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ORIGINAL ARTICLE

Serum Interleukin-6 levels in plaque type psoriasis

Varun Tyagi

Associate Professor, Department of Dermatology (DVL), Saraswathi Institute of Medical Sciences, Hapur, Uttar Pradesh, India

ABSTRACT:

Background: To study the serum levels of interleukin-6 in plaque type psoriasis. **Materials & methods:** A total of 40 subjects were enrolled. The age of patients included was 30 to 60 years. Blood samples were collected and centrifuged. Serum samples were collected. Patients were off topical treatment for 4 weeks prior to the PASI score evaluation and blood sample collection. Serum levels with IL-6 were measured using ELISA kit. Result was analysed using SPSS software. The statistical significance was accepted when $p < 0.05$. **Results:** The cytokines investigated in this study were detected in serum samples from all patients whereas IL-6 were below the minimum detection level of the kit in healthy controls. **Conclusion:** Serum levels of IL-6 were not correlated with PASI.

Keywords: Interleukin-6, psoriasis, PASI.

Corresponding Author: Varun Tyagi, Associate Professor, Department of Dermatology (DVL), Saraswathi Institute of Medical Sciences, Hapur, Uttar Pradesh, India

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INTRODUCTION

Psoriasis is a chronic immunologically based inflammatory skin disease which affects approximately 2% of the general population. [1, 2] Keratinocytes of the psoriatic skin are prematurely differentiated, as evident in the incomplete cornification of the stratum corneum, characterized by the retention of nuclei (i.e. parakeratosis) and the loss of the granular layer. The stratum corneum of psoriasiform skin is also thickened (i.e. hyperkeratosis). This heavy disruption of epidermal differentiation and skin barrier homeostasis coupled with altered levels of intercellular adhesion molecules result in the widespread scaling of psoriatic lesions. [3]Psoriasis is a chronic inflammatory skin disease that can be associated with other systemic disorders like cardiovascular disease, metabolic syndrome, and inflammatory bowel disease.[4] The financial and psychological impacts lead to anxiety and depression especially in individuals with active professional and social lives. [5] The predominant clinical presentation of psoriatic lesions is characterized by the formation of scaly, well-demarcated erythematous plaques due to hyperproliferation of keratinocytes. [6]

Increased production of interleukin-(IL-) 6 is well known in psoriasis andPsA.[7] Mice with epidermal overexpression of IL-6 (K14-IL-6 transgenic mice) exhibit a psoriasis phenotype.[8] The transcription factor signal transducer and activator of transcription 3 (STAT3) is upregulated in psoriasis. IL-6, which induces STAT3 phosphorylation, is also thought to be a potential therapeutic target.[9] In addition, serum IL-6 levels correlate with PsA disease severity.[10] IL-6 is thought to have similar roles in inflammatory arthritis associated with both RA and PsA. This supports the notion that targeted treatments against IL-6 might be effective. [11] Hence, this study was conducted to evaluate the serum levels of interleukin-6 in plaque type psoriasis.

MATERIALS & METHODS

A total of 40 subjects were enrolled. The age of patients included was 30 to 60 years. Blood samples were collected and centrifuged. Serum samples were collected. Psoriasis Area and Severity Index (PASI) assessment was used to grade the disease activity of patients with psoriasis at the time of blood collection as mild (0–10), moderate (11–20), and severe (>20). Patients were off topical treatment for 4 weeks prior to the PASI score evaluation and blood sample collection. Serum levels with IL-6 were measured using ELISA kit. Result was analysed using SPSS software. The statistical significance was accepted when $p < 0.05$.

RESULTS

A total of 40 subjects were enrolled. The cytokines investigated in this study were detected in serum samples from all patients whereas IL-6 were below the minimum detection level of the kit in healthy controls.The levels of IL-6 in mild, moderate and severe cases were 4.7 whereas in control groups was 7.5 pg/ mL.

