ORIGINAL ARTICLE

Alopecia areata in Egypt : Clinical features and associations

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ABSTRACT

Objective: To assess the clinical features and associations of alopecia areata in Egyptian population.

Patients and methods: A cross sectional study was carried out at the dermatology department, Mansoura University Hospital, Mansoura, Egypt during the period from January 2007 to January 2008. During the study period all patients presenting to the dermatology outpatient clinic with a possible diagnosis of alopecia areata were evaluated. Information regarding age of onset, duration, attack numbers, family history was recorded for every patient. Physical examination and investigations were done to assess the extent of hair loss, nail changes and associated diseases when indicated.

Results: 234 patients were enrolled during the study period. The total number of new dermatologic cases seen over this period was 7548. The incidence of AA was 3.1%. One hundred and twenty two (122) patients (52.14%) were males and 112 (47.86%) were females. Mean age of onset was 24.3 years (range 3-57 years). Duration of the illness varied from two weeks to 6 years. One hundred and seventy (170) patients (72.65%) presented with first episode. One hundred and sixty two (162) patients (69.23%) had patchy alopecia, 32 (13.68%) had ophiasis, 22 (9.40%) had reticulate alopecia, 10 (4.27%) had alopecia totalis (AT), and 4 (1.71%) had alopecia universalis (AU). The majority of patients (194 patients) (82.90%) had their first episode of alopecia areata before the age of 40 years. Of the patients with onset of alopecia areata before the age of 40 years, 38 patients (38/194) (19.58%) presented with extensive alopecia including AT and AU, compared with 2 patients (2/40) (5%) above the age of 40 years; the difference was statistically significant ($P< 0.05$). Nail changes were seen in 53 (22.64%) patients. Personal and family history of atopy was found in 21 (8.97%) patients. There was no significant association between a personal history of atopy and the extent of alopecia areata A family history of alopecia areata was recorded in 34 (14.53%) patients. There was no relation between the frequency of occurrence of family members with AA and the severity of the disease. Eight patients had thyroid disease (3.4%), vitiligo in 4 patients (1.7%), and diabetes mellitus in 5 patients (2.13%). No other autoimmune diseases were reported.

Conclusion: Similar to that reported in the Western and Asian literature, AA is predominantly a disease of the young, with most of severe forms occurring in first three decades of life. In contrary, a lower percentage of our patients had associated atopy, and thyroid disease. Larger studies are recommended to further validate our results.

INTRODUCTION

Alopecia areata is a common disorder of unknown etiology which has an estimated prevalence of 1 in 1000 and accounts for 2% of new dermatological outpatient attendances in Britain and United States and an incidence of 17.2 per 100,000 per year.[1,2] There is no known race, sex, or occupational predilection for the development of alopecia areata. Alopecia areata can occur at any age with a mean age of onset of 20 years.[3]

The clinical behavior of AA is unpredictable and the pathogenesis is not completely clear. Concerned etiologic factors include patient's genetic structure, the atopic state, nonspecific and organ-specific immune reactions, and emotional stress.[4] There is mounting evidence that a T-cell-mediated autoimmune response directed at the hair follicles may be responsible for this disease process.[5] The role of genetic factors in alopecia areata is suggested by occurrence of familial cases, occurrence

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in twins, and also by HLA studies.[6] Reported figure of familial incidence vary from 10% to 50%.[7] The mode of inheritance was thought to be autosomal dominant with variable penetrance.[8] There also have been reports of alopecia areata occurring in monozygotic twins, sometimes, with concurrent onset.[9,10] There are limited clinical studies on AA in Egyptian patients. The aim of this study was to review assess the clinical features and associations of alopecia areata in Egyptian patients.

PATIENTS AND METHODS

Patients with clinical diagnosis of alopecia areata during January 2007 to January 2008 who consulted the dermatology outpatient clinic of Mansoura University Hospital, Mansoura, Egypt were included in the study. Written informed consent was obtained from the all patients or their guardians.

