

## Evaluation of serum desmocollin 3 levels in patients with alopecia areata

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### Abstract

Background: Alopecia areata is an autoimmune condition that shows no symptoms of inflammation on the surface of the skin. The cause of hair loss in AA patients is still a mystery, however T cells and proinflammatory cytokines are thought to play a role. Many variables, including genetic predisposition, play a role in the development of an overactive immune response. Desmosomes, the cell adhesion junctions created by epithelial cells, are the source of our serum marker, type-1 transmembrane glycoproteins. Cell and tissue-specific expression patterns for the three Dsc genes expressed in mammals are evident. At Benha University Hospital's dermatology outpatient clinic, 60 individuals with AA were included in the research. In addition, a control group of 30 healthy adults of the same age and sex was used. All patients (100 percent) in this research had a quick onset of symptoms. Only 6.3 percent and 3.3 percent of the patients, respectively, had an intermitted or regressive course of action. The median length was three months, although it varied from two days to eleven years. Fewer than a quarter of the participants in our research (20 percent) had a history of the disease running in their families. Our findings indicated that 50% of patients had atopy and pitting of the nails (51.7 percent). AD was found in more than a third of the patients (38.3 percent). The median number of patches seen in this study was 1.5, with a range of one to nine. More than three-quarters of the patients were afflicted with occipital encephalitis, followed by parietal encephalitis, totalis encephalitis, vertex encephalitis, frontal encephalitis, ophiasis, and beard encephalitis (1.7 percent). According to our findings, 68.3% of the patients had undergone prior therapy, and more than a third (41.6%) had a positive response.

Key words: Alopecia Areata

### 1.Introduction

Hair loss due to an inflammatory condition known as alopecia areata (AA) is frequent and does not leave scars. In terms of appearance, AA may manifest as anything from modest, well-circumscribed patches of hair loss to an absence of all body and scalp hair. This is not an uncommon occurrence. AA affects people of all ages, genders, and nationalities (1).

It is an autoimmune condition that has no visible indications of skin irritation. Alopecia areata The cause of hair loss in AA patients is still a mystery, however T cells and proinflammatory cytokines are thought to play a role. As a consequence of a combination of environmental and viral causes, as well as genetic predisposition, the immune system becomes overactive (2).

Epithelial cells produce type-1 transmembrane glycoproteins, which are found in desmosomes, or cell adhesion junctions. Cell and tissue type-specific expression patterns are seen for the three Dsc genes expressed in mammals (3).

For hair structure and function, desmosomes play a key role. Desmogleins 1 and 3 (Dsg 1 and Dsg 3) as well as desmoplakins, plakophilin, and plakoglobin are some of these components. four points out of a possible five.

Pemphigus-infected mice lacking certain desmosomal proteins were shown to suffer from hair loss as well as skin and mucous membrane lesions. An undulating wave of hair loss that runs from head-to-tail.

Hair thinning was more noticeable in adults, and it occurred in areas. Gentle hair pulls using adhesive tape indicated that anagen (growing) hairs were securely fixed in animals missing certain desmosomal proteins. A pemphigus vulgaris patient's scalp biopsy revealed no follicles in the telogen phase. A desmosome-dependent cell adhesion in the deep stratified squamous epithelium, as well as the anchoring of telogen hair to its outer root sheath, has been shown in this study (5).

Serum marker levels in individuals with alopecia areata will be assessed, together with the clinical importance of those results, in this research.

### 2.Patients and Methods

This study will include 60 patients suffering from AA. In addition 30 apparently healthy individuals of matched age and sex will be chosen as a control group. All patient will be selected from the outpatient clinic of Dermatology, Venereology and Andrology Department of Benha University Hospitals.

### Administrative Design and Ethical Considerations

The study was approved by the local Ethic Committee of Benha Faculty of Medicine. Informed consents were taken from all participants or their parents before the start of the study.

- Confidentiality and personal privacy were respected in all levels of the study.
- Patients felt free to withdraw from the study at any time without any consequences.

- Collected data were not used for any other purpose.

### 2.1 Inclusion criteria:

- Diagnosis of AA will be based on clinical findings, different types, degrees of severity, no of patches, sex and age.
- Both sex are included.

### Exclusion criteria:

- Patients on topical therapy (2weeks) or systemic therapy (1month)
- Patients with other autoimmune diseases, as autoimmune thyroid, Atopy, Diabetes mellitus and inflammatory disorders.

### 2.2 Methods

#### Every participant was subjected to

#### 1- Complete history taking

- Personal history: Name, age, occupation, residence and smoking or special habit of medical importance.
- History of the present condition including; onset, course and duration of AA.
- Past history: history of medications (type and duration), associated systemic diseases, endocrinal problems and previous surgery.
- History of previous treatment of AA (type, dose and duration).

- Family history of AA.

