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ORIGINAL ARTICLE

# Alopecia areata in Turkey: demographic and clinical features

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## Abstract

**Background** Alopecia areata is a complex genetic disease with still many unknown aspects, and many studies have been tried to find some clues about it.
**Objective** We aimed to investigate the demographic and clinical characteristics of alopecia areata in Turkish patients.
**Methods** Demographic data, localization, attack number in addition to some parameters such as disease duration, severity, age of onset, family history and ophiasis pattern were evaluated in 539 alopecia areata patients.
**Results** The male to female ratio was 1.6 : 1. Occipital and beard-moustache areas were mostly affected. Positive family history was noticed in 24.1% of the patients. The age of onset was earlier in women than in men ($P = 0.04$). Severe forms showed more persistent (≥ 1 year) disease duration ($P = 0.00$). Ophiasis was more common in severe, long duration (≥ 1 year) and early onset (≤ 18 years) disease ($P = 0.00$ for all parameters). Childhood alopecia areata (≤ 18 years) was also associated with long duration of the disease ($P = 0.016$) and positive family history ($P = 0.008$) when compared with adult onset (> 18 years) alopecia areata.

## Introduction

Alopecia areata (AA) is a common, non-scarring type of hair loss, affecting approximately 1.7% of the population.[1] This high prevalence is interesting for researchers due to its unknown aetiology, severe cosmetic disturbance, unpredictable course and problems finding effective treatments.

There are many well-designed genetic and epidemiologic studies in AA.[2–7] Our scope was to perform a comparative analysis on 539 AA patients' demographic and clinical features as well as family history. This included using the variables such as sex, age of onset, severity, disease duration, ophiasis and family history.

## Materials and methods

A total of 539 AA patients attended to University Hospitals, Departments of Dermatology in Duzce, Istanbul and Bolu, and Şişli Governmental Hospital, Department of Dermatology in Istanbul were included. This study was carried out between March 1995 and March 2007. The diagnosis of AA was made on clinical grounds; biopsy was performed in suspicious cases.

A detailed AA evaluation regarding sex, age, age of onset, duration of the disease, attack number, ophiasis, family history and numbers of affected first-, second- and third-degree relatives were noted.

We adopted the AA investigational assessment guidelines collated by Olsen *et al*.[8] The extent of hair loss was classified as:

1 Mild: S1 (< 25% hair loss) or S2 (26–50% hair loss).
2 Severe: S3 (51–75% hair loss), S4 (76–99% hair loss) or S5 (total scalp hair loss, alopecia totalis, AT) or S5B2 (total scalp and body hair loss, alopecia universalis, AU).

AA *localization* was defined by five anatomical areas in the scalp (occipital, parietal, frontal, vertex and temporal) in addition to beard-moustache and eyebrow-eyelash areas.

Comparisons were made between characteristics such as sex, age of onset, severity, disease duration, family history and ophiasis.

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**Table 1** Clinical data of patients

| | *n* | % |
|---|---|---|
| Duration of AA | | |
| < 1 year | 443 | 82.2 |
| ≥ 1 year | 96 | 17.8 |
| Severity-Type of AA | | |
| Mild | 280 | 51.9 |
| Severe | *259* | *48.1* |
| Attack Number | | |
| First | 401 | 74.3 |
| 2nd | 88 | 16.3 |
| 3rd | 19 | 3.5 |
| ≥ 4 attack | 31 | 5.7 |
| Location | | |
| Occipital | 108 | 20.0 |
| Beard-moustache* | 92 | 27.6* |
| Vertex | 72 | 13.3 |
| Parietal | 64 | 11.8 |
| Temporal | 47 | 8.7 |
| Frontal | 39 | 7.2 |
| Eyebrows + Eyelashes | 13 | 2.2 |
| Ophiasis | | |
| Present | 125 | |
| Absent | 377 | |
| Not Applicable† | 36† | |
| Family history of AA | | |
| Negative | 407 | 75.5 |
| Positive (total) | 132 | 24.5 |
| First degree | 69 | 12.8 |
| 2nd degree | 40 | 7.4 |
| 3rd degree | 35 | 6.5 |
| > 1 family member | 14 | 2.5 |

*It is valid for male patients.
†AT/AU patients were not involved.

