

# Alopecia Areata and Alexithymia

Ayça CORDAN YAZICI, Ayşe BAŞTERZİ, Şenel TOT ACAR, Dilek ÜSTÜNŞOY, Güliz İKİZOĞLU,  
Deniz DEMİRŞEREN, Arzu KANIK

## SUMMARY

**Objective:** Whether or not psychological factors play an important role in the pathogenesis of alopecia areata (AA) is a controversial issue. AA has had a tendency to be associated with high avoidance in attachment relationships, high alexithymic characteristics, and poor social support. Some studies have suggested that personality characteristics might modulate individual susceptibility to AA. The role of stressful life events in the appearance of AA is uncertain. In addition to reports associating anxiety and affective disorders with the onset of AA, there have also been studies that have not confirmed such an association. This case-control study was undertaken with the aim of determining the significance of stressful life events and other psychological factors in the etiopathogenesis of AA.

**Method:** A total of 43 patients (26 male, 17 female) with AA and 53 age- and gender-matched healthy controls selected from hospital staff and their relatives (28 male, 25 female) were enrolled in the study. Both patients and controls were evaluated using the Hospital Anxiety and Depression Scale (HADS), Stress Scale, and Toronto Alexithymia Scale (TAS).

**Results:** There was no statistically significant difference between the patient and control groups with regard to the total scores of stressful major life events, depression, and anxiety ( $p > 0.05$ ). However, TAS scores in patients with AA were higher than in controls ( $p = 0.013$ ).

**Conclusion:** The present study found no evidence that stressful major life events, depression, or anxiety have a role in the etiopathogenesis of AA, but AA tended to be associated with alexithymia. It has been suggested that alexithymics may suffer from unnoticed chronic stress with physiological, endocrine, and immune consequences, and that alexithymia is associated with impaired immune response. We suggest that alexithymia may play a role in the pathogenesis of AA via stress-induced immunological mechanisms.

**Key Words:** Alopecia areata, stressful major life events, depression, anxiety, alexithymia

## INTRODUCTION

Alopecia areata (AA) is a clinical condition characterized by well circumscribed, round, or oval patches of hair loss on the scalp or other parts of the body. Sometimes, total alopecia, loss of all scalp hair, or alopecia universalis, loss of all body hair, may develop. It has no predominance based on gender, race, or age. Genetic factors, infections, psychological factors, autoimmune factors, and neuropeptides have been claimed to play a role in its etiopathogenesis (Arca and Kurumlu, 2003). In recent years, it has been considered as an organ-specific autoimmune disorder, which arises as a result of T lymphocyte orientation towards hair follicles (Randall, 2001). However, the autoantigen and triggering mechanisms are still unknown.

Onset of the disorder after a severe psychological stress is a common finding in AA cases (Garcia-Hernandez et al., 1999). There are also investigators who have detected emotional or mental traumas in only 4.8% of AA patients and report that stress is not a triggering factor (Mac Alpine, 1958), and this view is supported by many others (Van Der Steen et al., 1992; Paga et al., 1992; Russiello et al., 1995; Gupta et al., 1997; Picardi et al., 2003). As a opposing point of view, Liakopoulou et al. (1997) reported that in comparison to the control group, there was no increase in negative life events, but a decrease in positive life events was observed in AA children prior to onset of alopecia. Picardi and Abeni, (2001) concluded that the differences in various studies are because most of them were conducted without a control group, and the evaluation of stress was based on patient reports rather than standardized tests.

Results about the existence and frequency of psychiatric disorders associated with AA, such as anxiety and depression, are debatable (Colon et al., 1991; Koo et al., 1994; Çalikoğlu and Alpay, 2000; Güleç et al., 2002).