#### 2- General examination

Complete clinical examination were done to exclude other autoimmune or systemic diseases

#### 3- Clinical assessment of the skin (Local examination)

- Description of the AA lesions including site, type and number of patches.
- Nail examination to detect associated nail changes.
- Disease severity in AA patients was assessed by (SALT score).

Severity of Alopecia Tool (SALT) Score was used to find out the quantitative assessment of scalp hair loss. The entire scalp divided into 4 parts based on the surface area, top (40% -0.4), posterior (24% - 0.24), right side (18% - 0.18), and left side of scalp (18% - 0.18). Percentage of hair loss in each area is determined independently and is multiplied by the percentage of scalp covered in that area of the scalp, and covered in that area of the scalp, and summing the products of each area will give the SALT score. For example, the hair loss is 40%, 30%, 20% and 10% in top, back and right and left side respectively, then the SALT score can be calculated as-  $(40 \times 0.4) + (30 \times 0.24) + (20 \times 0.18) + (10 \times 0.18) = 16 + 7.2 + 3.6 + 1.8 = 28.6$ <sup>(6)</sup>.

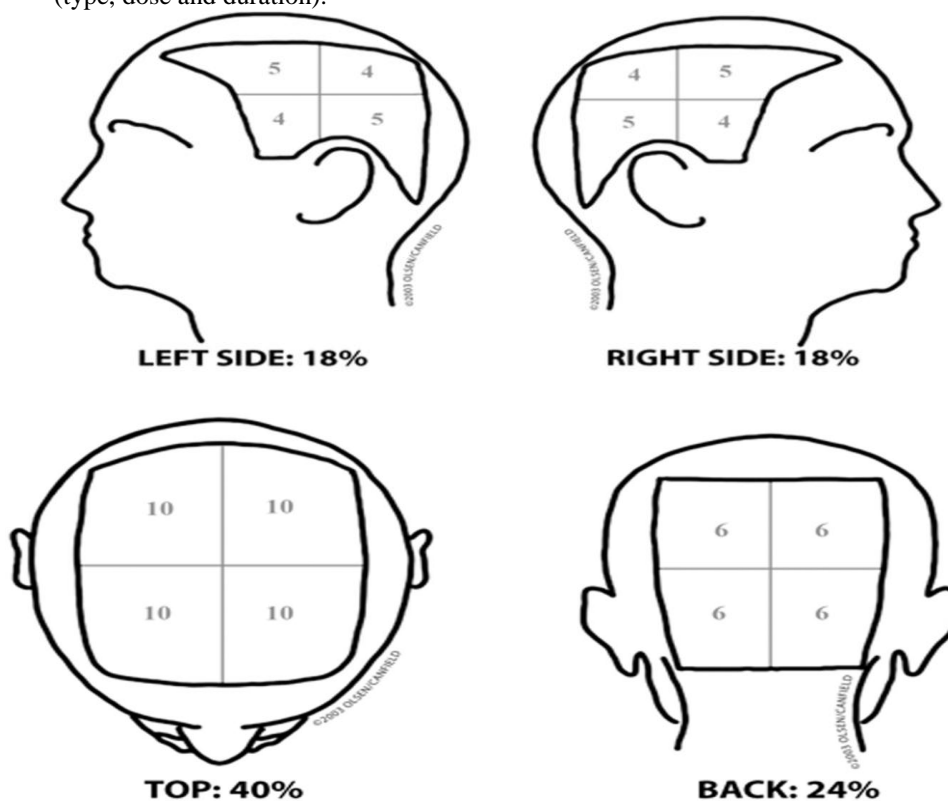


Fig. (1): SALT score aid for determining scalp surface area<sup>(6)</sup>.

#### Statistical analysis

A computer application called Statistical Package for the Social Sciences (SPSS)

version 16 was used to tabulate and evaluate the most important findings. Numbers and percentages were used to represent categorical

data, whereas the mean, SD, and range were used to represent quantitative data. We used Student t tests (also known as t tests) to compare the means of the numerical variables across the various research groups. When the numerical variables are normally distributed, the t-value is equal to the difference between the two groups' means divided by the corresponding standard deviation. Pearson's correlation coefficient is the statistical test

statistic used to determine the statistical link or association between two continuous variables (r- test). An relationship between two variables was evaluated using the non-parametric Spearman's Rho-test. A qualitative variable was compared between two groups using Fisher's exact test (F-test). P-values less than 0.05 were deemed statistically significant when the predicted frequency was less than 5.