Diagnosis of alopecia areata was made on the basis of the definition of Olsen et al.11 Patient's detailed history regarding age of onset, duration of illness and associated symptoms, was taken. Personal and/or family history (first-degree relatives) of alopecia areata, atopy, thyroid disease, vitiligo, diabetes mellitus and other auto-immune disorders were noted. Physical examination included evaluation of disease extent, presence of exclamation mark hairs, associated nail changes, and whole body examination for associated diseases. Laboratory tests were done in the light of physical examination where necessary.

Extent of hair loss was classified as scalp involvement <25% (group 1, mild), scalp involvement 25-75% (group 2, moderate), and scalp involvement >75% including alopecia totalis and universalis (group 3, severe). During the data collection main site of involvement, pattern, and extent of alopecia were recorded on a proforma for every patient which was a modification of the AA investigational assessment guidelines collated by Olsen et al.[11] Descriptive statistics and frequencies were reported for the desired variables. Fisher's exact probability test was used for statistical evaluation. A significance level of $P< 0.05$ was used

RESULTS

There were a total of 234 new cases of AA seen from January 2007 to January 2008. The total number of new dermatologic cases seen over this period was 7548. The incidence of AA was 3.1%. Out of the 234 patients studied, 122 were males and 112 were females. Age of onset varied from 3 to 57 years with a mean age of onset of 24.3 years. One hundred and ninety-four patients (194) (82.9%) experienced their first episode of AA within the first four decades of life.

Duration of the illness varied from two weeks to 6 years. One hundred and seventy patients (72.65%) patients presented with first episode, 46 patients (19.66%) with second episode and in 18 patients (7.69%), disease had been continuous with regular appearance of fresh patches. The disease was asymptomatic in all of the patients except for ten patients (4.27%) who had mild itching in the lesions. Scalp was the most common site affected; 182 patients making up to 77.78% of the total had lesions over scalp, face and eye brows, while in 48 (20.51%) patients, patches of alopecia were exclusively confined to the face. (Table 1)

Different patterns of alopecia areata seen during this study are summarized in Table 1. In 21 patients a combination of patchy, reticulate and ophiasis pattern were seen. Three patients with persistent patchy disease had past history of alopecia universalis.Of the patients with onset of

Table 1 Age groups, patterns, and associations of alopecia areata.

Character	Number (%)
Sex :	
Males	122 (52.14%)
Females	112 (47.86%)
Age groups according to disease onset	
0-10 years	48 (20.51)
11-20 years	34 (14.51)
21-30 years	70 (29.91)
31-40 years	42 (17.94)
41-50 years	31 (13.24)
>50 years	9 (3.84)
Duration of illness	
0-6 months	146 (62.39)
6-12 months	46 (19.66)
1-5 Years	24 (10.26)
>5 Years	18 (7.69)
Extent of hair loss	
Group I =<25% scalp involvement	150 (64.1)
Group II =25-75% scalp involvement	44 (18.8)
Group III group III=>75% scalp involvement including alopecia totalis and universalis	40 (17.09)
Patterns of alopecia areata	
Patchy	162 (69.23)
Reticulate	22 (9.40)
Diffuse	4 (1.71)
Ophiasis	32(13.68)
Alopecia totalis	10 (4.27)
Alopecia universalis	4 (1.7)
Family history of alopecia	34(14.53)
Personal and family history of atopy	21 (8.97)
Nail changes	53 (22.64)
Thyroid disease	8 (3.42)
Vitiligo	4(1.71)
Diabetes mellitus	5 (2.13)

alopecia areata before the age of 40 years, 38 patients (38/194,19.58%) presented with extensive alopecia including AT and AU, compared with 2 patients (2/40, 5%) above the age of 40 years; the difference was statistically significant (P< 0.05) . Alopecia totalis and universalis occurred in 14 patients, all of them was before age of 40 except two. Nail changes were seen in 53 (22.64%) patients; longitudinal ridging, fine pitting were the most common findings. Nail roughening and transverse ridging were also seen. No patient had severe nail dystrophy. (Table 1)