The relationship between alexithymia and AA has surfaced in recent years (Panconesi and Hautman, 1996; Picardi et al., 2003; Poot, 2004). Alexithymia is defined as difficulty being aware of, recognizing, differentiating, and defining emotions, both of self and others (Sifneos, 1988). It has been reported that alexithymic features are more frequent in some psychosomatic disorders (Guilbaud et al., 2003). A relation between alexithymia and some other disorders, such as irritable colon syndrome and fibromyalgia, has been observed in Turkey as well (Sayar et al., 2000, Güleç et al. 2004). Furthermore, it has been detected that depressed patients are in a more alexithymic structure (trait) and they have higher introverted rage and lower rage (healthy) controls (Güleç et al., 2005). This can result in a greater interiorization of stress, which may, in turn, alter immune responses related to neuropeptides, such as migration of macrophages, vasodilator or vasoconstrictor responses, phagocytosis, lymphocytic cellular immunity and expression of some factors of leukocytic adhesion to the microvascular endothelium, etc., thus helping us to understand the complex nature of the triggering factors involved in AA. (Ruiz-Doblado et al., 2003).

In this study, we aimed to detect the relationship of AA with stress-producing events, whether psychiatric symptom frequency is higher AA patients than in healthy controls, and AA's relationship to alexithymia.

## METHODS

The study included 43 patients (17 female, 39.5% and 26 male, 60.5%) who presented to the dermatology outpatient clinic with complaints of hair loss and were diagnosed with AA. The severity of hair loss was assessed by measuring the percentage of the alopecic area on the scalp with a modified version of the method suggested by Olsen et al. (1999). Patients were divided into 4 groups according to disease severity: S1-S2 (n=37, 86%): hair loss below 50%; S3-S4 (n=4, 9.3%): hair loss of 50-99%; S5-S6 (n=2, 4.7%): total scalp hair loss (total alopecia) or total body hair loss (alopecia universalis). Age, gender, AA type, duration of

the disorder, age at onset, and family history of the AA patients was recorded.

The control group included 53 healthy adults recruited from the hospital staff and their relatives (25 female (47.2%) and 28 male (52.8%)), who did not currently or previously have any psychiatric and dermatological disorders. The controls were age- and gender-matched, and did not work in an area related to psychiatry-psychology.

All of the patients and controls were evaluated by a psychiatrist using the Toronto Alexithymia Scale (TAS), Stress Scale, and Hospital Anxiety and Depression Scale (HADS). Persons who were illiterate, who demonstrated a lack of communication, and who had mental retardation were not included in the study. All the patients and controls gave informed consent to participate in the study.

A psychiatric interview was conducted with all the of patients and controls with the following scales:

**Toronto Alexithymia Scale (TAS) 26:** TAS is a 26-item self-report scale, in which questions (in Turkish) are responded to as true or false although the original form that was developed by Taylor et al. (1988) was a Likert-type scale. Its validity and reliability in Turkey were established by Dereboy (1990) and the scale cut-off point was assessed as 11 in this study.

**Hospital Anxiety and Depression Scale (HADS):** HADS is a self-report scale, which is used to determine the risk of anxiety and depression, and their level and severity of change. It is administered to patients with physical diseases who consult to primary care units. It consists of 14 questions, 7 of which measure anxiety, while the other 7 measure depression. The cut-off points for anxiety and depression are 11 and 8, respectively (Aydemir et al., 1997).

**Stress Scale:** The Stress Scale is a self-report scale that was developed by Holmes and Rahe (1967). It investigates a series of stressful life events in the preceding 6 months, such as the death of a spouse, divorce, menopause, and death of a close relative other than a spouse. It consists of 42 items and is used in cases of psychosocial stress and disorders, particularly in studies with psoriasis, AA, acne vulgaris patients.

**Statistical Analysis:** SPSS version 11.5 was used for statistical analysis. Statistical evaluations

**Table 1.** Clinical features of AA patients and the control group.

	Patient (n=43)	Control (n=53)	p
Gender (female/male)	17/26	25/28	p>0.05
Age (years) (mean±SSmean±SD*)	33.80±10.02	30.80±11.70	p>0.05
Duration of illness (mean ±SS*)	54.83±101.37		

\*SD: Standard Deviation

performed were student t test, chi-square test, linear regression analysis, and binary logistic regression analysis.

