

The Role of IL-10, IL-36 in Psoriasis Patients in Iraq

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Abstract

This study aimed to evaluate the immune role of the tenth and thirty-sixth leukemia (IL-10, IL-36) in a sample of psoriasis patients. As 80 blood samples were collected from patients and 50 samples from healthy blood (as a control group) during the extended period (1/2020/10 to 1/2/2021) from the visitors of the Medical City Hospital. The results of this study showed a significant increase ($p \leq 0.001$) in the level of (IL-36, IL-10) concentration in psoriasis patients compared to the control group.

Key words: between the tenth white and thirty-sixth, psoriasis patients.

Introduction

Psoriasis is a chronic disease that affects 2-3% of people. This disease was previously considered one of the types of disorders that Keratinocyte cells (KC) suffer from, which is similar to the increase in the division of cells that are not controlled by them and their abnormal differentiation (Boyman *et al.*, 2004) either at the time. The present has been classified as one of the types of autoimmune diseases mediated by T cells (Zangenen and Shooshtary, 2013) and there are several factors that help in the emergence of the disease, including the factor of obesity, stress, smoking, alcohol consumption, injury in addition to the genetic factor (Kofink *et al.*, 2013) as there is an increase in the number of T-lymphocytes in the region The epidermis (Ferencz *et al.*, 2000) that produces with endothelial and keratinocytes different types of cellular motility, including TNF- α , IL-22, IL-17, IFN-which have a role in the pathogen development of psoriasis (Kim *et al.*, 2015)

Psoriasis is a common skin disease that is affected by different factors in its etiology. However, it is difficult to know how these different environmental factors have affected the occurrence of psoriasis through disturbance of the interaction of the immune system and the skin epithelium with connective tissue (6) Psoriasis occurs from disorders and irregular reactions of the system The innate and adaptive immune system and these disorders affect the epithelial tissue of the skin and connective tissue (7) IL-10 is a cytokine anti-inflammatory known as Human cytokine synthesis (Girndt, 2003), and IL-10 is called a cytokine inhibitory factor because it acts to inhibit

Synthesis of cellular motility Th1 (Elgert , 2009), and is produced by immune sub-cells including Th1, Th2, Th17 and monocytes, dendritic cells, macrophages, mast cells, B cells, granulocytes and eosinophils, as well as produced by non-immune cells, including epithelial cells, Creatine cells, tumor cells, and IL-10 exerts its anti-inflammatory effect by inhibiting inflammatory precipitating cytokines including IL-1, IL-6, IL-12, and TNF- α (Saraiva and Garra ,2010)

IL-36- is a group of cytokines of the IL-1 family that are mainly produced by keratinocytes, monocytes and dendritic cells (Bassoy et al, 2018) Of the studies it was reported that epidermal growth factors activate the production of IL-36 α and IL-36 in the skin, indicating the contribution of IL-36 cytokines to homeostasis of the skin (Franzke et al ,2012) , IL-36 signaling plays an important role in epithelial immune homeostasis and recurrent disorganization and this may cause psoriasis (Bassoy et al, 2018)

Materials and methods

The study included two groups, the first group (the patient group): This group included 80 patients with psoriasis from the consultancy at Baghdad Teaching Hospital in the Medical City of Baghdad Governorate during the period from February to July. Those included in the study, after putting on the paws and masks and sterilizing the blood drawing area with ethyl alcohol 70% and other sterile materials such as Dettol and using medical syringes where blood was drawn and placed in laboratory tubes, after which the blood was left for 10-15 minutes at room temperature until clotting occurred, after which the serum was separated By using a Centrifuge at a rate of 3000 revolutions / minute for 10 minutes, the serum was placed in laboratory tubes, and then the serums were kept at a temperature of (-20 ° C) until the necessary tests were carried out. Group II (Control Group): This group included 50 non-psoriasis individuals who had no recent history or clinical evidence of psoriasis or any associated infection. The two patient and health control groups are arranged by age, gender and address, and serum levels of cytokines (IL-10 and IL-36) were measured by the direct enzyme-linked immunosorbent assay (ELISA) technique using a ready-made kit for this purpose. The data of all the tests were analyzed statistically by a computer using the (SPSS) Statistical Package of Social Science program, as the T-Test was adopted at a probability level (0.05) to identify the degree of significance of the differences between the transaction rates (Nisi, 2004)

Results and discussion

Measurement of the level of cytokines (IL-10) in serum blood

The results of the current study showed a statistically significant increase ($p \leq 0.001$) in IL-10 levels of psoriasis patients compared to the control group, as the mean values were (225.99 ± 226.76) pg / ml, (29.64 ± 71.55), respectively. Table (4—1)

Table (1-4) shows the level of IL-10 for psoriasis patients and the control group

		Study groups	
		Patients	Control
IL10	Mean	226.76	71.55
	Standard Deviation	225.99	29.64
	Median	109.33	70.14
	Percentile 25	63.75	51.38
	Percentile 75	352.93	85.11
P value		<0.001**	

** : highly statistical significance ($p < 0.001$)

Through these results of the current study, it is noted that the levels of IL-10 increased in psoriasis patients, and this study came in line with other studies. Also, a study (Owczarczyk-Saczonek et al 2019, Khalaf, 2015) .