Personal and family history of atopy (atopic dermatitis, asthma, and allergic rhinitis) was found in 21 (8.97%) patients. Atopy was present in 17 patients (17/194, 8.67%) with limited alopecia, compared with 4 patients (4/40, 10%) with severe alopecia. The difference was not statistically significant (P> 0.05). A family history of alopecia areata was recorded in 34 (14.53%) patients. There was no relation between the frequency of occurrence of family members with AA and the severity of the disease. Family history of alopecia areata was present in 28 patients (28/194, 14.4%) with limited alopecia, compared with 6 patients (6/40, 15%) with severe alopecia. The difference was not statistically significant (P> 0.05) Eight patients had thyroid disease (3.4%), 4 patients had vitiligo (1.7%), and 5 patients had diabetes mellitus in (2.13%). No other autoimmune diseases were reported. (Table 1)

DISCUSSION

There have been few clinical epidemiologic studies on AA worldwide.[12,13] It accounts for about 2% of new dermatology outpatient attendances in the West,[14,15] 0.7% in one report from India12, and 3.8% in Singapore.[16] A study from Kuwait, reported that alopecia areata was observed to constitute 6.7% of the total number of cases in pediatric dermatology clinics.[17] In our study, it accounts for 3.1 of new dermatology outpatients.

Sex incidence in our study is almost equal, which agrees with other reports with the same sex

incidence.[18,19] Most of our patients (82.9%) were in the first four decades of life and the mean age of onset was 24.3 years. This age of onset is consistent with that of previous reports,[3,12,14] where 85.8% experienced their first episode of AA before the age of 40.On the average 20% patients of alopecia areata have been reported to have long term disease.[2] In this study 42 patients (17.95%), however, had duration of disease greater than one year, while in 18 of them patients it was greater than 5 years.

Various disease associations with AA were examined in our study. Personal and family history of atopy was found in 21 of our patients(8.97%), which is lower than that of earlier studies which reported frequencies between 10 and 50%.[12,13,20-23] The lower frequency of atopy in our study may be a reflection to lower incidence of atopy in our locality which ranges between 7 to 9 % and may be attributed to genetic and environmental factors.[24,25] A study conducted in The Netherlands reported a positive significant correlation between atopy and the severity of AA.[26] In our study, however, there was no significant correlation between a personal history of atopy and the extent of AA , which was similar to reports from Singapore[16] and India.[27]

We found a family history of alopecia areata in 14.53% of our patients. Review of literature, however has revealed variable results. Kavak et al.[19] have shown a positive family history in 24.1% of their patients in Turkey. A family history of AA was seen in 4.6% of AA patients in Singapore[16], and in (1.7%) of an Indian population.[12,27] In China, Yang S, et al.,[28] reported family history in 8.4% of patients. A stronger family history of AA (frequency of 20-40%) was reported in the Western population.[13,15,20] These differences may be due to the different genetic backgrounds.

Patchy alopecia areata was the most common pattern seen in 162 (69.23%) patients followed by ophiasis in 32 (13.68%) patients. Ophiasis pattern was often associated with chronicity of the disease.. In accordance with earlier studies,[12,13,26] most of severe forms of AA in our study affected young individuals. Of the patients with onset of alopecia areata before the age of 40 years, 38 patients (38/194,19.58%) presented with extensive alopecia including AT and AU, compared with 2 patients (2/40, 5%) above the age of 40 years; the difference was statistically significant ($P< 0.05$). Alopecia totalis and universalis occurred in 14 patients (5.98% of the total), all of them was before age of 40 except two. Seyrafi H, et al.[18] have shown very high percentage of severe forms of disease.

