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ALOPECIA AREATA

A follow-up investigation of outpatient material

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Alopecia areata (A.a.) is not an entirely unusual disease. According to the literature its frequency varies. The prevalence of A.a. in the population of Sweden in 1965 has been given by Hellgren (31) based on studies in two regions: in the county of Norrbotten the prevalence was 0.1 % among men and 0.1 % among women, and in the county of Kristianstad 0.06 % among men and 0.03 % among women. Data available on the frequency of A.a. in selected hospital material (skin clinics) vary between 1 and 4.5 % (19).

The cause of A.a. remains unknown and no causal therapy exists.

To elucidate problems of frequency, recovery and relapse the outpatient material over a five year period at the Department of Dermatology, Karolinska sjukhuset, Stockholm was investigated.

Material

All patients with A.a. who attended outpatient clinics during 1955–1959 received a mailed questionnaire regarding age and localization at onset of the disease, time when healing began, any relapse, other diseases and of A.a. among close relatives which might have occurred.

There was no possibility of evaluating the occurrence of important nail changes. This detail of the clinical picture is therefore not dealt with in the study material.

During the five year period in quests in a total of 269 patients with A.a.—130 men and 139 women—were diagnosed. They represented 0.6 and 0.5 % respectively of all patients who then attended the outpatient department.

In no case had the patients received corticosteroid treatment—intralesionally or perorally. No hormone treatment other than local application of ointment or cream had been given.

The material has been processed by a punch-card routine.

Results

Sex and age distribution and localization at onset for the total group are shown in table 1. Answers to the questionnaire were received from 124 of these persons (response group), 63 men and 61 women. The non-response group thus consisted of 145 persons, of whom 3 were stated to be dead, 81 letters were returned by the postal authorites ("addressee unknown"), and for 61 no information was received.

Sex and age distribution and localization at onset for the response group are shown in table 2. The period from onset to incipient healing, and relapse frequency are given in tables 3 and 4. Concomitant diseases, mental stress and pregnancy, with temporal relation to onset, as well as familial occurrence of A.a. are presented in tables 5 and 6.

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Table 1. Total group: *Age and localization at onset*

Localization at onset	Age at onset														Age not stated		Total		
	0—9		10—19		20—29		30—39		40—49		50—59		>60						
	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	
Temporal	3	4	3	3	4	5	2	4	3	2	1	5	1	1	–	–	17	24	41
Parietofrontal	3	6	8	9	10	9	5	12	2	8	3	1	1	3	–	–	32	48	80
Occipital	5	3	8	5	8	6	7	4	3	6	3	3	–	1	–	–	34	28	62
Beard bed	–	–	–	–	4	–	9	–	2	–	–	–	–	–	–	–	15	–	15
Multiple	5	3	1	4	5	6	3	3	2	3	–	3	1	–	–	–	17	22	39
Eyebrows and eyelashes	1	–	1	1	–	–	–	1	–	–	–	–	–	–	–	–	2	2	4
No information	1	2	1	2	2	3	2	–	2	2	1	2	–	3	4	1	13	15	28
Total	18	18	22	24	33	29	28	24	14	21	8	14	3	8	4	1	130	139	269
	36		46		62		52		35		22		11		5				

Table 2. Response group: *Age and localization at onset*

Localization at onset	Age at onset														Age not stated		Total		
	0—9		10—19		20—29		30—39		40—49		50—59		>60						
	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	
Temporal	3	4	2	3	2	2	2	3	3	–	1	4	1	1	–	–	14	17	31
Parietofrontal	3	3	6	6	7	2	3	7	1	6	1	–	–	–	–	–	21	24	45
Occipital	3	2	6	3	3	3	7	2	4	5	2	3	–	–	–	–	25	18	43
Beard bed	–	–	–	–	1	–	2	–	1	–	–	–	–	–	–	–	4	–	4
Multiple	2	1	–	1	1	–	1	1	–	1	–	1	–	–	–	–	4	5	9
Eyebrows	–	–	–	–	–	–	–	1	–	–	–	–	–	–	–	–	–	1	1
No information	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Total	11	10	14	13	14	7	15	14	9	12	4	8	1	1	–	–	68	65	133
	21		27		21		29		21		12		2		–				

Table 3. Response group: *Age at onset contra incipient healing*

Incipient healing	Age at onset														Total		
	0—9		10—19		20—29		30—39		40—49		50—59		>60				
	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	
0– 6 months	3	3	4	8	6	3	4	7	4	7	2	6	–	1	23	35	58
7–12 months	1	3	2	2	3	–	5	4	3	2	1	1	–	–	15	12	27
1– 2 years	2	2	2	–	1	–	–	–	1	–	–	–	–	–	6	2	8
>2 years	2	–	2	2	–	1	–	–	–	1	1	1	–	–	5	5	10
No healing	2	1	4	1	4	2	5	2	–	1	–	–	1	–	16	7	23
No information	1	1	–	–	–	1	1	1	1	1	–	–	–	–	3	4	7
	11	10	14	13	14	7	15	14	9	12	4	8	1	1	68	65	133