Table: serum cytokines in healthy and psoriasis subjects

Cytokines (pg/mL)	Patients Mild (n=5)	Moderate (n=20)	Severe (n=5)	Control (n=10)
IL-6	4.7	4.7 (4.6-11)	4.7 (4.6-468)	7.5 (7.5-55.8)

DISCUSSION

The precise mechanisms by which IL-6 blockade leads to improvements in RA are not well understood.[12] IL-6 promotes synovitis by inducing neovascularization, infiltration of inflammatory cells, and synovial hyperplasia. IL-6 also causes bone resorption by inducing osteoclast formation via the induction of the receptor activator of NFκB ligand (RANKL) in synovial cells and cartilage degeneration by inducing the production of matrix metalloproteinases in synovial cells and chondrocytes. Moreover, IL-6 is involved in autoimmunity by altering the balance of Th17 cells by inducing the differentiation of Th17 cells from naïve CD4+ T cells. IL-6 blockade inhibits type II collagen-induced arthritis and requires CD4 T cells, which leads to the production of anti-type II collagen IgG.[13] However, another arthritis model, anti-type II collagen antibody-induced arthritis, bypasses the priming phase of T-cell-dependent antibody generation and is not suppressed in IL-6−/− mice.[14] These findings indicate that IL-6 is involved in the priming phase of RA but not in the effector phase of RA. Therefore, the major mechanism of TCZ is inhibiting the immune activation that leads to the development of RA. [15]Hence, this study was conducted to evaluate the serum levels of interleukin-6 in plaque type psoriasis.

In the present study, a total of 40 subjects were enrolled. The cytokines investigated in this study were detected in serum samples from all patients whereas IL-6 were below the minimum detection level of the kit in healthy controls. A study by Ogata A et al, studied that psoriatic arthritis (PsA) is a clinical manifestation of psoriatic disease. Although the pathogenesis of PsA remains unknown, PsA can be managed by treatments similar to those used for rheumatoid arthritis (RA). Because interleukin-(IL-) 6 has been suggested to have a pathogenic role in PsA, a humanized anti-IL-6 receptor antibody tocilizumab treatment for PsA was recently tried. However, the efficacy of tocilizumab for PsA was not favorable. They suggested that the pathogenic roles of IL-6 in PsA and RA are different. In RA, tumor necrosis factor (TNF) primarily contributes to the arthritis effector phase and IL-6 contributes to the arthritis priming phase. In PsA, the TNF-related effector phase is similar to that in RA, but the IL-6-related priming phase might not be critical. [16]

In the present study, the levels of IL-6 in mild, moderate and severe cases were 4.7 whereas in control groups was 7.5 pg/ mL. Another study by de Oliveira PS et al, showed that psoriasis is a chronic inflammatory skin disease characterized by alterations in cytokines produced by both Th1 and Th17 pathways. They evaluated serum levels of pivotal cytokines and correlate them with clinical parameters. Serum samples from 53 psoriasis patients and 35 healthy volunteers, matched by the proportion of sex and age ratios, were collected for ELISA cytokine detection. Psoriasis Area and Severity Index (PASI) was assessed at the time of sampling in psoriasis patients. Their findings demonstrated that IL-17A, IL-22, and IL-6 serum concentrations were significantly higher in psoriasis patients than in the control group. No statistical correlation could be found between cytokines concentrations, PASI score, and age. Although their results do not show any correlation between serum levels of IL-17A, IL-22, and IL-6 and disease activity, they confirms that there were increased in Brazilian psoriasis patients in comparison to healthy volunteers.[17]In a recent study Cordiali-Fei et al. [18] showed increased IL-6 serum levels in patients before biological therapy, but no correlation with PASI was attempted. Elango and colleagues reported a positive correlation with only two components of PASI, namely, infiltration and desquamation. [19]

CONCLUSION

Serum levels of IL-6 were not correlated with PASI.

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