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# Expression of CD86 on Circulating Monocytes in Patients with Alopecia Areata

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**Abstract**

An introductory word on alopecia areata (AA), a disease that causes hair loss without leaving scars. The emotional toll may be high, resulting in melancholy and withdrawal from friends and family. The peak incidence rate occurs between the ages of 30 and 40. Sixty-six percent of patients are under the age of 30, while just twenty percent are 40 or older. Dendritic cells (DCs), macrophages, B-cells, and other antigen-presenting cells produce the protein CD86, also known as cluster of differentiation 86. (APCs). Both CD80 and CD86 are required for T-cell activation and survival by sending out costimulatory signals. CD86 may signal for either upregulation and cell-cell interaction or downregulation and dissociation, depending on the ligand bound. The goal of this study is to examine CD86 expression on circulating monocytes in AA patients and to analyse the potential function of CD86 in the aetiology of AA. Patient and Method Information: Forty alcoholics-in-recovery (AA) participated in this research. Together with a control group of 20 people of same age and gender who all seem to be healthy. All patients enrolled between September 2020 and November 2021 from the Benha University Hospitals' Dermatology, Venereology, and Andrology Department's outpatient clinic. CD 86+ surface expression was substantially greater in individuals with alopecia areata than in the control group (p=0.014). Thus, it is possible that serum CD86 level contributes to AA aetiology. In addition, its expression level may be used as a surrogate marker for AA risk, activity, and severity. Hair pull test, dermoscopy, and similar terms.

**Keywords:** Expression of CD86, Alopecia Areata, marker for AA.

## 1. Introduction

Nonscarring hair loss is the hallmark symptom of the autoimmune illness alopecia areata (AA). The emotional toll may be high, sometimes leading to sadness and withdrawal from others [1]. The peak incidence rate occurs between the ages of 30 and 40. Sixty-six percent of patients are under the age of 30, while just twenty percent are above the age of forty [2].

Environmental variables, autoimmunity, and the inheritable susceptibility have all been proposed as potential contributors to the genesis of AA [3]. The fact that alopecia areata is associated with other autoimmune diseases, that autoantibodies can be found in the affected hair follicles (HFs), that inflammatory lymphocytes can be found in and around the HFs, and that immunosuppressive drugs can stimulate hair regrowth all lend support to the hypothesis that an autoimmune process is at play [4]. Dendritic cells (DCs), macrophages, B-cells, and other antigen-presenting cells produce the protein CD86, also known as cluster of differentiation 86. (APCs). Both CD80 and CD86 are required for T-cell activation and survival by sending out costimulatory signals. CD86 may serve as a signal for either increased self-regulation and cell-cell interaction, or decreased regulation and dissociation, depending on the ligand bound [5]. The CD86 is made up of a transmembrane region, a longer than CD80 cytoplasmic domain, and two Ig-like extracellular domains (one variable and one constant) [6]. The part it plays in the development of AA has not been examined extensively, however. We aimed to investigate this link between AA and a sample of Egyptians in the present research.

## 2. Aim of the work

Examine the involvement of CD86 on circulating monocytes in the aetiology of AA, and determine the relationship between CD86 expression and other measures of disease severity.

## 3. Patients and Methods

This study included 40 patients suffering from AA. In addition to 20 apparently healthy individuals of matched age and sex as a control group. All patients recruited from the outpatient clinic of Dermatology, Venereology and Andrology Department of Benha University Hospitals on the period between September 2020 and November 2021.

**3.1Inclusion criteria:** All patients were enrolled in the study had different degrees of severity of AA. Dermoscopic features of AA are yellow dots, black dots, broken hairs, tapering hair (exclamation marks) and short vellus hairs**[7]**.

**3.2Exclusion criteria:** Any subject was excluded from the study if he\she:

1. Is AA patient with systemic disease as inflammatory bowel disease, systemic lupus erythromatosus and pernicious aneamia.
2. Has other autoimmune disorder as vitiligo and psoriasis.

**3.3All patients were subjected to the following:**

1- Full history taking:

- Personal history: name, age, sex, occupation, residence and smoking or special habit of medical importance.
- Present history: course, onset and duration of AA in patients.
- Past history: other skin diseases such as acne and atopic dermatitis.
- Family history of AA.
- Drug intake: dose and duration.

2-Complete general examination:

- To exclude other systemic or inflammatory diseases (acne vulgaris, atopic dermatitis and psoriasis).

3-Complete dermatological examination:

Severity of AA was assessed according to sum percentage of hair loss in all 4 scalp areas (SALT score) **[8]**.

4-Laboratory investigations:

Assessment of CD86 expression on circulating monocytes on both AA patient and control groups by flow cytometric analysis.

- Sampling:

One ml venous blood was collected from each subject by clean venipuncture using disposable plastic syringe and placed on a tube (containing EDTA anticoagulant) then stored at refrigerator for flow cytometric analysis within 24 hours-as a whole blood.

**3.4Statistical analysis**

The collected data was revised, coded, tabulated and introduced to a PC using Statistical package for Social Science (IBM Corp. Released 2017. IBM SPSS Statistics for Windows, Version 25.0. Armonk, NY: IBM Corp.). Data were presented and suitable analysis was done according to the type of data obtained for each parameter.

