A study of serum cortisol levels in psoriasis and its association with the severity of the disease

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SN- Concept, and design of the study results interpretation, review of the literature and preparing the first draft of the manuscript. AK- Concept, and design of the study, results interpretation, review of the literature, and preparing the first draft of the manuscript. SSKG- Concept, and design of the study, statistical analysis and interpretation, revision of the manuscript

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Abstract

Background: Psoriasis is a chronic inflammatory, skin disease that is characterized by hyper-proliferation of keratinocytes and inflammatory infiltration in the epidermis and dermis. Genetic, environmental, and hormonal factors have been implicated inthe etiopathogenesis of the disease. Glucocorticoids (GC), released as cortisol is a stress-response hormone with potent immunoregulatory mechanisms. It was hypothesized that loss of effective skin-GC synthesis would form a pathogenic mechanism of psoriasis.

Objective: The present study was undertaken to compare the levels of Cortisol in psoriasiscases and healthy controls and to study the role of stress in the disease.

Materials and methods: A case-control study was done, in which 50 patients with psoriasis (Group 1) and 50 healthy controls (Group 2) were included. Group 1 was further divided into three subgroups, mild cases (n=15) and moderate cases (n=25), and severe cases (n=10) based on PASI score. Based on PSS4, group 1 was further divided into stressors (n=20) and nonstressors (n=30). Serum Cortisol was estimated by CLIA in all the cases and controls.

Results: There was a significant difference in the cortisol levels of cases and controls. Cortisol was lower in the cases when compared to the control group (table 1). Cortisol levels in the subgroups of group 1 were presented in table 2. There was no significant difference in the cortisol levels among subgroups. Cortisol levels in the subgroups of group 1 based on PSS4 were presented in table 3. There were significantly higher cortisol levels in non-stressors than in stressors.

Conclusion: According to the results of the present study, it may be suggested that Cortisol may play a role in the etiopathogenesis of psoriasis. It can also be concluded that patients with psoriasis, and in particular those whose disease appears to be stress-responsive, exhibitan altered HPA response to stress. It can be emphasized that stress influences disease outcomes in patients with psoriasis by affecting cortisol levels at moments of high stress.

Key words: Psoriasis, stress, cortisol.

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INTRODUCTION

Psoriasis is a chronic inflammatory skin disease affecting 2% of population worldwide. [1] Psoriasis is characterized by well defined red, scaly plaques, typically located on the scalp, knee or elbows. [2] It can have a significant negative impact on the physical, emotional, and, psychosocial wellbeing of affected patients. [3] Studies have shown that patients with psoriasis are at greater risk of cardiovascular disease (CVD), metabolic syndrome (MetS), type 2 diabetes mellitus, dyslipidemia, hypertension, and obesity [4].These disease associations may be due to the systemic infla-

mmatory mediators generated in psoriasis, shared risk factors (i.e, smoking, alcohol consumption), or treatment.[1] The worldwide

array of cytokines by activated T cells, inflammatory cells, and keratinocytes. The characteristic lesionof psoriasis is due to the hyper-proliferation of the keratinocyte.[8]

Cortisol , one of the steroid hormones is the mammalian principal gluco corticoid hormone synthesized in zona fasciculata of adrenals. [9] Under normal conditions, cortisol secretion during acutestress response serves to mobiliz-

e glucose reserves for energy, inhibit pain and non–vital organ systems, and promote an adaptive fight-or-flight response. [10] Stress repeatedly resulted in altered HPA axis activit-

y with decreased cortisol levels in patients with psoriasis , suggesting that altered cortisol levels might act as an HPA axis mediator in this stress disease process of chronic inflammation

,[11]

However, evidence has shown that the main physiological pathways involve the hypothala-

mic– pituitary–adrenal (HPA) axis, sympathet-

ic–adrenal–medullary (SAM) axis, peripheral nervous systemand immune mediated pathw-

ays.[12]

Methods: 5 ml of venous blood was collected from patients.Serum cortisol was estimated by Chemiluminiscent Immunoassay (CLIA) in Siemens Advia Centaur XPT.

Ethical considerations: The study protocol was approved by the institutional human ethical committee. Confidentiality of data was maintained. Written informed consent was obtained from all the participants prior to the study.

burden of psoriasis is 2%, and the prevalence of psoriasis in India ranges from 0.44% to 2.8% [5], Whilesome studies indicated the average age of onset for psoriasis was 33 years of age, and 75% of cases occurred before 46 years of age.[6] Henseler et al., suggested that the onset of psoriasis was bimodal with two peaks of the disease – the first between 16 and 22 and the second between 57 and 60 years of age. [7] Complete cure for psoriasis does not exist to date; therefore, treatment is mainly suppressive in natureand is aimed at achieving a period of remission or alleviating the severity of the disease.[2] Genetic factors play a significant role in the pathogenesis of psoriasis. It has been hypothesized that the disease starts with the activation of T cell by an unknown antigen, which leads to secretion of an

Materials and methods

Study design: Case-control study

Study setting: Department of Biochemistry, Osmania General Hospital, Hyderabad.