## RESULTS

The average age of the patients was 33.80±10.02 years. and 41 of them were diagnosed with AA, while 2 were diagnosed with alopecia totalis. The average age at alopecia onset was 29.31±10.95 years. The average age of the controls was 30.80±11.70 years. There was no difference between the patients and controls in terms of age and gender (p>0.05) (Table 1). Duration of the disorder was between 1 week and 30 years, with an average of 54.83±101.37 months. Family history of AA was present in 3 patients (7%).

When the combined effects of age, gender, stress, alexithymia, and significant or insignificant anxiety and depression scores in AA manifestation were assessed by two-variable regression analysis, it was found that the patients with higher TAS scores had a 3.6x higher risk for having the disorder compared with those who had lower scores (95% confidence interval: 1.313-10.102). Other variables had no significant effects on AA.

Nineteen (44.2%) of the AA patients and 13 (23.2%) of the controls had TAS scores higher than 11. When the two groups were compared with chi-square test, the result was significant (p=0.013). The effect of variables such as age, gender, family history, AA type, disorder duration, stress, anxiety, and depression on TAS was statistically insignificant (p> 0.05).

When the patients and controls were compared in terms of anxiety and depression scores, anxiety scores in HADS were higher than 11 in 10 (23.8%)

of the patients and 7 (13%) of the controls. No difference was detected between the patients and controls in terms of anxiety when compared with chi-square test (p=0.167). Depression scores in HADS were higher than 8 in 13 (31%) of the patients and 11 (20.4%) of the controls. When the patients and controls were compared with chi-square test, there was no difference in terms of depression as well (p=0.235). Regarding the Stress Scale, no statistically significant difference between the patients and controls was found (p=0.115).

Mean and standard deviation values for TAS, Stress Scale, HADS scores are shown in Table. 2.

## DISCUSSION

For years it has been asserted by many researchers that stressful life events cause the onset of AA, and AA patients had more stressful life events than control group subjects during the preceding 6 months (Muller and Winkelmann, 1963; Gip et al., 1969; Griesemer, 1978; De Weert et al., 1984; Perini et al., 1984; Lyketsos et al., 1985; Invernizzi et al., 1987). However, in most of the studies, no relationship was shown between the level of stress and the AA duration and prevalence (Muller and Winkelmann, 1963; Van Der Steen et al., 1992; Gupta et al., 1997).

There are researchers who concluded that stress is not a triggering factor (Mac Alpine, 1958; Van Der Steen et al., 1992; Paga et al., 1992; Russiello et al., 1995; Gupta et al., 1997; Picardi et al., 2003). Also in Turkey, Güleç et al. (2002) reported that there was no difference between the AA patient and control groups with regard to stressful life events. However, when they assessed the results of Short Form of Quality Life-36 (SF-36), one of the

**Table 2.** Results of Hospital Anxiety and Depression Scale (HADS), Stress Scale, and Toronto Alexithymia Scale (TAS) for the patient and control groups.

	Patient group mean $\pm$ SD* (n=43)	Control group mean $\pm$ SD* (n=53)	p
HDS*	6.55 $\pm$ 3.85	5.19 $\pm$ 3.79	p>0.05
HAS*	7.55 $\pm$ 4.24	6.91 $\pm$ 3.39	p>0.05
Stress Scale	111.74 $\pm$ 84.83	80.89 $\pm$ 78.55	p>0.05
TAS	10.02 $\pm$ 3.94	7.52 $\pm$ 3.85	p=0.013

\*SD: standard deviation, HDS: hospital depression scale, HAS: hospital anxiety scale

commonly used scales to measure quality of life, which is specific to general health, quality of life was worse in AA patients than the control group, in terms of vitality and psychological health. Furthermore, Köse et al. (2000) reported that they did not find any difference between the pre- and post-treatment psychiatric measurements (Beck Depression And Hopelessness Scales, State-Trait Anxiety Inventory, Short Symptom Inventory, and Toronto Alexithymia Scale) in AA patients. In the present study, we also did not encounter any finding implying stress was the initiator of AA.

