ORIGINAL ARTICLE

Beard alopecia areata: a multicentre review of 55 patients

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Abstract

Background Alopecia areata on the beard area (BAA) is a common clinical manifestation, but there are no studies about its characteristics.

Objective To describe the epidemiology, comorbidities, clinical presentation, evolution, diagnostic findings and therapeutic choices in a series of patients with BAA.

Methods This retrospective multicentre review included patients diagnosed with BAA as the first and unique clinical manifestation with at least 12 months of follow-up. Diagnosis was performed based on the typical clinical features. Extra-beard involvement was monitored in all cases.

Results Overall, 55 male patients with a mean age of 39.1 years (range 20–74) were included. Twenty-five patients (45.5%) developed alopecia of the scalp during follow-up and more than 80% of cases appeared in the first 12.4 months. Clinical presentation of AA on the scalp was patchy AA (less than 5 patches) (52%), multifocal AA (28%), AA totalis (12%) and AA universalis (8%). Multivariate analysis revealed a trend of association between scalp involvement and family history of AA without statistical significance.

Conclusions According to this study, BAA may progress to scalp AA in a significant number of patients (45.5% of the patients with a follow-up interval of at least 12 months). In the group of patients who developed scalp AA, 80% of them did it within the first 12 months, so follow-up of patients with BAA is highly encouraged.

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Conflicts of interest

None declared.

Funding sources

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Background

Alopecia areata (AA) is an autoimmune disease[1] with a prevalence of 0.1–0.2% in the general population, and a lifetime risk of 2%.[2,3] It is the most frequent cause of inflammation-induced hair loss,[4] with a not fully understood pathogenesis. Genetic predisposition[5–8] and environmental factors[1,9–12] contribute to develop this non-scarring alopecia. It is thought that a breakdown of the immune privilege precedes an infiltration of the hair follicle by T lymphocytes.[13] This may happen in the context of an impaired state of immunity, as some autoimmune disorders are more prevalent in AA patients.[14–16]

Alopecia areata can occur on any hair-bearing area, but the most affected area in more than 90% of patients is the scalp.[16] AA can present as single or multiple delimited patches of hair

loss, or extensive involvement. Several prognostic factors for determining progression of AA have been reported,[17] such as early onset and lengthy duration. Its course is variable, although spontaneous remission occurs frequently. The treatment is not curative. Different therapies are used, including immunosuppressive drugs, systemic glucocorticoids, intradermal injections of triamcinolone acetonide and immunotherapy with diphenylcyclopropenone.[18–20] Severity of AA at time of first consultation is an important prognostic factor and response to treatment is associated with better prognosis.[21]

Alopecia areata on the beard area (BAA) is a very well-known clinical entity. Well-demarcated, smooth-surfaced patches of hair loss can appear on the beard area in male patients (Fig. 1). In spite of being a non-infrequent entity, there are only anecdotal references to BAA in the literature.[4,22] To our knowledge, there are no large studies that accurately reflect the profile, clinical presentation and prognosis of patients with BAA. The objective of this study was to describe the epidemiology, comorbidities, clinical presentation, evolution, laboratory findings and therapeutic options in a series of patients given the diagnosis of BAA.

Methods

A multicentre retrospective observational study was designed including patients diagnosed with BAA as the first manifestation of AA between 1998 and 2015 at eight Spanish dermatologic centres. Diagnosis of BAA was made based on the typical clinical presentation (round or oval patches of non-scarring hair loss in the beard area) and characteristic dermoscopic findings (yellow dots, short regrowing hairs and exclamation mark hairs). The inclusion criteria were as follows: male patients over 16 years of age, diagnosis of BAA as the first and only manifestation of AA in life, and a minimum follow-up of 12 months.

Data regarding epidemiology (age, age of onset of BAA, family history), clinical presentation (number of episodes), evolution (scalp involvement, body hair involvement and nail involvement), comorbidities, need of psychiatric treatment due to AA, laboratory evaluation (complete blood cell count, biochemistry, thyroid evaluation and antinuclear antibodies), treatment (therapies used, duration, adverse effects, response to treatment and mean time of response), evolution and time to relapse were analysed.