Table 4. Response group: *Healing and relapse contra age at onset*

Prognosis	Age at onset														Total		
	0–9		10–19		20–29		30–39		40–49		50–59		>60				
	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀	
Healing complete	1	2	3	4	6	1	5	6	5	6	3	4	–	1	23	24	47
Healing incomplete	1	1	–	–	–	1	2	–	–	2	1	–	–	–	4	4	8
No healing	1	1	4	1	4	2	4	2	–	1	–	–	1	–	14	7	21
Prim comp healing, relapse	4	3	4	6	4	1	4	3	4	2	–	3	–	–	20	18	38
Prim incomp healing, relapse	4	2	3	2	–	2	–	3	–	1	–	1	–	–	7	11	18
No information	–	1	–	–	–	–	–	–	–	–	–	–	–	–	–	1	1
	11	10	14	13	14	7	15	14	9	12	4	8	1	1	68	65	133
Relapse																	
within 1 year	4	2	2	4	3	–	2	3	1	1	–	1	–	–	12	11	23
1–5 years	4	2	2	3	–	1	–	1	2	1	–	1	–	–	8	9	17
>5 years	–	1	3	1	1	2	2	2	1	1	–	2	–	–	7	9	16
	8	5	7	8	4	3	4	6	4	3	–	4	–	–	27	29	56
Relapse(s)																	
1	–	1	1	2	1	1	2	–	2	1	–	2	–	–	6	7	13
2	1	–	2	1	–	–	–	1	–	–	–	1	–	–	3	3	6
3 or more	7	4	4	5	3	2	2	5	2	2	–	1	–	–	18	19	37
	8	5	7	8	4	3	4	6	4	3	–	4	–	–	27	29	56

Table 5. Response group: *Heredity 21 cases*

Sex	Age at onset	Relapse	Heredity conditions
♂	6	+	Cousin
♂	16	–	Maternal uncle
♂	15	+	Maternal aunt
♂	14	+	Cousin
♂	17	+	Father
♂	19	–	Maternal grandfather + his brothers
♂	20	–	Father
♂	25	+	Father
♂	25	–	Father
♂	23	+	Brother
♂	29	–	Father
♂	34	–	Mother
♂	7	+	Father
♀	15	+	Brother
♀	13	+	Cousin
♀	22	+	Father
♀	28	+	Sister
♀	46	+	Two brothers
♀	46	–	Brother + son
♀	49	–	Sister
♀	23	–	Father + two brothers

Table 6. Response group: *Occurrence of mental stress, somatic disease*

	♂	♀		
Mental stress	21	8		29
Infection, high fever	8	4	12	
Metabolic disease	1	1	2	
Obesity	1	5	6	
Joint disease, rheumatic disease	2	2	4	
Skull injury	0	1	1	25
Sideropenia	0	7	7	
Skin disease	3	1	4	11

Discussion

Onset of A.a. may occur at all ages. The literature contains many data on the age at onset. In one study onset occurred in 60 % of the cases between 6 and 16 (19), in 66 % between 5 and 30, with maximal frequency at 15 (63), in 84 % before 40 (74), in 70 % before 30 (15), in 57 % before 21 and in 9 % after 40 (9), in 44 % before 21 and in 19 % after 40 (2).

(i) Among the 269 persons in the *total group* 15 were children (6 %) with onset before 6 and 93 persons (35 %) with onset before 21. The youngest in the material were two boys aged 1 1/2 and 2 years, and two boys and one girl aged 3 years.

Onset before 3 years of age is unusual, but cases are given in the literature: Gertler (21) in a girl aged 2 months, Warren (75) in a boy aged 6 months, Haldin-Davis (26) in a boy aged 18 months, Sobel (67) in a girl aged 2 years, and Riehl (57) in a girl aged 2 3/4 years.

Onset of A.a. after 60 is also unusual. Friedrich (19) gives a figure of 3 %, and Walker and Rothman (74) only 0.9 %. Haldin-Davis (26) described onset in a man of 77; we have not found any case mentioned in the available literature where onset was at a still more advanced age.

In our material (total group), in 68 cases (25 %) onset occurred after 40, and in 11 cases (4 %) after 60; 3 of these were men and 9 were women. The oldest man is 77 and the oldest woman 70.

There is no difference in sex distribution in the total group, which agrees with the data given in the literature (2, 25, 30, 37, 66, 74). However, Sanz y Benitez (63) found that the ratio men : women was 3 : 1, and Jannarone (36) 2 : 1.