Due to the fact that stress directly (via neuro-endocrine changes) or indirectly (a person may eat and sleep less, or may use alcohol) affects the immune system, relationships between stressful life events and immunological T cell disorders, along with consequent immune deficiency, have been investigated (Al'Abadie et al., 1994; Panconesi and Hautmann, 1996). Katsarou-Katsari et al. (2001) reported that acute emotional stress might trigger AA by activating excessive expression of  $2\beta$  corticotropin-releasing hormone receptors around hair follicles. Increased expressions of neurotransmitter substance P and the neuropeptide-destroying enzyme, neutral endopeptidase, have also been found around hair follicles over the alopecic area. Furthermore, decrease in the calcitonin gene-related protein has been detected, particularly in the serum of patients with alopecia totalis. Thus, the influences of stress on the immune system are thought to be set in motion by the release of neuropeptides from the systemic or follicular nerves (Randall 2001).

It has been claimed that the incidence of psychiatric disorders such as anxiety, depression,

neurotic disorders, phobias, and schizophrenia is high in patients with AA (Greenberg, 1955; Colon et al., 1991; Koo et al., 1994; Garcia Hernandez et al., 1999; Ruiz-Doblado et al., 2003). Gupta et al. (1997) reported high correlation between high stress scores and depression, and that stress during depressive clinical conditions may trigger AA. On the contrary, there are also studies, which have reported no significant correlations between AA and neurotic symptoms (Cipriani et al., 1983). In their study of 12 AA patients, Çalıkoğlu and Alpay (2000) found no significant difference between the patient and control groups when they compared the scores of the Beck Depression, and State and Trait Anxiety Inventories. Güleç et al. (2002) also reported no difference between the patient and control groups with regard to anxiety and depression levels. We did not observe any statistically significant difference between the patients and controls in terms of HADS scores as well.

In recent years, researchers have focused on the relationship between AA and alexithymia (Panconesi and Hautman, 1996; Picardi et al., 2003; Poot, 2004). Alexithymia is defined as a deficit in the awareness of and identification of emotional states. In interpersonal relationships, where the emotions are of central importance, alexithymics create the impression of beings strangers from another world. In daily life, they can think and communicate; however, they have problems making connections between their emotions and feelings, as well as identifying them and communicating them to others. They can be intelligent, though they use their intelligence to avoid their feelings (Sifneos, 1988). Poot (2004) interpreted the cause of alexithymia as the duty, which was taken over to avoid from the discussions within the family and suggested that

family therapy would be helpful. In a recent study by Picardi et al. (2003), it was found that AA tended to be associated with high avoidance in attachment relationships, high alexithymic characteristics and poor social support. Furthermore, it was shown that most AA patients who did not respond to therapy were alexithymic (Panconesi and Hautmann, 1996). It has been proposed that alexithymics are exposed to chronic stress, which they do not perceive and which leads to negative psychological and endocrinological consequences. It has also been reported that alexithymia causes disorders in immune functions (Guilbaud et al., 2003). In the present study, AA patients had higher scores on the TAS than did the control group. However, we did not discern any relationship between TAS scores and AA duration, age at onset, family history, AA type, and HADS and Stress Scale scores. In other words, although alopecia patients had higher

alexithymia scores than the control group, both groups had experienced the same levels of stressful life events.