Study participants: The study included 100 subjects who were divided into 2 groups. Group 1 with 50 patients newly diagnosed with psoriasis as cases and Group 2 with 50 patients healthy subjects as controls

The clinical severity of psoriasis can be assessed by using the psoriasis area severity index (PASI) scoreStress levels of Patients are classified based on the perceived stress scale 4(PSS4).

Inclusion criteria: patients in all age groups suffering from psoriasis attending the dermatology department in Osmania general hospital were included in the study.

Exclusion criteria: Patients with renal disorders and cardiovascular disorders, autoi-

mmune Disorders, pregnant and lactating women and history of smoking and alcohol intake , Patients on drugs like systemic corticosteroids were excluded from the study.

Statistical analysis: The data were analyzed

using SPSS software version 23. The results were expressed as mean andSD of various parameters in different groups The results were expressed in µg/dl for serum Cortisol. Student t-test and one-way ANOVA was used to determine the significance of the difference between the groups.

Results: There was a significant difference in the cortisol levels of cases and controls. Cortisol was lower in the cases when compared to the control group (table 1). Cortisol levels in the subgroups of group 1 were presented in table 2. There was no significant difference in the cortisol levels among subgroups. Cortisol levels in the subgroups of group 1 based on PSS4 were presented in table 3. There were significantly higher cortisol levels in non-stressors than in stressors.

Table 1: Cortisol levels in cases and controls

Parameter	Cases	Controls	P value
Cortisol (µg/dl)	6.43 ± 2.42	7.60 ± 2.04	0.011*

Data were presented as mean and SD (*P<0.05 is significant)

Table 2: Cortisol levels in the subgroups of group 1 based on PASI

Parameter	Mild (n=15)	Moderate (n=15)	Severe (n=10)	P value
Cortisol (µg/dl)	5.70±1.35	7.052±3.14	5.98±0.67	0.189

Data were presented as mean and SD

Table 3: Cortisol levels in the subgroups of group 1 based on PSS4

Parameter	Non-stressors (n=30)	Stressors (n=20)	P value
Cortisol (µg/dl)	7.057±2.91	5.495±0.79	0.024*

Data were presented as mean and SD (*P<0.05 is significant)

Discussion:

Psoriasis is a papulo squamous disease with variable morphology, distribution, severity, and course; classically very well circumscribe-

d, circular, red papules or plaques with a grey or silvery-white, dry scale.[3] In addition, the lesions are typically distributed symmetrically on the scalp, elbows, knees, lumbosacral area, and in the body folds.[3] Psoriasis may also develop at the site of trauma or injury, known as Koebner's phenomenon. A hypopigmented ring (Woronoff ring) surrounding individual psoriatic lesions may be seen [13]. The amount of scaling varies from patient topatient.[14] The successful removal of scales reveals a glossy, a red membrane with small bleeding points (Auspitz sign).[14] Whether psoriasis represents a fundamental disease of the skin or the immune system, has been debated for several years.[8]

Psoriasis is an immune-mediated genetic skin disease.[15] The underlying pathomechanisms involve complex interactions between the innate and adaptive immune systems. Role of T cells, antigen-presenting cells, keratinocytes, Langerhans' cell, macrophages, natural killer cells, an array of Th1 type Cytokines, certain growth factors like vascular endothelial growth factor (VEGF) keratinocyte growth factor (KGF) and others have been suggested to play a key role in the pathogenesis of psoriasis.[8]

A blunted cortisol response to acute stress has been demonstrated to lead to an upregulation of proinflammatory cytokines.[16] As a stress response involves activation of both the hypothalamic– pituitary–adrenal (HPA) axis and the autonomic nervous system, which interact with the immune system, stressful events could contribute to the maintenance and exacerbations of chronic inflammatorydiseases such as psoriasis. Specifically, experimental stress repeatedly resulted in altered HPA axis activity with decreased cortisol levels in patients with psoriasis,[19] suggesting that lowered cortisol levels might act as an HPA axis mediator in this stress–disease process of chronic inflammation.

A study conducted by Evers A et al (2010) concluded that patients who persistently experienced higher levels of daily stressors, and are possibly more stress reactive, had relatively lower mean cortisollevels suggests that the HPA axis is hypoactive in these individuals, which might directly influence the disease-related

outcome.[11] A case-control study done by Richard et al(2005) showed that patients with highly stress responsive psoriasis had significantly lower salivary cortisol levels at baseline ('P' < 0.01) and lower serum cortisol levels following the social performance stressor ('P' = 0.016) than patients with nonstress responsive disease[17]

Conclusion: According to the results of the present study, serum Cortisol seems to have a role in the etiopathogenesis of psoriasis. Furthermore, variation of serum cortisol levels in subgroups based on PSS4 (stressors and nonstressors) of psoriasis may indicate that daily stress influencesdisease outcomes in patients by affecting cortisol levels at moments of high stress. Furthermore, patients with persistently high levels of stress seem to have a specific psychophysiological profile of lowered cortisol levels and may be particularly vulnerable to the influence of stress on their psoriasis

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