T-helper T cells of type II (Th2) play a central role in the development of kinetics production including IL-10 and induce formation of inducible regulatory T cells from CD4 + na +ve T cells (Song et al 2015). It also works by inhibiting inflammatory precipitating cytokines (IL-12, IL-1, IL-6, and TNF- α (Saraiva , Garra , 2010). that IL-10 is a regulator. Strongly controls inflammation through autocrine and paracrine mechanisms (Steen et al 2020) and reduces inflammation by inhibiting the action of natural killer cells, and T-cell differentiation (Duellet al 2012). Suppresses inflammation and macrophage activity by inhibiting the production of IL-2, IL-18, IL-12. And other cytokines (Couper et al 2008).

Measurement of the level of cytokinetics (IL-36) in serum blood

The results of the current study showed a statistically significant ($p \leq 0.001$) high significant increase in IL-36 levels of psoriasis patients compared to the control group, as the mean values

were (513.59 ± 483.25) pg / ml, (179.16 ± 217.59) , respectively. Table

Table (4-4) shows the level of IL-36 for psoriasis patients and the control group

		Study groups	
		Patients	Control
IL36alpha	Mean	483.25	217.59
	Standard Deviation	513.59	179.16
	Median	274.00	149.00
	Percentile 25	210.00	98.00
	Percentile 75	485.50	308.00
P value		<0.001**	

** : highly statistical significance ($p < 0.001$)

These results are consistent with recent reports and the researcher's findings (Khalaf, 2015), who found that interleukin-36 IL-36 contributes to the pathogenesis of psoriasis, as it stimulates proinflammatory cytokines, a group of antimicrobial peptides, and mineral proteins. Of matrix metalloproteinases from reconstituted human skin (**Johnston et al , 2011**)

It is also possible that interleukin-36 (IL-36) is able to initiate dermatitis in psoriasis patients in the absence of adaptive immunity in association with TNF- α . This may partly explain the observation that gene overexpression of the IL-36a protein at the basic level of keratinocytes could lead to psoriasis-like inflammation in mice with lymphocyte deficiency (**Blumberg et al 2017**). The inflammatory precursor cytokines, which are predominantly produced by immune cells, and the IL-36 cytokines that are expressed conventionally, mainly in skin (Ma et al., 2008). Interestingly, we found that IL-36 cytokines had a stimulating effect. Antimicrobial peptides by IL-17A or TNF- α , suggesting that Th17 and IL-36 cytokines could enhance a similar response in keratinocytes (Wolk et al 2009) Several studies have highlighted the importance of early IL-36 production in the development of psoriasis

(Lande et al, 2007 •Kahlenbergand Kaplan ,2013)

and have been observed for epidermal colonization of Staphylococcus, which have a role in stimulating psoriasis development by inducing Th17 cells, (Chang et al 2018) which Role in inducing IL-36 production in(Nakagawa et al , 2017•Liu et al 2017)keratinocytesIL-36 induces various pro-inflammatory cytokines in endocytic cells, such as neutrophilic chemo-attractants

(CXCL1, CXCL2, and CXCL8) (Hashiguchi et al ,2018) and antimicrobial peptides (Nguyen et al 2012) Cytokines IL-17, IL-23 and IL-22 are closely related to interleukin IL-36 in human-induced psoriasis (Boutet et al , 2016) , as IL-17 and IL-22 stimulate the production of IL-36 in keratinocytes (44, 43) Several studies have shown that IL-36 responses are elevated in PsV skin PsV(Swindell et al 2018, Mahil et al 2017 ,D'Ermeet al ,2015 , Quaranta et al , 2014). In addition, we have shown that IL-36 stimulates chemokine production and amplifies the effects of IL-17 signaling in psoriasis lesions (Mahil et al 2017). Finally, animal studies have demonstrated that IL-36 enhances the activation of dendritic cells and T-lymphocyte polarization in Th17 cells (Tortola et al 2012) . Thus, the mechanisms by which IL-36 contributes to dermatitis has been extensively investigated. However, its systemic effects are still not well understood

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