Results of this study do not support the idea that stressful life events, and psychopathologies such as depression, or anxiety play a role in the pathogenesis of AA. However, our findings confirm that most of AA patients are alexithymic. Thus, we suggest that alexithymia makes coping with stress difficult and leads to AA via immunological mechanisms, which are triggered by stress. We think that it will be worthy to investigate the relationship between alexithymia and the immune system. As AA patients are frequently referred to psychiatrists by their physicians, we postulate that it would be helpful to evaluate alexithymia and treat it with appropriate psychotherapeutic interventions (Teshima et al., 1991).

#### REFERENCES

Al'Abadie MS, Kent GG, Gawkrödger DJ (1994) The relationship between stress and the onset and exacerbation of psoriasis and other skin conditions. *Br J Dermatol*, 130: 199-203.

Arca E, Kurumlu Z (2003) Etiopathogenesis, clinical features, and diagnosis in Alopecia areata. *Dermatose*, 2: 83-89.

Aydemir Ö, Güvenir T, Küey L et al. (1997) Validity and reliability of the Turkish version of Hospital Anxiety and Depression Scale. *Turkish Journal of Psychiatry*, 8:280-287.

Cipriani R, Veller Fomasa C, Peserico A (1983) Symptom questionnaire and alopecia areata. *G Ital Dermatol Venereol* 118: 281-282.

Colon EA, Popkin MK, Calliess AL et al. (1991) Lifetime prevalence of psychiatric disorders in patients with alopecia areata. *Comp Psychiatry*, 32: 245-251.

Çalıkoğlu E, Alpay FB (2000) The Evaluation Of The Beck Depression And State-trait Anxiety Inventories In Universal Pruritus, Alopecia Areata, Psoriasis Vulgaris And Chronic Urticaria. *Türkiye Klinikleri Journal of Dermatology*, 10: 229-232.

De Weert J, Temmerman L, Kint A (1984) Alopecia areata: a clinical study. *Dermatologica*, 168: 224-229.

Dereboy İF (1990) A preliminary study on the psychometric aspects of alexithymia self-report scales (Specialization Thesis). Ankara, Hacettepe University Medical School, Psichiatri Department.

Garcia-Hernandez MJ, Ruiz-Doblado S, Rodriguez-Pichardo A et al. (1999) Alopecia areata, stress and psychiatric disorders: a review. *J Dermatol*, 26: 625-632.

Gip L, Lodin A, Molin L (1969) Alopecia areata. A follow-up investigation of outpatient material. *Acta Derm Venereol (Stockh)*, 49: 180-188.

Greenberg SI (1955) Alopecia Areata: a psychiatric survey. *Arch Dermatol*, 72: 454-457.

Griesemer RD (1978) Emotionally triggered disease in a dermatologic practice. *Psychiatr Ann*, 8: 407-412.

Guilbaud O, Corcos M, Hjalmarsson L et al. (2003) Is there a psychoneuroimmunological pathway between alexithymia and

immunity? Immune and physiological correlates of alexithymia. *Biomedicine & Pharmacotherapy*, 57: 292-295.

Gupta MA, Gupta AK, Wateel GN (1997) Stress and alopecia areata: a psychodermatologic study. *Acta Derm Venereol (Stockh)*, 77: 296-298.

Güleç AT, Taşkıntuna N, Duru Ç et al. (2002) The role of psychological factors in alopecia areata and the effect of the disease on quality of life. *TÜRKDERM*, 36: 178-181.

Güleç H, Sayar K, Topbaş M et al. (2004) Alexithymia and Anger in Women With Fibromyalgia Syndrome. *Turkish Journal of Psychiatry*, 15: 191-198.

Güleç H, Sayar K, Ozkorumak H (2005) Somatic Symptoms of Depression. *Turkish Journal of Psychiatry*, 16: 90-96.

Holmes TH, Rahe RH (1967) The Social Readjustment Rating Scale. *J Psychosom Res*, 11: 213-218.

Invernizzi G, Gala C, Russo R et al. (1987) Life events and personality factors in patients with alopecia areata. *Med Sci Res*, 15: 1219-1220.