Group definitions used in these comparisons were as follows:

1 Age of first attack: Childhood group, ≤ 18 years; Adulthood group, > 18 years
2 Duration of disease: D1, 1–359 days; D2, ≥ 360 days
3 Family history: F0, Negative; F1, Positive
4 Attack number: N1, 1–5; N2, ≥ 6
5 Ophiasis: O (–), Negative; O (+), Positive
6 Severity: Mild, Severe

### Statistical analysis

A statistical package program was used for the calculations with a maximum significance level set at 0.05. Chi-squared test was used to examine differences with categorical variables such as age group, ophiasis and age of onset, etc. First, we provide descriptive statistics (frequencies, means and standard errors) and calculate odds ratios (OR) with 95% confidence intervals (95% CI).

## Results

### General evaluation

The ages of the 539 patients ranged from 2 to 75 years (mean ± SE, 24.32 ± 0.54). The age of onset ranged from 1 to 60 years (mean ± SE, 22.12 ± 0.53). The study population comprised 206 females (38.2%) and 333 males (61.8%) with a male/female ratio of 1.6 : 1.

One hundred eighty-two patients (33.8%) were in the child group (≤ 18 years) during the last attack, whereas 357 patients (66.2%) were in the adult group (> 18 years). According to the age of onset, 220 patients (40.8%) were in the child group (≤ 18 years), and 319 patients (59.2%) were in the adult group (> 18 years). Duration of AA ranged from 1 day to 22 years with a median of 60.00 days. Additional demographic and clinical data of the patients were illustrated in Table 1.

### Assessment for sex

There were no significant differences amongst disease duration, severity, ophiasis and family history ($P > 0.05$) for sex.

Childhood onset of the disease occurred in 48.5% (100 patients) of females and 36% (120 patients) of males showing statistical significance ($P = 0.04$, $\chi^2 = 8.24$; OR, 1.67; 95% CI, 1.17–2.38; Table 2).

### Assessment for age of onset

There was no correlation between severity of disease and age of onset ($P > 0.05$).

**Table 2** Results of some comparisons

| | Age of first attack | | Duration | | Severity | | Ophiasis | | Family history | |
|---|---|---|---|---|---|---|---|---|---|---|
| | *P* | $\chi^2$ | *P* | $\chi^2$ | *P* | $\chi^2$ | *P* | $\chi^2$ | *P* | $\chi^2$ |
| Sex | 0.04 | 8.24 | 0.31 | 0.99 | 0.37 | 0.79 | 0.16 | 1.88 | 0.27 | 1.21 |
| First attack age | – | – | 0.13 | 6.13 | 0.76 | 0.09 | 0.000 | 17.66 | 0.008 | 7.02 |
| Duration | 0.016 | 14.41 | – | – | 0.000 | 20.04 | 0.000 | 18.00 | 0.62 | 0.23 |
| Severity | Done | Done | Done | Done | – | – | 0.000 | 42.73 | 0.26 | 1.24 |


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AA episodes of greater than 1-year duration (D2) occurred in 22.7% of childhood (50 patients) and in 14.4% of adult (46 patients) AA cases ($P = 0.016$, $\chi^2 = 6.13$; OR, 1.75; 95% CI, 1.09–2.79; Table 2).

Positive family history was 1.7 times more common in patients with an onset of childhood AA ($P = 0.008$, $\chi^2 = 7.02$; OR, 1.71; 95% CI, 1.13–2.59) compared with adult onset group. Ophiasis was 2.39 times more common for the same patients ($P = 0.000$, $\chi^2 = 17.66$; OR, 2.39; 95% CI, 1.55–3.69; Table 2).

### Assessment for severity

In this step, we compared *mild* and *severe* groups. No significant difference was found with regard to family history ($P > 0.05$).

AA episodes lasting more than 1 year were present in 10.7% (30 patients) and 25.5% (66 patients) of patients in mild and severe groups, respectively ($P = 0.00$, $\chi^2 = 20.04$; OR, 2.8; 95% CI, 1.78–4.56; Table 2).

Ophiasis was found in 13.6% of individuals (38 patients) in mild group and in 39% of individuals (87 patients) in severe group (only patients with 51–99% involvement was included; $P = 0.00$, $\chi^2 = 42.73$; OR, 4.05; 95% CI, 2.65–6.27; Table 2).

### Assessment for disease duration

(D1, < 360 days; D2, ≥ 360 days)

No difference in family history was found amongst patients in the D1 and D2 groups ($P > 0.05$).

Ophiasis was determined to be 2.9 times more common in the D2 than in the D1 group ($P = 0.00$, $\chi^2 = 18.00$; OR, 2.95; 95% CI, 1.76–4.91; Table 2).