Shapiro test was done to test the normality of data distribution.

- Descriptive statistics:

1. Mean standard deviation (± SD) for numerical data.
2. Frequency and percentage of non-numerical data.

- Analytical statistics:

- Student T Test was used to assess the statistical significance of the difference between two study group means.
- Chi-Square test was used to examine the relationship between two qualitative variables. Fisher's exact test: was used to examine the relationship between two qualitative variables when the expected count is less than 5 in more than 20% of cells.
- Correlation analysis: To assess the strength of association between two quantitative variables. The correlation coefficient defines the strength and direction of the linear relationship between two variables.
- The ROC Curve (receiver operating characteristic) provides a useful way to evaluate the sensitivity and specificity for quantitative diagnostic measures that categorize cases into one of two groups. The optimum cut off point was defined as that which maximized the area under the ROC curve (AUC) value. AUC is that a test with an area greater than 0.9 has high accuracy, while 0.7–0.9 indicates moderate accuracy, 0.5–0.7, low accuracy and 0.5 a chance result (Fischer et al., 2003).
- Regression analysis: Logistic and linear regression analyses were used for prediction of risk factors, using generalized linear models.
- N.B: p is significant if <0.05 at confidence interval 95%.

## 4. Results

This case-control study included fourty patients with alopecia areata (Group A) and fourty age and sex matched apparently healthy subjects as a control group (Group B).

Table 5 showed that Alopecia areata patients showed significantly higher expression level of CD $86^{+}$ on monocytes surface when compared to control group (p=0.014).

**Table (1)** SALT score in all studied AA patients**.**

| | | | | **Total N=40** | |
|---|---|---|---|---|---|
| **Salt Score** | **S1B0N0** | | **N, %** | 19 | 47.5% |
| | **S1B0N1** | | **N, %** | 15 | 37.5% |
| | **S2B0N0** | | **N, %** | 2 | 5% |
| | **S2B0N1** | | **N, %** | 2 | 5% |
| | **S3B0N0** | | **N, %** | 2 | 5% |
| | **S** | **1** | **N, %** | 34 | 85% |
| | | **2** | **N, %** | 4 | 10% |
| | | **3** | **N, %** | 2 | 5% |
| | **N** | **0** | **N, %** | 23 | 57.5% |
| | | **1** | **N, %** | 17 | 42.5% |

**Table (2)** Correlations of CD86 levels with age, onset, duration and SALT score in AA group

| | **AA N=40 CD86** | |
|---|---|---|
| | **rs** | **p** |
| **Age** | -0.084 | 0.612 |
| **Onset** | -0.202 | 0.219 |
| **Duration** | 0.226 | 0.166 |
| **SALT** | 0.793 | **<0.001** |

rs, correlation coefficient

CD86 expression level showed significant positive correlation with SALT score (rs=0.793; p<0.001); but not with age, onset, or duration (p>0.05 for each).

## 5. Discussion

Hair loss without any clinical inflammatory indications characterises the prevalent type of alopecia, alopecia areata, which affects the scalp and/or body [9].

It strikes both young and old, people of diverse races, and men and women equally. Around 1.7% of people will have at least one episode of AA at some point in their lives, and 0.2% of the population currently has AA [10].

Not much was known about the specific molecular pathways that cause hair loss and the pathophysiology of AA until recently [11]. The aetiology of AA has been linked to a wide variety of risk factors, including but not limited to stress, hormones, food, infectious agents, vaccines, and many more [12].

Significant advances in fundamental and clinical immunology imply that AA is an autoimmune response of the CD86 cell, Th1-type, targeting hair follicles in the anagen stage (13). The skin has a high concentration of immune cells and has been shown to be home to separate populations of resident and recirculating memory T cells that migrate and operate in unique ways [14].

Having a molecular weight of 70 kDa, 329 amino acids, a transmembrane region, and a more extensive cytoplasmic domain than CD80, CD86 is a glycoprotein. Interdigitating dendritic cells, Langerhans cells, peripheral blood dendritic cells, memory B cells, germinal centre B cells, and macrophages all express CD86 in a constitutive manner [15]. Natural ligand for CD28 and CTLA-4 [16].

The current study's goals were to [1] analyse the expression of CD86 on circulating monocytes in AA patients, [2] determine the significance of this expression in the pathogenesis of AA, and [3] determine the relationship between these expressions and the other clinical parameters investigated.

Forty AA patients and 20 controls of similar age and sex were included in this research. Subjects were gathered from the Benha University Hospitals' Dermatology and Andrology Outpatient Clinic. Human subjects research was authorised by the Benha Faculty of Medicine's local ethics committee. Before taking anyone's sample, we made sure they were willing to give us their informed permission.

Based on these data, we conclude that CD 86+ expression is considerably greater in AA patients than in the control group. It seems that our findings are in line with those of McElwee et al [17]. Who documented a dramatic upregulation of CD86 in AA-affected mouse models.