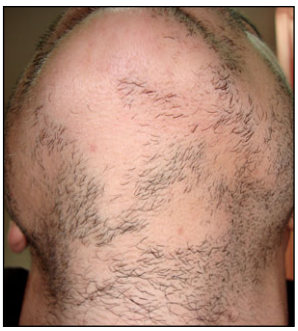

Figure 1 Beard alopecia areata. A 32-year-old man with alopecia areata on the beard area for 6 months treated with topical steroids without response.

The response to therapy was assessed clinically on three groups: more than 75% of regrowth, regrowth of 75% or less and no hair regrowth. The effectiveness of the treatment was evaluated by clinical interview and physical examination separated at least 3 months. Relapse was considered when a hair loss more than 10% of the previous area under/after treatment occurred.

For all continuous variables, mean and range were calculated; and for categorical variables, frequencies were reported. The Mann–Whitney and X^2 tests were used to assess the statistical significance of differences observed between groups for continuous and categorical variables, respectively. To identify the best combination of independent factors associated with scalp involvement, a multivariate (i.e., logistic regression) analysis was performed. Independent factors were expressed as Odds Ratio, 95% confidence interval and a P-value < 0.05 was considered statistically significant. For all statistical analyses, the SPSS 21.0 statistical software package (IBM Corp. Released 2012. IBM SPSS Statistics for Macintosh, Version 21.0. Armonk, NY, USA: IBM Corp.) was used.

Results

The study included 55 male Caucasian patients with a mean age of 39.1 years (range 20–74). The mean age of onset of BAA was 34.5 years (range 18–73). A family history of any type of AA was present in eight patients (14.5%). Skin comoborbidities were as follows: atopic dermatitis in eight patients (14.5%), vitiligo in two patients (3.6%) and psoriasis in two patients (3.6%). Other associated autoimmune comorbidities were as follows: Crohn's disease in three patients (5.4%), asthma in two patients (3.6%) and hyperparathyroidism in one patient (1.8%). A total of six patients (10.9%) required antidepressants as adjuvant therapy for their AA.

Clinically, 41 patients (74.5%) presented only one episode, 6 patients (10.9%) two episodes, 4 patients (7.3%) three episodes and 4 patients (7.3%) four or more episodes of BAA (Table 1). No symptoms were detected in any case. In all, 25 of the 55 patients (45.5%) developed alopecia areata of the scalp during follow-up (Table 2). The mean time after onset of the BAA was 9.9 months (range 2–40), with more than 80% of cases appearing in the first 12.4 months. The most frequent clinical presentation of AA of the scalp was less than five patches of alopecia (13 patients, 52%), followed by multifocal AA of 5 or more patches (7 patients, 28%, Fig. 2), AA totalis (3 patients, 12%) and AA universalis (2 patients, 8%). Extra-beard involvement was detected in six patients (10.9%), four had eyebrow hair loss and

Table 1 Number of episodes related to age of onset, scalp involvement during follow-up, family history and associated autoimmune disorders

Episodes of BAA	Number of patients	Mean age of onset in years and range	Scalp involvement during the follow-up	Mean time in months between BAA and scalp AA and range	Family history of AA	Associated autoimmune disorders
One episode	41 (74.5%)	35.1 (18–73)	20 (48.8%)	8.5 (2–24)	3 (7.3%)	4 (9.8%)
Two episodes	6 (10.9%)	31.0 (21–48)	2 (33.3%)	36 (32–40)	2 (33.3%)	2 (33.3%)
Three episodes	4 (7.3%)	35.0 (30–45)	2 (50%)	23.5 (10–36)	2 (50%)	0 (0%)
Four or more episodes	4 (7.3%)	32.8 (18–45)	1 (25%)	4	1 (25%)	0 (0%)
Total	**55 (100%)**	**34.5 (18–73)**	**25 (45.5%)**	**9.9 (2–40)**	**8 (14.5%)**	**6 (10.9%)**

AA, alopecia areata; BAA, beard alopecia areata.

Table 2 Characteristics of beard alopecia areata patients with involvement of the scalp hair in the follow-up and comparison with beard alopecia areata patients without scalp hair loss.