In men localization at onset in the total group was usual on the crown and nape (25 % in each instance). Worthy of note is the large number of onsets in the bed of the beard: 12 %. Among women onset is predominant on the crown (34 %). In a remarkably large number of cases the disease is manifested simultaneously in several places in the hair bed, about 14 %; the same for men and for women.

In his material of 114 persons, Anderson (2) states that onset occurred in the nape in 60 % and on the crown in 25 % of the men, and only in 5 % in the bed of the beard. Among the women onset was in the nape in 27 % and in 56 % on the crown. Like in Anderson's material, no onset occurred in our total group except on the head.

(ii) Age and sex distribution for the *response group* (133 persons) does not differ essentially from that of the total group. The youngest among the boys was 1 1/2 years and among the girls 4 years. The oldest among the men was 77 and the oldest among the women 68.

Distribution of localization at onset in the response group is the same as that in the total group, i.e. among the men most frequently on the crown and nape, and among the women on the crown.

Table 3 shows that in 54 % of the women regrowth began within 6 months, in 72 % within 1 year and in 82 % within 5 years. The corresponding figures for men were: in 34 % within 6 months, in 56 % within 1 year and in 70 % within 5 years. Two persons, both men, stated that their hair had begun to grow again after more than 5 years (7 and 8 years respectively). These results show a significantly greater primary healing tendency in women.

Jannarone (36) stated that regrowth began in 33 % within 6 months, in 50 % within 1 year. Walker and Rothman (74) also give the same figures: 33 % within 6 months and 50 % within 1 year. The latter also stated that regrowth occurred in 67 % within 5 years and in 33 % there was never any healing tendency. Anderson (2) asserted that there was regrowth in 71 % within 1 year.

For healing to begin after 5 years is regarded as unusual (51), but there are reports where healing occurred after a long time, Apfelthaler-Kumer (3) after 7 years, and Freemann (18) after 16 years.

Table 4 shows that complete healing resulted in 34 % of the men and in 37 % of the women. There was complete primary healing with subsequent relapse in 29 % of the men and in 28 % for the women. The total number of cases of relapse was 40 % for the men and 45 % for the women.

Total frequency of relapse varied widely in different materials. Anderson (2) stated 28 %, Fivaz (15) 20 %, Walker and Rothman (74) 86 % and Hagerman (25) 50–75 %.

In our response group 11 % of the women and 21 % of the men stated that no regrowth had occurred within the observation period, which varied between 10 and

15 years. Anderson (2) maintained that healing did not take place only in 1 %. Total baldness developed in only 4 of our cases. In 2 other cases there was loss of hair on all parts of the body. In this connection two persons are of interest who stated that total baldness developed, followed by hair growth a new: after four years in a man where onset occurred when he was 11, and after 8 years in a woman with onset at 52. The man declared that recovery was complete, whereas the woman had a relapse within a year after the hair started to grow. Nobl (51) asserts that total baldness which has persisted for more than one month without any sign of regrowth has a very poor prognosis.

Table 4 also shows how rapidly relapse occurs. In the response group 18 % stated that relapse had taken place within one year; there was no difference between the sexes, but a certain preponderance of onset at an early age. It was stated by 29 % that relapse occurred within 5 years, and after more than 5 years in 12 %. The longest time given between onset and relapse was 11 years, in one man and one woman; in both cases primary healing had begun within 6 months.

Table 4 likewise shows that 27 % asserted that relapse had occurred three or more times. This represents slightly more than two thirds of those where relapse had taken place.

Like Jannarone (36), Pillsbury (53), Hagerman (25) and other investigators, but contrary to Walker and Rothman (74) we found a certain increase in the frequency of relapse when onset of the disease was at an early age.

On the basis of the present investigation, localization at onset does not influence either the healing tendency or the frequency of relapse. This is remarkable, since A.a. in the nape (ophiasis) is regarded as having a worse prognosis than attacks in other localizations (1, 5, 30 etc.).

The *etiology* of alopecia areata is much discussed. The *hereditary* factor is emphasized to a different degree by various authors (62). Thus, for example, Hollander (34) gives 16 %, Anderson (2) 19 %, Brown (9) and Hagerman (25) 29 %, Sabouraud (61) 22 %, Müller and Winkelmann (50) 30 %. It is often difficult, however, on the basis of a disease having a familial manifestation to postulate heredity. The importance of the hereditary factors is strengthened, however, by the fact that A.a. appears simultaneously in enzygotic twins (14, 32, 71).

In table 5 data are presented on the persons in the response group who stated that A.a. had occurred in close relatives. They represent 15 % and appear to have a worse prognosis than the other members of the response group.

Severe mental strain is asserted to be a precipitating factor in the occurrence of A.a. by most authors who discuss the etiologic component (2, 5, 19, 25, 30, 50, 55, 74 etc.).