## Discussion

AA is hypothesized to be an organ-specific autoimmune disease with genetic predisposition and environmental triggers.[9] Although classic data suggest that AA affects men and women equally,[10] many studies in English literature suggest AA is more common in females[3,4,6,11] than in males.[2,5,7] This might be due to a heightened awareness of AA in women. In our study, increased rate (more than 1.5 times) seen in male patients should be interpreted carefully. Our results derived from an outpatient clinic application, not a field study. As such, it may not be indicative of the actual presentation of the disease in the general population. In addition, the number of women with AA in this study could be skewed due to religious practices such as head covering. Women may not feel the need to consult a dermatologist for their hair loss condition if it is socially acceptable to cover their heads.

Our study agrees with the notion that AA onset for most affected patients occurs in the 3rd decade of life, as noticed in some other studies.[2,3,7,11] The earlier age of onset in women than in men was a remarkable finding that was also mentioned by Sharma *et al*.[7]

It is well known that AA may manifest itself in any hair-bearing area. Which location does AA mostly prefer? We found that AA is mainly located on the occipital area as Ro has found.[5] However, the more impressive result was that AA actually is mostly located on the beard-moustache area in male patients. Again, because this study is an application study, this finding should be taken with a disclaimer: AA in the beard area may cause more distress for male patients than that hair loss in other areas, thus provoking more clinical consultations.

Long-term follow-up has revealed that AA is a recurrent disease.[10,12] Most of our cases were experiencing their first AA episode. It is worth noting that the more AA episodes patients' undergo, the less likely we have found they consult a dermatologist. The reasons for this phenomenon may be numerous. When AA is noticed for the first time, it could be disturbing enough for the patient to make an immediate dermatological appointment. Subsequent attacks may increase patients' knowledge of the disease and its course, particularly the evidence of spontaneous recovery seen in the majority of patients.[11] This is a possible rationale that patients suffering from more than one AA episode could use as a deterrent for further clinical visits.

To date, the genetic basis for AA remains unknown. Whereas a combination of environmental factors and possibly autoimmune responses are likely to play a role in the aetiology of the disorder, evidence from measures of heritability, twin studies and congenital case series support the notion that AA is a complex genetic trait.[13] In our study, one of the important investigational criteria was family history, which is controversial and may emphasize the genetic aspect of AA. The majority of cases of AA seem to be sporadic and the reported frequency of a positive family history varies widely between the series, from around 4.6% to 42%.[2–7,11] Many factors other than true differences between populations might explain this wide apparent range in heritability and variation in the frequency of a positive family history is greatest in the smaller series.[14] Our observation is that whereas we studied 88 AA patients before and found positive family history in 26.3% of the patients,[15] in our current study, together with increased number of patients, we found a small decrease (24.1%) in this rate supporting this comment. Although this finding is valid for some studies, it has not been universal. In a large study conducted by Shellow *et al*.[4] out of 800 AA patients (50% of which had AT *or* AU), 42% of them had a positive family history. This result reinforces the notion that the number of patients in

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a study is crucial, although racial-national differences should not be ignored. The risk is highest in the closest relatives, and falls with more distant relationships.[2] We also observed a 'decrescendo' rate from 12.8% to 6.5% of positive family history, in the first- to third-degree relatives, respectively.

Some previous studies revealed that positive family history was associated with early age of onset[11] and severe AA.[7] Our results only corroborate the theory that there is a relationship between early onset and a positive family history of AA. In other words, we suggest that AA in families of affected patients is more likely to present in childhood. Similarly, Colombe *et al*.[16] found that a family history was more common in relatives developing AA before the age 30 years (37% compared with 7.1% in cases with onset after 30 years of age). It seems that this finding is also valid for patients having first attack at 18 years or younger according to our study grouping. Similarly, Yang *et al*.[2] noted that patients with an early onset of AA had more affected first- and second-degree relatives. We failed to show any relationship between family history and severity, duration of disease or sex.

We found that patients presenting with a childhood onset of AA have a longer disease duration than patients with adult onset of the disease. Early onset of AA is a well-known poor prognostic factor,[12] and this was also supported in a long-term follow-up study by Tosti *et al*.[17] We also observed that patients in severe group have a longer disease duration, similar to patients with an early onset of AA.

Probably, the most noticeable finding in this study was about ophiasis being other poor prognostic factor. While not including AT *or* AU patients, we found that ophiasis is not only associated with earlier age of onset (childhood) and longer duration of disease (Group D2), but is also more often in severe group patients. These findings emphasize that ophiasis is indeed a valuable criteria for AA and may be used as a useful prognostic marker for future studies.