Patients with AA had considerably greater serum CD8+ expression levels compared to the control group, and this was correlated positively with disease severity [18].

Increases in IL-2 and IL-15 expression were seen in biopsies of AA scalp lesions compared to non-lesional scalps [19].

Specifically, C3H/HeJ transplanted animals whose production of these pro-inflammatory cytokines was inhibited showed a decrease in AA development and a decrease in the formation of CD8+NKG2D+ T cells [20].

## 6. Conclusion

In terms of AA pathogenesis, serum CD86 level may be relevant. In addition, its expression level may be used as a surrogate marker for AA risk, activity, and severity.

Profession of Interest and Conflict Proclamation

All authors have stated that they have no competing interests.

Moral sanction

The Benha Medical School's Ethical Review Board accepted the study's use of human volunteers. All individuals who took part in the research gave their informed permission beforehand.

**Authorscontribution**

Each author has made an equal contribution to the work.

## References


**[1]. D. Davis and V. Callender.** Review of quality of life studies in women with alopecia.Int Dermatol J Women.vol.4(1),pp.18-22,2018.

**[2]. M. Abedini, F. Shariatmadari, M. Torshizi.** Effects of Zinc Oxide Nanoparticles on Performance, Egg Quality, Tissue Zinc Content, Bone Parameters, and Antioxidative Status in Laying Hens. Biol Trace Elem Res.vol.184(1),pp.259-267,2018.

**[3]. N. Islam, P. Leung, A. Huntley..** The autoimmune basis of alopecia areata: a comprehensive review. Autoimmun Rev; 14(2): 81-89,2015.

**[4]. Bakry O, El Shazly R, Basha M.** Total serum immunoglobulin E in patients with alopecia areata. Indian Dermatol Online J.vol.5(2),pp.122-127,2014.

**[5]. Y. Ohue and H. Nishikawa.** Regulatory T (Treg) cells in cancer: Can Treg cells be a new therapeutic target? Cancer Sci.vol.110 (7),pp.2080–2089,2019.

**[6]. X. Zhang, X. Zhang, P. Wang.** Identification of another primordial CD80/86 molecule in rainbow trout: Insights into the origin and evolution of CD80 and CD86 in vertebrates. Development Comparative Immunol,pp.73-82,2018.

**[7]. M. Mane, A. Nath and D. Thappa.** Utility of dermoscopy in alopecia areata. Indian J Dermatol.vol.56,pp.407-411,2011.

**[8]. U. Bhor and S. Pande.** Scoring systems in dermatology. Indian J Dermatol Venereol Leprol; 72:315-321,2006.

**[9]. . T. Dainichi and K. Kabashima.** Alopecia areata: what is new in epidemiology, pathogenesis, diagnosis and therapeutic options? J Dermatol Sci; 86(1):3-12,2017.

**[10]. J. Choi, D. Suh, B. Lew.** Simvastatin/Ezetimibe Therapy for Recalcitrant Alopecia Areata: An Open Prospective Study of 14 Patients. Ann Dermatol.vol.29(6),pp.755-760,2017.

**[11]. M. Bertolini, Y. Uchida and R. Paus.** Toward the clonotype analysis of alopecia areata-spe-551 cific, intralesional human CD8+ T lymphocytes. J Invest Dermatol Symp Proc.vol.17,pp.9–12,2015.

**[12]. E. Wang and K. McElwee** Etiopathogenesis of alopecia areata: Why do our patients get it? Dermatol Ther.vol.24,pp.337-347,2011.

**[13]. K. McElwee, A. Gilhar, D. Tobin.** What causes alopecia areata? Exp Dermatol,pp.22(9),pp.609-626,2013.

**[14]. R. Watanabe, A. Gehad, C. Yang.** Human skin is protected by four functionally and phenotypically discrete populations of resident and recirculating memory T cells. Sci Transl Med,vol.7(279),pp.279-289,2015.

**[15]. M. Mir.** Introduction to Costimulation and Costimulatory Molecules. Academic Press; Med book,pp.12-15,2015

**[16]. C. Chambers.** The expanding world of co-stimulation: the two-signal model revisited. Trends Immunol.vol.22,pp. 217–223,2001.

**[17]. K. McElwee, R. Hoffmann, P. Paul.** Resistance to Alopecia Areata in C3H/HeJ Mice Is Associated with Increased Expression of Regulatory Cytokines and a Failure to Recruit CD4+ and CD8+ Cells. J Invest Dermatol.vol.119(8),pp.1426-1433,2002.

**[18]. A. Ebrahim, R. Salem, A. El Fallah.:** Serum Interleukin-15 is a Marker of Alopecia Areata Severity. Int J Tricho.vol.11(1),pp.26–30,2019.

**[19]. J. Fuentes-Duculan, N. Gulati, K. Bonifacio.** Biomarkers of alopecia areata disease activity and response to corticosteroid treatment. Exp Dermatol.vol.25(4),pp.282–286,2016.

**[20]. L. Xing, Z. Dai, A. Jabbari.** Alopecia areata is driven by cytotoxic T lymphocytes and is reversed by JAK inhibition. Nat Med.vol.20(9),pp.1043–1049,2014.