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Serum levels of proinflammatory cytokines in psoriasis patients from Saudi Arabia

Psoriasis vulgaris is a multifactorial, chronic, inflammatory skin disease of unknown etiology. The characteristic histologic features of the disease are epidermal hyperproliferation and infiltration of both dermis and epidermis by inflammatory cells including neutrophils, lymphocytes, macrophages and mast cells. Interactions between infiltrating T cells and skin resident cells (keratinocytes, fibroblasts, endothelial cells) are often mediated by the synthesis and release of different proinflammatory cytokines[1]. Although the cytokine-mediated response is an essential part of the natural protective mechanism, excessive production of proinflammatory cytokines, or production of cytokines in the wrong biological context are associated with the pathology in a wide range of diseases including psoriasis. At the present time, one of the main areas of research in the psoriasis field concerns the role of cytokines in the pathogenesis of this disease.

Different cytokines play a part in sustaining the two main characteristics of a psoriatic lesion; keratinocyte hyperproliferation and inflammation.[1] Interleukin-2 (IL-2), interleukin-6 (IL-6), interleukin-8 (IL-8) and tumor necrosis factor-alpha (TNF-α) are the hallmark cytokines in a psoriatic cytokine network. Several investigators have suggested the possible use of TNF-α, IL-6, IL-8 and soluble interleukin-2 receptor (IL-2R) as markers of disease severity in psoriasis.[2–5] Sagawa *et al.*[6] pointed out that TNF-α in combination with other cytokines like IL-6 may be highly injurious due to complex interactions between these cytokines, suggesting a rationale for monitoring of multiple cytokines in the sera of psoriatic patients. Moreover, the cytokine assay results may vary due to the clinical stage and type of disease, methods used for cytokines detection and their sensitivities, lesion activity, interferences due to different drugs used, demographic differences in the patient groups, and the effect of concomitant pathologies.[2]

This preliminary study compares the serum levels of TNF-α, IL-6, IL-8 and IL-2R between 29 psoriasis patients and 25 age- and sex-matched healthy controls from the Saudi population. All the patients were untreated, both locally and systemically, for at least 14 days before enrolment. It was also ensured that control subjects had no medication during the 2 weeks before blood sampling. Blood samples (6 ml) were collected from patients and control subjects in serum separator vacutainers (BD Vacutainer Systems, Plymouth, UK). Sera were separated and immediately stored at −80 °C until analysis by commercially available enzyme-linked immunosorbent assay (ELISA) kits (R & D Systems, Minneapolis, MN).

Our results showed significantly higher levels of serum cytokines in Saudi psoriasis patients as compared with healthy controls (Table 1), which is in agreement with earlier studies demonstrating a significant increase in IL-6, IL-8, IL-2R and TNF-α in sera or skin blister fluids of psoriasis patients from different ethicities.[3,5,7–10] The maximal changes were observed in the levels of IL-6 (543% increase) followed by IL-8 (126%), IL-2R (77%) and TNF-α (76%). It has been suggested that proinflammatory cytokines not only play a fundamental role in the worsening of the disease or activating its pathogenetic mechanisms, but are also directly related to the clinical symptoms and disease evolution after effective therapy.[2] Earlier studies have observed a significant reduction of IL-6, IL-8, IL-

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Table 1 Serum cytokines in psoriasis patients and healthy controls

Cytokines	Psoriasis ($n = 29$)	Control ($n = 25$)	P
IL-6	37.5 ± 2.09	5.8 ± 0.79	**
IL-8	14.9 ± 1.75	6.7 ± 0.76	**
IL-2R	505.0 ± 43.2	286.0 ± 28.0	**
TNF-α	14.8 ± 2.84	8.4 ± 0.72	*

Values are mean ± SE (pg/ml of serum).
*P < 0.05 and **P < 0.001 vs. control (t-test).

2R and TNF-α levels following effective therapies in psoriasis patients.[2,4] We conclude that serum of proinflammatory cytokines are significantly increased in Saudi psoriasis patients, and an array of these cytokines may be considered as useful follow-up marker for monitoring of psoriatic patients and optimizing therapeutic strategies. However, detailed time-course studies on sequential analysis of these cytokines in relation to disease severity and/or treatment modalities are warranted to ascertain its real application.

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Mondor's phlebitis of penis following recurrent candidal balanoposthitis

Dear Sir,

A 35-year-old married serviceman presented to dermatology outpatients with a cord-like structure over the penis of 5 days' duration and small erosions over the prepuce of 7 days' duration. He complained of recurrent superficial erosions over the prepucial mucosa and glans with erythema and maceration following sexual intercourse with his wife since 2 months. These lesions used to subside after a topical application and abstaining from sex for 4–5 days. He denied any urinary complaint or extramarital contact. During a present episode, 2 days after sexual contact, he developed an asymptomatic firm cord-like swelling approximately 3 mm in thickness, almost encircling the penis just proximal to corona (Fig. 1). His wife also complained of thick curdy white vaginal discharge with itching; examination revealed erythema and maceration of external genitalia and vagina with a normal cervix.

There was no regional lymphadenopathy; and a cutaneous and systemic examination did not reveal any abnormality. Complete blood count and urine microscopy were within normal limits. Serology for HIV and syphilis (Venereal Disease Research Laboratory test) was nonreactive in both partners. Scraping from the erosions and vaginal discharge showed budding yeast cells and culture grew *Candida albicans* in both.

A diagnosis of Mondor's phlebitis with *Candidal balanoposthitis* and *Candidal vulvovaginitis* was made in the patient and his wife, respectively. Both were treated with oral Fluconazole 150 mg stat and topical 1% Clotrimazole cream, and advised abstinence for 2 weeks. At 2 weeks the candidiasis

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