	Scalp involvement during follow-up	No scalp involvement	Total	*P*-value
Number of patients	25 (45.5%)	30 (54.5%)	**55 (100%)**	NS
Mean age at onset in years and range	35.6 (18–73)	33.5 (18–48)	**34.5 (18–73)**	NS
Mean time of follow-up in months and range	21.2 (12–63)	36.1 (12–144)	**29.3 (12–144)**	NS
Forms of scalp AA	-Patchy AA (<5 patches): 13 (52%) -Multifocal AA: 7 (28%) -AA Totalis: 3 (12%) -AA Universalis: 2 (8%)	NA	**NA**	NA
Mean time between BAA and scalp AA in months and range	9.9 (2–40)	NA	**NA**	NA
Family history of AA	6 (24%)	2 (6.7%)	**8 (14.5%)**	NS
Other autoimmune disorders	3 (12%)	3 (10%)	**6 (10.9%)**	NS
Mean number of episodes of BAA	-One: 20 (80%) -Two: 2 (8%) -Three: 2 (8%) -Four or more: 1 (4%)	-One: 21 (70%) -Two: 4 (13.3%) -Three: 2 (6.7%) -Four or more: 3 (10%)	**See Table** 1	NS
Need for antidepressants	5 (20%)	1 (3.3%)	**6 (10.9%)**	NS

AA, alopecia areata; BAA, beard alopecia areata; NA, not applicable; NS, no statistical significance ($P > 0.05$).
Bold values indicate total values.

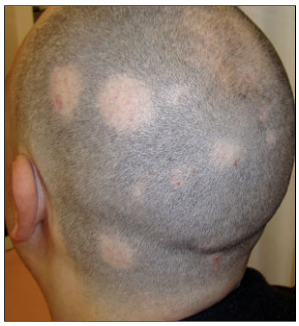

Figure 2 Multifocal alopecia areata. The same patient 1 year later with several patches of hair loss on the scalp.

two had hair loss on the arms during the follow-up. Nail involvement was absent in all patients.

A blood test was performed in 50 patients (90.9%). Laboratory abnormalities included thyroid disorders in five patients (10%) – three patients with hypothyroidism, one patient with subclinical hyperthyroidism and one patient with subclinical hypothyroidism – with negative antithyroid antibodies in all cases. Antinuclear antibodies were evaluated in 47 patients (85.5%), with a positive result (1/80 in dilution) in 1 patient. Other laboratory abnormalities were detected in 2 patients who had increased total IgE levels.

No statistical association was found between the progression of AA to scalp hair and age of onset, concomitant autoimmune diseases and number of episodes. Family history of AA was present in patients with scalp hair loss more frequently than in patients without scalp hair involvement, although statistical significance was not achieved (24% vs. 6.7%, respectively; $P = 0.076$). Multivariate analysis also revealed a trend of association between scalp hair loss and family history without statistical significance.

The mean time of follow-up of patients with BAA was 29.3 months (range 12–144). Regarding therapy, the most frequently used treatments were as follows: 34 topical steroids (32.4%), 18 intralesional steroids (17.1%), 17 expectant attitude

Table 3 Different therapeutic options related with time after onset, duration of the treatment, response, time until response, relapse and time until relapse

