Egyptian patients. Indian J Dermatol 2014;59:49-55.
12. Kurtev A, Ilev E. Thyroid autoimmunity in children and adolescents with alopecia areata. Int J Dermatol 2005;44:457-61.