Association of alopecia areata with autoimmune diseases is well established. In our study, the frequency of most associated autoimmune diseases (thyroid diseae and vitiligo) was relatively low compared with that in other reports. A study done exclusively for the purpose of finding autoimmunity in children with alopecia areata has found statistically significant association of thyroiditis, as well as family history of other autoimmune diseases.[29] These findings have been shown by many other researchers.[18,23]. Some reports have shown an incidence of 8-28% of AA patients with thyroid disease.[13,15] Our results, however, are on the contrary. Only eight (3.42%) of our patients had thyroid disease. Our finding of lower frequency of associated thyroid disease is similar to that from India, Singapore, and Netherlands and.[12,16,26] One reason could be that thyroid function tests were not done as a routine in this study. These were requested on clinical suspicion; thus some subclinical cases may have been missed.

The frequencies of associated vitiligo were 1.8% reported by an Indian group;[12] 3% by Snellow et al.,[20] 4% by Muller and Winkelmann13,16% by Cunliffe et al.[15] and 4.1% by Tan et al.[16] The frequency of associated vitiligo was 1.71% in our study. Again, these differences can be attributed to different genetic factors in different races. Type I DM reportedly occurs more often in family members than in AA patients themselves, while no difference has been reported for type II DM.[15] Reports generally show a < 1% rate of DM in AA patients. Goh et al,[23] showed a rate of 0.6% in their AA patients. Tan et al,[16] reported that 3.2% of their AA patients and 6.8% of their family members had diabetes mellitus. In our study, only 5 patients (2.13%) had diabetes mellitus. It is not clear from our data whether patients or their family members had type I or type II DM.

Nail changes were observed in 53 (22.64%) patients in this study. Nail involvement in alopecia areata has been variably reported from 7% to 66%.1 Sharma et al.,[12] from India, have reported 20% chance of having nail changes in their patients. They also found a significant association of nail changes with disease severity. Ahmed et al.[30] found nail changes in 35% of their Pakistani patients.

CONCLUSION

Alopecia areata is a psychologically debilitating disease for which no cause has yet been found. Disease pathogenesis has been unraveled in the last few decades and polygenic inheritance potential has been suggested. Among various races no large differences are apparent in the clinical features and associations of alopecia areata. Similar to that reported in the Western and Asian literature, AA is predominantly a disease of the young, with most of severe forms occurring in first three decades of life. In contrary, a lower percentage of our patients had associated atopy, and thyroid disease. Further studies are recommended to validate our results and to better understand the disease process.

REFERENCES

1. De Berker DAR, Messenger AG, Sinclair RD. Disorder of hair. In: Burns T, Breathnach S, Cox N, Griffiths C, eds. Rook's textbook of dermatology Vol IV eds. Blackwell scientific publications, Oxford 2004; p 63.36-63.46.
2. Price VH. Alopecia areata; Clinical aspects. J Invest Dermatol 1991; 96 (Suppl):68S.
3. Shellow W VR, Edward JE, Koo JYM. Profile of alopecia areata: A questionnaire analysis of patient and family. Int J Dermatol 1992; 31:186-9.
4. Wasserman D, Guzman-Sanchez DA, Scott K, McMichael A. Alopecia Areata. Int J Dermatol 2007; 46:121-31.
5. Alexis AF, Dudda-Subramanya R, Sinha AA. Alopecia areata: autoimmune basis of hair loss. Eur J Dermatol 2004; 14:364-370.
6. Gilhar A, Paus R, Kalish RS. Lymphocytes, neuropeptides, and genes involved in alopecia areata. J Clin Invest 2007; 117:2019-27.
7. Mitcholl AJ, Krull EA. Alopecia areata: Pathogenesis and treatment. J Am Acad Dermatol 1984; 11:763-75.
8. Simpson NB. Alopecia areata. In: Rook AJ, Dawber RPR, eds. Diseases of Hair and Scalp, 2nd edn. Oxford; Blackwell 1991; p 331.
9. Scerri L, Pace JL. Identical twins with identical alopecia areata. J Am Acad Dermatol 1992; 27:766-7.
10. Colombe BW, Price VH, Khoury EL, Lon CD. HLA class-II alleles in long standing alopecia totalis/alopecia universalis and long standing patchy alopecia areata differentiate these two clinical groups. J Invest Dermatol 1995; 104 (Suppl):4S-5S.
11. Olsen E, Hordinsky M, McDonald-Hull S et al. Alopecia areata investigational assessment guidelines. National Alopecia Areata Foundation. J Am Acad Dermatol 1999; 40:242-6.
12. Sharma VK, Dawn G, Kumar B. Profile of alopecia areata in Northern India. Int J Dermatol 1996; 35:22-7.