Treatment	Topical steroids	Intralesional steroids	Expectant attitude	Topical minoxidil	Oral steroids	Topical calcineurin inhibitors	Total of treatments
Number of patients	34 (32.4%) 1 Hydrocortisone 7 Fluticasone propionate 18 Mometasone furoate 8 Clobetasol propionate	18 (17.1%)	17 (16.2%)	16 (15.2%) Concentration 5% 15 Concentration 3% 1	15 (14.3%) Prednisone 11 Dexamethasone 4	5 (4.8%) Tacrolimus 2 Pimecrolimus 3	105 (100%)
Mean time after onset and range (months)	8.0 (1–71)	7.13 (1–36)	NA	4.88 (1–12)	17.46 (2–120)	24.2 (3–60)	8.23 (1–120)
Duration of treatment and range (months)	3.8 (1–10)	10.3 (1–48)	NA	9.44 (2–30)	4.62 (0.3–20)	3.2 (1–8)	6.16 (0.3–48)
Response*(more or less than 75% of regrowth)	>75% 10 (30.3%) <75% 16 (48.5%) No 8 (21.2%)	>75% 8 (44.4%) <75% 9 (50%) No 1 (5.6%)	>75% 4 (23.5%) <75% 6 (35.9%) No 7 (40.6%)	>75% 3 (18.8%) <75% 7 (43.8%) No 6 (37.5%)	>75% 6 (40%) <75% 4 (26.7%) No 5 (33.3%)	>75% 0 (0%) <75% 1 (20%) No 4 (80%)	>75% 31 (29.5%) <75% 43 (41%) No 31 (29.5%)
Mean time of response and range (months)	2.4 (1–4)	3.19 (1–12)	5.8 (2–12)	2.69 (1–4)	2.35 (1–6)	2	2.31 (1–12)
Relapse*	8 (23.5%)	4 (22.2%)	NA	2 (12.5%)	4 (26.7%)	1 (20%)	19 (18.1%)
Mean time until relapse and range (months)	4.4 (1–12)	5.5 (3–12)	NA	Data not available	5.5 (3–12)	4.38 (1–12)	3 (1–12)
Adverse* effects	None	Skin atrophy 1 (5.6%)	None	None	High blood pressure 1 (6.7%) Mialgia 1 (6.7%) Facial swelling 1 (6.7%)	None	4 (3.8%)

*Response, relapse and adverse events percentages are relative to the total of treatments in each group. The rest of percentages are relative to the total of treatments (105).
There was no statistical significance between groups ($P > 0.05$). NA, not applicable.

(16.2%), 16 topical minoxidil (15.2%), 15 oral steroids (14.3%) and 5 topical calcineurin inhibitors (4.8%). Adverse effects from therapy were noted in only four occasions (a case of skin atrophy due to intralesional steroids and three patients with high blood pressure, myalgia and facial swelling, respectively, due to oral steroids). Data regarding the duration of therapies, response and frequency of relapse are detailed in Table 3.

Discussion

Although BAA is a non-infrequent clinical presentation of AA, it is scarcely reported in the literature.[4,16,23,24] The mean age of onset in our study is similar to previous reports of epidemiology of alopecia areata.[3] However, a wide range of age of onset was observed, indicating that BAA may appear at any age in adults.

Forty-one patients (74.5%) presented only one episode of BAA, whereas 25.5% presented two or more episodes. There were no differences in the age of onset between these groups. Family history of AA was more frequent among patients with two or more episodes (35.7%) than those who had only one episode (7.3%), although statistical significance was not achieved. We hypothesize that patients with family history of AA may present forms of AA with a greater tendency to relapse. Despite nail involvement has been classically associated with AA,[3,4,16,25] we did not observe any nail changes among our patients.

The number of patients who developed AA on the scalp was 25 (45.5%) during a mean time of follow-up of 21.2 months. The mean time of follow-up was higher in the group of patients without scalp involvement (36.1 months), so later onset of AA on the scalp in this group seems to be improbable, even if it cannot be excluded. Based on these data, the proportion of patients with BAA who will develop scalp AA may be higher than generally accepted, highlighting the importance of monitoring patients with BAA to detect scalp hair loss. Nevertheless, there may be a selection bias as data were collected only from patients who consulted a dermatologist, and probably we missed patients who had mild or self-limiting disease.

The mean time between onset of BAA and the development of AA on the scalp was 9.9 months, with more than 80% of cases appearing in the first 12.4 months. There is no current agreement in the management updates of AA[18] about the adequate follow-up period of patients with BAA in clinical practice. Based on our data, we propose a follow-up period of at least 12 months, to detect the majority of patients who will eventually develop alopecia of the scalp.