In the response group in our material 22 % stated that loss of hair had begun in connection with mental stress. A more detailed analysis of the importance of mental factors in A.a. will be reported on later (24).

A large number of different *somatic illnesses* have been regarded as connected with the occurrence of A.a.—focal infections expecially in teeth and tonsils (15, 35, 49), virus infections [measles and influenza, (2)], refractive abnormalities, particularly astigmatism (28), pulmonary tuberculosis (35), pernicious anemia (48, 65), sickle cell anemia (12), encephalitis (45, 46), ventricular polyps (4), diffuse formation of cancer metastases (13), sideropenic anemia and hypercholesteremia (15), hyperthyreosis (74, 77), hypothyreosis (72), hyperinsulinism (17), pluriglandular endocrine disturbances (7, 33, 38), testicle dysfunction (7), ovarian disturbances (74), polyarthritis (64), ulcerous colitis (56).

A.a. has also been described as an infectious condition (2, 11).

In our material 19 % of the persons in the response group have supplied information on somatic diseases, which could be correlated in time with the onset of A.a.; this is presented in table 6. The table also includes data on the presence of sideropenia and other skin diseases, which were

obtained from the case records of the outpatient ward. It must be pointed out, however, that these data, apart from sideropenia and other skin diseases, are supplied by the patients themselves.

A survey of these etiologic problems—so difficult to judge—is given by Klingmüller (40) and Friedrich (19), who both assert, however, that the etiology of A.a. remains obscure.

Some authors have described A.a. in *combination with other skin diseases*—Besnier's prurigo (23) and ichthyosis vulgaris (54), Xeroderma (10), tinea capitis (22, 70), acne vulgaris and acne cystica in the area for A.a. (58), keratoma palmare et plantare (40), congenital hyperkeratoses (68), vitiligo (2, 33, 46, 68), psoriasis (8), lichen spinulosus (41), neurodermitis diffusa (27).

It is stated in the literature that nail changes occur comparatively frequently in A.a., especially in the more widespread forms of the disease (2, 30, 39, 42, 47, 76). The changes usually consist in punctiform pits and/or longitudinal whitish lines in the nails.

Two persons in our material had psoriasis, they were 25 and 28 years of age at onset of A.a., and two with Besnier's prurigo, aged 5 and 19 years at onset of A.a. No other skin diseases were recorded.

The relation between A.a. and *pregnancy* has been given special consideration by some authors.

Onset and relapse have been described by Anderson (2), Klingmüller (40), and others. In our response group one woman stated that onset of A.a. had occurred during pregnancy, another asserted that a relapse of A.a. had taken place during pregnancy, 6 years after the onset of the disease.

Improvement in A.a. during pregnancy is described by Meachen and Provis (44), Strandberg (69), Foerster (16), Waisman and Kepler (73), Marx (43), Walker and Rothman (74), and Funck (20). In our group three women asserted that there had been manifest improvement during pregnancy followed by deterioration after parturition.

As a curious phenomenon in this connection a case is reported by Rogers (59), of a man who, during his wife's first, second and fourth pregnancies, developed A.a., which was cured within a year. At the third pregnancy the child was stillborn, and the husband had no A.a. His wife did not have A.a.

Finally, we point out that treatment consisted mainly in the application of local preparations such as alcohol for the hair, and ointments and creams containing steroids, as well as systemic treatment with different vitamins, A, B, D, E and vasodilators. On the other hand, as was stated in the introduction, no one in the material had been treated with corticosteroids intralesionally or perorally, nor was any form of hormonal therapy given other than the local application of cortisone ointment or cream.

SUMMARY

A follow-up investigation of an outpatient material with alopecia areata was carried out. The 5-year period covered by the investigation was from 1955 to 1959. The material consisted of 269 persons, 130 men and 139 women, who were sent a questionnaire in regard to the healing tendency and relapse frequency, any concomitant diseases, mental stress, pregnancy, and heredity. Answers were received from 133 persons, 68 men and 65 women. The entire material was analyzed with regard to age and localization at onset. In 6 % onset of the disease was before 6 years of age, in 35 % before 21 years of age, in 25 % after 40 years of age, and in 4 % after 60 years of age. The earliest and the most advanced age at which onset occurred was respectively 1 1/2 years and 77 years. There was no difference between men and women. Among the men the most usual localization at onset was on the crown and nape, 25 % in each case; among women it was on the crown, 34 %. Among the men regrowth began within 6 months in 34 %, within 5 years in 70 %. For the women the corresponding figures were: within 6 months in 54 %, within one year 72 %, and within 5 years

in 82 %. There was complete healing in 34 % of the men and in 37 % of the women. Relapse occurred in 40 % of the men and in 45 % of the women. A slightly higher risk of relapse was observed where onset occurred at an early age. Localization at onset had no influence on either the healing tendency or the relapse frequency. The observation period varied between 10 and 15 years.

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