Nevertheless, some uncertain aspects should not be ignored in AA studies. Many limitations should be considered such as severity. Generally, patients are classified only once when they have their first clinical visit. However, severity may change over time (from mild to severe types). This is the case not only for severity but also for ophiasis, disease duration and family history, etc. In fact, a long-term follow-up retrospective study of 191 AA patients (mean, 17 years) by Tosti *et al*.[17] revealed some interesting findings particularly for children: when onset of the disease occurs in childhood, the evolution of hair loss is difficult to predict; mild disease shows a tendency to progress to more severe disease. Thirteen of 30 children (43.3%) developed AT or AU after presenting with mild AA at first appointment.[17]

When we compared with previous AA studies, this Turkish study suggests that there are some racial-national differences as well as similarities about AA. In the last years, we observe that the number of patient in AA studies show steady increase leading to more original and confident results. Our aim is to increase our patient population that may contribute the data about AA and to observe with great interest the change or stability of the parameters seen in this paper.

## Conclusion

In this study, based on 'poor prognostic factors', we have shown some conclusions:

1 Early onset, severe involvement and ophiasis are linked with a more persistent case of AA, indicating these parameters as 'poor prognostic factors',
2 Family history has an association only with early onset of the disease, indicating that family history may be 'a poor prognostic factor',
3 Early onset of AA is more common in women common than in men, indicating that future studies should support this finding to become 'a poor prognostic factor', and
4 Ophiasis was found to be a 'highly significant indicator' for poor prognosis at the end of comparisons for many clinical parameters.

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## References

1 Safavi KH, Muller SA, Suman VJ, Moshell AN, Melton LJ 3rd. Incidence of alopecia areata in Olmsted County, Minnesota, 1975 through 1989. *Mayo Clin Proc* 1995; **70**: 628–633.
2 Yang S, Yang J, Liu JB *et al*. The genetic epidemiology of alopecia areata in China. *Br J Dermatol* 2004; **151**: 16–23.
3 Goh C, Finkel M, Christos PJ, Sinha AA. Profile of 513 patients with alopecia areata: associations of disease subtypes with atopy, autoimmune disease and positive family history. *J Eur Acad Dermatol Venereol* 2006; **20**: 1055–1060.
4 Shellow WV, Edwards JE, Koo JY. Profile of alopecia areata: a questionnaire analysis of patient and family. *Int J Dermatol* 1992; **31**: 186–189.
5 Ro BI. Alopecia areata in Korea (1982–94). *J Dermatol* 1995; **22**: 858–864.
6 Tan E, Tay YK, Goh CL, Chin Giam Y. The pattern and profile of alopecia areata in Singapore – a study of 219 Asians. *Int J Dermatol* 2002; **41**: 748–753.


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7 Sharma VK, Dawn G, Kumar B. Profile of alopecia areata in northern India. *Int J Dermatol* 1996; **35**: 22–27.
8 Olsen EA, Hordinsky MK, Price VH *et al*. Alopecia areata investigational assessment guidelines-Part II. National Alopecia Areata Foundation. *J Am Acad Dermatol* 2004; **51**: 440–447.
9 Green J, Sinclair RD. Genetics of alopecia areata. *Australas J Dermatol* 2000; **41**: 213–218.
10 Madani S, Shapiro J. Alopecia areata update. *J Am Acad Dermatol* 2000; **42**: 549–566.
11 Blaumeiser B, van der Goot I, Fimmers R *et al*. Familial aggregation of alopecia areata. *J Am Acad Dermatol* 2006; **54**: 627–632.
12 MacDonald Hull SP, Wood ML, Hutchinson PE, Sladden M, Messenger AG. Guidelines for the management of alopecia areata. *Br J Dermatol* 2003; **149**: 692–699.
13 Aita VM, Christiano AM. The genetics of alopecia areata. *Dermatol Ther* 2001; **14**: 329–339.
14 McDonagh AJG, Tazi-Ahnini R. Epidemiology and genetics of alopecia areata. *Clin Exp Dermatol* 2002; **27**: 405–409.
15 Kavak A, Baykal C, Özarmaðan G, Akar U. HLA in alopecia areata. *Int J Dermatol* 2000; **39**: 589–592.
16 Colombe BW, Price VH, Khoury EL, Garovoy MR, Lou CD. HLA class II antigen associations help to define two types of alopecia areata. *J Am Acad Dermatol* 1995; **33**: 757–764.
17 Tosti A, Bellavista S, Iorizzo M. Alopecia areata: a long term follow-up study of 191 patients. *J Am Acad Dermatol* 2006; **55**: 438–441.

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