Although an earlier onset of AA has been related with worse prognosis in some studies,[23] we could not observe differences between age of onset of the group with no scalp involvement and the group with scalp alopecia. We neither found differences between rate of prevalence of other autoimmune disorders or number of episodes of BAA. The majority of patients with scalp affectation presented patchy alopecia (52%). Nevertheless, five patients (20% of patients with scalp affectation and 9.1% of the total of BAA patients) developed AA totalis or universalis with BAA as the first and unique clinical manifestation, supporting the need for follow-up in patients with BAA. In our series we did not find other less common clinical forms of AA such as ophiasis or sisaipho.[15]

The development of AA has an important genetic component[6,8–26] and positive family history has been reported to be between 4% and 28%,[16] or even higher in other reports.[23] In our series, 14.5% of patients presented family history of AA. Regarding comorbidities, inflammatory disorders such as atopic dermatitis, asthma, vitiligo and psoriasis were found associated with BAA in the same frequency than previous reports.[23] Thyroid disease was found in a higher frequency than in general population,[27] so thyroid evaluation is advisable in patients with BAA. In spite of no abnormalities in the thyroid autoantibodies were found, we think antithyroid antibodies should be performed in patients with BAA, as we consider this entity as a subgroup of AA and the same exams have to be performed.[4]

There are currently many therapies for AA, but a lack of evidence-based data for their efficacy is still a problem.[18] Treatment choice depends on disease activity, extent of area affected, duration of disease and patient's age.[28] To our knowledge, clinical guides and reviews do not establish specific recommendations of therapeutic approaches in BAA.[18,29] Isolated BAA can be considered as a patchy alopecia areata, so local therapies should be the first therapeutic option. In concordance with this approach, we could observe that the most used therapy in our study was topical steroids (32.4% of therapeutic courses), followed by intralesional steroids (17.1% of therapeutic courses). Expectant attitude was the management choice in 17 occasions (16.2%), and can be an appropriate therapeutic option considering AA as a disease with spontaneous remission in many cases and BAA as an entity without severe aesthetic negative impact.[18] Topical minoxidil, oral steroids and topical calcineurin inhibitors are other therapies used in our study. Although it was not a main objective of the study due to the retrospective design, we sought to describe the response of the different treatments and also the global response. Due to the multicentre approach and the different authors' traditions, we found variability in how drugs were administered, and even some of the patients were treated concomitantly with different drugs. For this reasons, this study did not enable us to evaluate efficacy and safety of the different therapeutic options properly. However, it is a good starting point to design future studies to assess the efficacy of therapeutic interventions in BAA.

Limitations of this study must be noted, apart from the retrospective design. First, the possible recall bias in some epidemiologic data; and second, the potential bias in the assessment of the response of the used therapies. Nevertheless, the main objective of our study was not to evaluate the effectiveness of different therapies for BAA, but to describe the epidemiology, clinical

findings, laboratory abnormalities and therapeutic options in a series of patients. Lastly, the high percentage of progression to scalp AA (45.5%) could be overestimated due to a selection bias (self-limited cases might not search medical assistance and some patients are kept on follow-up less than 12 months).

In conclusion, to our knowledge, this is the largest series of patients diagnosed with BAA as the first and unique clinical presentation of AA that has been reported in the literature to date. Although BAA may have a relatively low impact in quality of life, it can be the first manifestation of AA with later scalp involvement, even to AA totalis or universalis. Therefore, we strongly recommend to monitor patients with BAA for at least 12 months, which reflects the time in which the majority of patients will develop extra-beard affectation.

Author contributions

Study concept and design: Saceda-Corralo, Jaén, Camacho, Vañó-Galván. Acquisition of data: Saceda-Corralo, Grimalt, Fernández-Crehuet, Clemente, C. Bernárdez, García-Hernandez, Arias-Santiago, Rodrigues-Barata, Rodríguez-Pichardo, García-Lora, Vañó-Galván. Analysis and interpretation of data: Saceda-Corralo, Camacho, Vañó-Galván. Drafting of the manuscript: Saceda-Corralo, Bernárdez, Camacho, Vañó-Galván. Critical revision of the manuscript for important intellectual content: Saceda-Corralo, Grimalt, Fernandez-Crehuet, Bernárdez, Jaén, Camacho, Vañó-Galván. Administrative, technical, and material support: Rodrigues-Barata, Bernárdez, García-Lora. Study supervision: Saceda-Corralo, Jaen, Camacho, Vañó-Galván.

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