13. Muller SA, Winkelmann RK. Alopecia areata: an evaluation of 736 patients. Arch Dermatol 1963; 88:290-297.
14. Dawber RPR, de Berker D, Wojnarowska F. Disorders of hair. In: Champion RH, Burton JL, Burns DA, Rook/Wilkinson/Ebling Textbook of Dermatology. Boston: Blackwell Science, 1998:2869-2973.
15. Cunliffe WJ, Hall R, Stevenson CJ, Weightman D. Alopecia areata, thyoid disease and autoimmunity. Br J Dermatol 1969; 81:877-881.
16. Tan E, Tay Y-K, Goh C-L, et al. The pattern and profile of alopecia areata in Singapore – a study of 219 Asians. Int J Dermatol 2002, 41, 748-753.
17. Nanda A, Al-Fouzan AS, Al-Hasawi F. Alopecia areata in children: a clinical profile. Pediatr Dermatol 2002; 19:482-485.
18. Seyrafi H, Akhiani M, Abbasi H et al. Evaluation of the profile of alopecia areata and the prevalence of thyroid function test abnormalities and serum auto antibodies in Iranian patients. BMC Dermatology 2005, 5:11.
19. Kavak A, Yesildal N, Parlak A et al. Alopecia areata in Turkey: demographic and clinical features. J Eur Acad Dermatol Venereol 2008; 22:977-81.
20. Snellow WVR, Edwards JE, Koo JYM. Profile of alopecia areata: a questionnaire analysis of patient and family. Int J Dermatol 1992; 31:186-189.
21. Penders AJM. Alopecia areata and atopy. Dermatologica 1968; 136:395-399.
22. Young E, Bruns HM, Berrens L. Alopecia areata and atopy. Dermatologica 1978; 156:306-308.
23. 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: 1055-60.
24. Magdy Zedan,1 Ahmed Settin,2 Mohamed Farag et al. How do Egyptian children describe asthma symptoms?. Egyptian Journal of Bronchology, 2009, 3:74-80.
25. Georgy V, Fahim HI, El Gaafary M et al. Prevalence and socioeconomic associations of asthma and allergic rhinitis in nothern Africa. Eur Respir J 2006; 28:756-762.
26. DeWaard-Van Der Spek FB, Oranje AP, De Raeymaecke DMJ, Wynia RP. Juvenile versus maturity onset alopecia areata - a comparative retrospective clinical study. Clin Exp Dermatol 1989; 14:429-433.
27. Sharma VK, Kumar B, Dawn G. A clinical study of childhood alopecia areata in Chandigarh. Indian Pediatr Dermatol 1996; 13:372-377.
28. Yang S, Yang J, Liu JB et al. The genetic epidemiology of alopecia areata in China. Br J Dermatol 2004; 151:16-23.
29. Kakourou T, Karachristou K, Chrousos G. A case series of alopecia areata in children: impact of personal and family history of stress and autoimmunity. J Eur Acad Dermatol Venereol 2007; 21:356-9.
30. Ahmed I, Nasreen S, Bhatti R. Alopecia areata in children. J Coll Physicians Surg Pakistan 2007; 17:587-90.