### 3.Results

Alopecia onset, course, and duration in the studied patients, regarding onset, all patients (100%) showed sudden onset. Regarding course, most of the patients (90%) showed progressive course, only 6.3% and 3.3% showed intermitted and regressive course, respectively. The median duration was three months and ranged from 2 days to 11 years (Table 1)

**Table (1):** Alopecia onset, course, and duration in the studied patients

		n (%)
<b>Onset</b>	Sudden	60 (100.0)
<b>Course</b>	Intermitted	4 (6.7)
	Progressive	54 (90.0)
	Regressive	2 (3.3)
<b>Duration (months)</b>	Median (range)	3 (2 d - 11 years)

Clinical characteristics of the studied patients, the median SALT score was 9.95 and ranged from 3.2 to 99.4. The mean number of recurrences was 2 with a standard deviation of one recurrence. About two-thirds of the patients (68.3%) received previous treatment, and more than one-third of them (41.6%) showed good response. Below one-quarter of

the patients (20%) had a positive family history. Half of the patients showed atopy (50%) and nail pitting (51.7%). More than one-third of the patients showed atopic dermatitis (38.3%). The median number of patches was 1.5 and ranged from 1 to 9 (**Table 2**)

**Table (2):** Clinical characteristics of the studied patients

		n (%)
<b>SALT score</b>	Median (range)	9.95 (3.20 - 99.4)
<b>Number of recurrences</b>	Mean ±SD	2 ±1
<b>Previous treatment</b>	n (%)	41 (68.3)
<b>Response to previous treatment*</b>	No response	13 (31.7)
	Bad	11 (26.8)
	Good	17 (41.6)
<b>Family history</b>	n (%)	12 (20.0)
<b>Atopy</b>	n (%)	30 (50.0)
<b>Nail pitting</b>	n (%)	31 (51.7)
<b>Atopic dermatitis</b>	n (%)	23 (38.3)
<b>Number of patches</b>	Median (range)	1.5 (1 – 9.0)

\* Percentages were calculated based on 41 patients received previous treatment  
SALT = Severity of Alopecia Tool

Affected sites in cases, the most frequent affected site was occipital (31.7%), followed by parietal (28.3%), totalis (26.7%), and vertex (25.0%). In contrast, the least frequent site was beard (1.7%)

### 4.Discussion

Of epithelial cells, serum marker is a key adhesive force and one of the cadherin adhesion molecules in the super family identified in desmosomes (7).

cell adhesion and hair loss since both of these cell layers exhibit high levels of our serum marker. Hair loss and the formation of skin vesicles were caused by a mutation in the serum marker gene (8).

Two epithelial cell layers surround the bottom part of the hair shaft, which serves as an anchor for hair in the skin. It is likely that the lack of this protein in tissue leads in loss of

The basal and suprabasal layers of the human epidermis, the place where loss of adhesion occurs in PV, have been shown to display our serum marker preferentially (9).

Many breast tumours have reduced expression of the serum marker protein as a result of epigenetic silencing (10).

Carcinogenesis in many cancers may be attributed to the transmembrane adhesion protein desmosomes, which is the serum marker. Reduction in Colorectal cancer has been linked to the marker (11).

Using serum marker-null mice, we were able to establish the importance of our serum marker in maintaining interfollicular epidermal structure and anchoring the telogen hair shaft, as well as the proper function of desmosomes. Hairs were present at birth in all four members of the afflicted household who were present. It was only after shaving ritually that a few areas of hair loss recurred. This hair loss pattern revealed a problem with the telogen follicles' anchoring (9).

In terms of both morphological and ontogenetic continuity, hair follicles are part of the epidermis. Immunohistochemistry has revealed that our serum marker was detected in all cell types examined, Dsc1 was detected only in the outer root sheath companion cell layer and the inner root sheath, Dsg2 was observed in basal cells of the outer root sheath as well as in the central cell layers of the subinfundibular outer root sheath, matrix cells, and the outer root sheath (12).

Clefts formed between the cells surrounding the Telogen Club and the basal layer of the outer root sheath epithelium, dilated telogen hair follicles are responsible for severe alopecia in pemphigus patients (13).

Acantholysis of the keratinocytes surrounding the telogen club hairs was seen in the conditional serum marker knockout mice, which also lost hair. There are two layers of epithelial cells around the lowest portion of the hair shaft that attach a telogen hair in the skin (the telogen club). In wild-type mice, the serum marker is expressed in these two cell layers. As a result, we came to the conclusion that hair loss might be caused by a lack of cell adhesion (acantholysis) between these two cell sheets (9).

An autoimmune blistering condition, such as PV, may lead to alopecia, a word that describes a variety of hair loss patterns (14). As with interfollicular epidermis, desmosomal proteins are expressed in anagen HFs in the same way as they are in interfollicular epidermis (15).

Pseudomonal autoantibodies against desmosomal proteins are implicated in pemphigus, a potentially fatal autoimmune bullous skin condition. It's worth noting that a portion of pemphigus individuals has the same clinical profile as other sufferers (16).

## 5. Conclusion

Atopy, nail pitting, and atopic dermatitis are all linked to alopecia areata since it is an autoimmune illness with a changeable relapsing or remitting history.

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