Katsarou-Katsari A, Singh LK, Theoharides TC (2001) Alopecia areata and affected skin CRH receptor upregulation induced by acute emotional stress. *Dermatology*, 203: 157-161.

Koo JY, Shellow WV, Hallman CP et al. (1994) Alopecia areata and increased prevalence of psychiatric disorders. *Int J Dermatol*, 33: 849-850.

Köse O, Sayar K, Ebrinç S (2000) Psychometric assesment of alopecia areata patients before and after dermatological treatment. *Bulletin of Clinical Psychopharmacology*, 10: 21-25.

Liakopoulou M, Alifieraki T, Katideniou A et al. (1997) Children with alopecia areata: psychiatric symptomatology and life events. *J Am Acad Child Adolesc Psychiatry*, 36: 678-684.

Lyketsos GC, Stratigos GC, Tawil G et al. (1985) Hostile personality characteristics, dysthymic states and neurotic symptoms in urticaria, psoriasis and alopecia. *Psychother Psychosom*, 44: 122-131.

MacAlpine I (1958) Is alopecia areata psychosomatic? A psychiatric study. *Br J Dermatol*, 70: 117-131.

- Muller SA, Winkelmann RK (1963) Alopecia areata. An evaluation of 736 patients. *Arch Dermatol*, 88: 106-113.
- Olsen E, Hordinsky M, McDonald-Hull S et al. (1999) Alopecia areata investigational assessment guidelines. *J Am Acad Dermatol*, 40: 242-246.
- Paga G, Faina P, Teodori A et al. (1992) Psychological factors in alopecia areata: results from a preliminary, controlled study. *New Trends Exp Clin Psychiatry*, 8: 73-79.
- Panconesi E, Hautmann G (1996) Psychophysiology of stress in dermatology. *Psychodermatology*, 14: 399-421.
- Perini GI, Fornasa CV, Cipriani R et al. (1984) Life events and alopecia areata. *Psychother Psychosom*, 41: 48-52.
- Picardi A, Abeni D (2001) Stressful life events and skin diseases: disentangling evidence from myth. *Psychother Psychosom*, 70: 118-136.
- Picardi A, Pasquini P, Cattaruzza MS et al. (2003) Psychosomatic factors in first-onset alopecia areata. *Psychosomatics*, 44: 374-381.
- Poot F (2004) Psychological consequences of chronic hair diseases. *Rev Med Brux*, 25: A286-288.
- Randall VA (2001) Is alopecia areata an autoimmune disease? *Lancet*, 358: 1922-1924.
- Ruiz-Doblado S, Carrizosa A, Garcia-Hernandez MJ (2003) Alopecia areata: psychiatric comorbidity and adjustment to illness. *Int J Dermatol*, 42: 434-437.
- Russello F, Arcierob G, Decaminada F et al. (1995) Stress, attachment and skin diseases: a case control study. *J Eur Acad Dermatol Venereol*, 5: 234-239.
- Sayar K, Solmaz M, Trablus S et al. (2000) Alexithymia in Irritable Bowel Syndrome. *Turkish Journal of Psychiatry*, 11: 190-197.
- Sifneos PE (1988) Alexithymia and its relationship to hemispheric specialization affect and creativity. *Psychiatric Clinics of North America*, 11: 287-293.
- Taylor GJ, Bagby RM, Ryan DP (1988) Criterion validity of the Toronto Alexithymia Scale. *Psychosom Med*, 50:500-509.
- Teshima H, Sogawa H, Mizobe K et al. (1991) Application of psychoimmunotherapy in patients with alopecia universalis. *Psychother Psychosom*, 56: 235-241.
- Van der Steen P, Boezeman J, Duller P et al. (1992) Can alopecia areata be triggered by emotional stress? *Acta Derm Venereol (Stockh)*, 72: 279-280.