

Assessment of Serum Levels of Interleukin-23 in Patients with Alopecia Areata

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ABSTRACT

Alopecia areata is a relatively common skin disease. Scientific theories that explain the causes of this disease vary, but it is often known to be an organic disease of immunogenic origin. Alopecia may be specific or partial through the appearance of one or more spot free of hair and may be complete through the absence of hair For the entire scalp. The actual causes are not known, but the closest perception of the majority of scientists indicates a dysfunction of autoimmunity. The aim of this study was to evaluate the levels of IAA expression in blood in alopecia patients compared to normal individuals in an attempt to assess the role of IAA.

KEY WORDS: Alopecia, IL-23, pathogenesis

INTRODUCTION

Alopecia areata (AA) is a disease that causes marked cosmetic deformity and

psychological morbidity, leading to hair loss on the scalp, face and body. Although, the pathogenesis of AA is poorly understood, evidence which suggests that T cells and cytokines play an important role is accumulating [1].

Interleukin-23 (IL-23) is a heterodimeric cytokine produced predominantly by activated antigen presenting cells, such as macrophages and dendritic cells. The cytokine is composed of a unique p19 subunit and a p40 subunit that is shared with the Th1 signature cytokine, IL-12 [1].

Cytokines produced by Th17 cells are highly pro-inflammatory and are now associated with various autoimmune diseases, such as psoriasis, rheumatoid arthritis and AA [2]. The IL-23/IL-17 axis, therefore, is one of the main cytokine axes driving the pathogenesis of various autoimmune diseases. Sera IL-23 and the number of Th17 cells were elevated in various autoimmune diseases patients compared to control subjects [3].

PATIENTS AND METHODS

This study was carried out in the dermatology department of Fayoum University Hospital. Lab work was performed at the Clinical Pathology Department of Fayoum University Hospital cross sectional study.

Our study included 80 individuals divided into two groups:

Group 1: This group included 40 patients of AA without atopic background.

Group 2: This group included 40 apparently healthy volunteers as controls which are age and sex matched with the patient group.

Exclusion criteria:

Patients were excluded from the study if there was any of the following:

1. Patients receiving systemic drugs (systemic corticosteroids, cyclosporine, or other immunosuppressive therapy) during last six months.
2. Patients who received topical therapy (monoxidl,corticosteroids).
3. Pregnancy or breast feeding women.

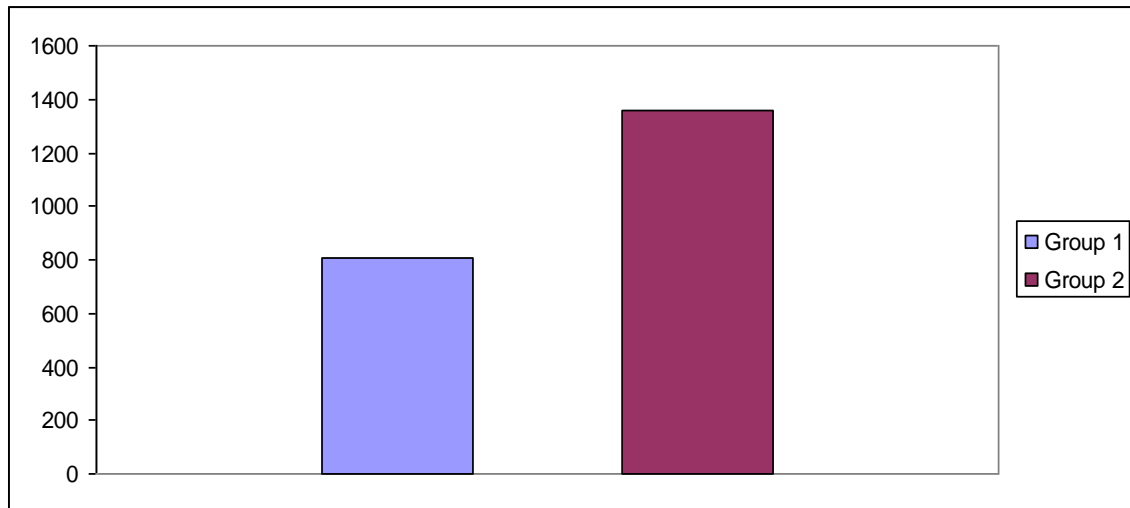
Determination of serum IL-23:

Three ml of blood was drawn in sterile plain tubes. Blood samples were centrifuged and the serum was frozen at -20°C until assayed. Serum levels of IL-23 were measured using enzyme-linked immunosorbent assay (ELISA) technique (Human IL-23 Quantikine ELISA kit Shanghai Korain Biotech CO. ITD).

Results

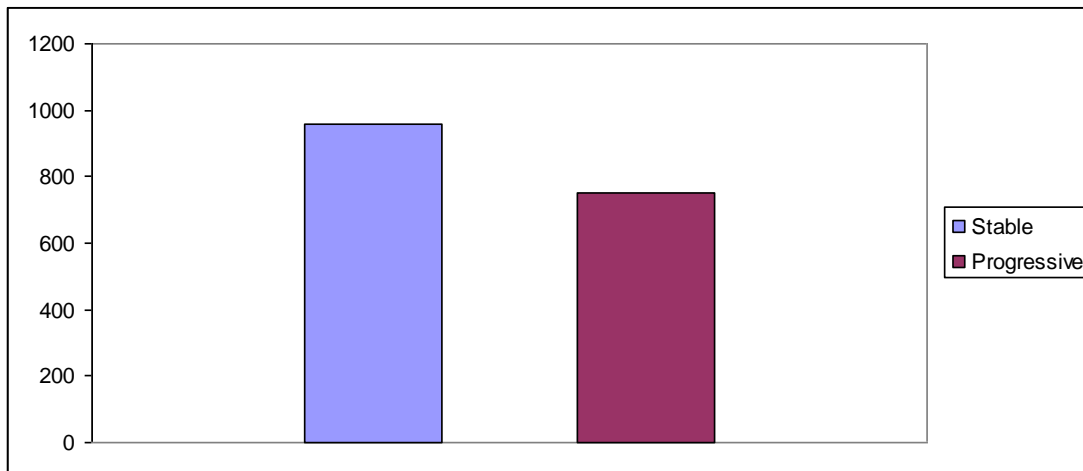
1. Comparison between two groups regarding serum IL-23.

	Scalp	Eye brows	Scalp & abdomentleg	Bread	χ^2	P-value
No	35	3	1	1	83.6	<0.001
%	87.5	7.5	2.5	2.5		



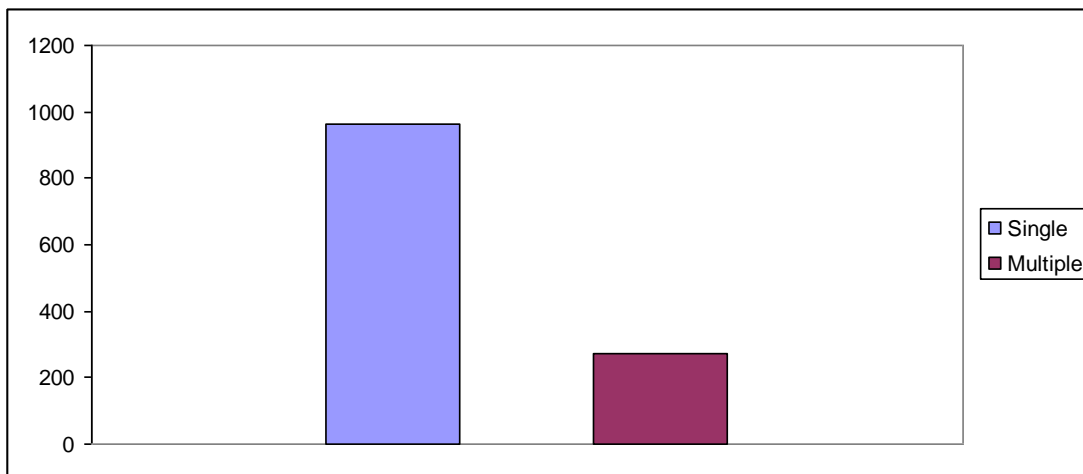
2. Comparison between levels of IL-23 in patients according to disease progression

	Acreata single (31)	Acreata multiple (9)	T-test	P-value
Mean± SD	962.91±1042.9	270.819±289.6842	1.95	0.0583
<i>Range</i>	15.139-2500	25.507-923.059		



3. Comparison between level of IL-23 in patients according to disease type

	Stable (11)	Progressive (29)	T-test	P-value
Mean± SD	958.955±1026.821	749.62±958.96	0.604	0.5488
Range	20.689-2500	15.139-2500		



DISCUSSION

Alopecia areata is a common, chronic inflammatory disease characterized by nonscarring hair loss on the scalp or any hair-bearing area of the body. Although the pathogenesis of AA has not been fully elucidated, clinical and experimental studies have shown that AA is a T cell-mediated autoimmune disease that targets the anagen-stage hair follicles [4]. Furthermore, an alteration of the CD4+T cell subsets, including Th1, Th2, Th17 cells, and regulatory T cells, has been found in AA subjects [5].

There was a statistically significant difference between the 2 groups regarding level of IL-23 as IL-23 in group 1 was 807.19±969.293 and in group 2 was 1359.8±1149. This was in agreement with [6].

Another study found that immunohistochemistry of IL-23 showed positive cells in the intra-follicular epithelium and peribulbar dermis in affected hair follicles of pretreatment lesional scalp and very few cells in post-treatment AA scalp [7], raising stronger suggestion for IL-23 role in the AA disease pathogenesis [8].

CONCLUSION

Our study revealed higher serum level of IL-23 in AA patient, so it may suggest its role in AA pathogenesis.

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