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ORIGINAL ARTICLE

ADA and hsCRP level in patients with psoriasis

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ABSTRACT:
Background: Hyperproliferation of keratinocytes is a hallmark of psoriasis, a chronic inflammatory autoimmune disease with a complex pathophysiology that includes environmental and genetic variables. The present study was conducted to assess serum ADA and hsCRP level in psoriasis patients. **Materials & Methods:** 70 patients of Psoriasis of both genders were selected. According to their PASI score, psoriatic patients were classified as mild if their score was less than 10, moderate if their score was between 10 and 20, and severe if it was greater than 20. There were thirty-five healthy participants in the control group. Patients with psoriasis had their serum ADA and hsCRP levels tested. **Results:** Group I had 17 males and 19 females and group II had 18 males and 16 females. The difference was non- significant (P> 0.05).ADA level was 28.2 U/L in group I and 9.1 U/L group II. The mean hsCRP level in group I was 59.0 ng/ml and 10.2 ng/ml in group II. The difference was significant (P< 0.05). **Conclusion:** Compared to healthy controls, psoriatic patients showed higher levels of serum ADA and hsCRP.
Keywords: keratinocytes, psoriatic, adenosine deaminase

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INTRODUCTION

Hyperproliferation of keratinocytes is a hallmark of psoriasis, a chronic inflammatory autoimmune disease with a complex pathophysiology that includes environmental and genetic variables. The breakdown of adenosine from meals and the turnover of nucleic acids in tissues depend on the enzyme adenosine deaminase (ADA), which is engaged in purine metabolism. It is regarded as an indicator of T-cell activation that is not specific. There are 3 other less-frequently observed variants of psoriasis: guttate psoriasis, erythrodermic psoriasis, and pustular psoriasis.[1] Guttate psoriasis comprises 2% of psoriasis cases and is characterized by multiple 3- to 5-mm confetti-like, pink scaly patches. Approximately 66% of new-onset guttate psoriasis is preceded by an upper respiratory tract infection such as streptococcal infection, and most of these cases resolve spontaneously in weeks to months but can become chronic.[2]

High levels of ADA were found in the epidermis of psoriatic patients, and these levels were associated with hyperproliferative keratinocyte states with prominent DNA synthesis. Furthermore, psoriatic patients had higher plasma ADA activity than controls, and this activity dropped following propylthiouracil (PTU), PUVA, or cyclosporine treatment.[3]

The breakdown of adenosine from meals and the turnover of nucleic acids in tissues depend on the enzyme adenosine deaminase (ADA), which is engaged in purine metabolism.[4] It is regarded as an indicator of T-cell activation that is not specific. An essential laboratory metric for tissue damage, infection, and inflammation is C-reactive protein (CRP). Compared to the usual CRP assay, high-sensitive CRP (hsCRP) can detect lower levels of CRP.[5] Numerous skin conditions, such as psoriasis, mycosis fungoides, hidradenitis suppurativa, and allergic contact dermatitis, are associated with elevated hsCRP. Active arthritis, the psoriasis area severity index (PASI) score, and a higher prevalence of cardiovascular illnesses were all associated with elevated CRP in psoriatic patients.[6]The present study was conducted to assess serum ADA and hsCRP level in psoriatic patients.

MATERIALS & METHODS

The present study comprised of 70 patients of Psoriasis of both genders. All enrolled patients gave their written consent for the participation in the study.

Data such as name, age, gender etc. was recorded. According to their PASI score, psoriatic patients were classified as mild if their score was less than 10, moderate if their score was between 10 and 20, and severe if it was greater than 20. There were thirty-five healthy participants in the control group. Patients with psoriasis had their serum ADA and hsCRP levels tested. Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Gender	Group I	Group II	P value
Male	17	19	0.94
Female	18	16	

Table I, graph I shows that group I had 17 males and 19 females and group II had 18 males and 16 females. The difference was non- significant (P> 0.05).

Graph I Distribution of patients

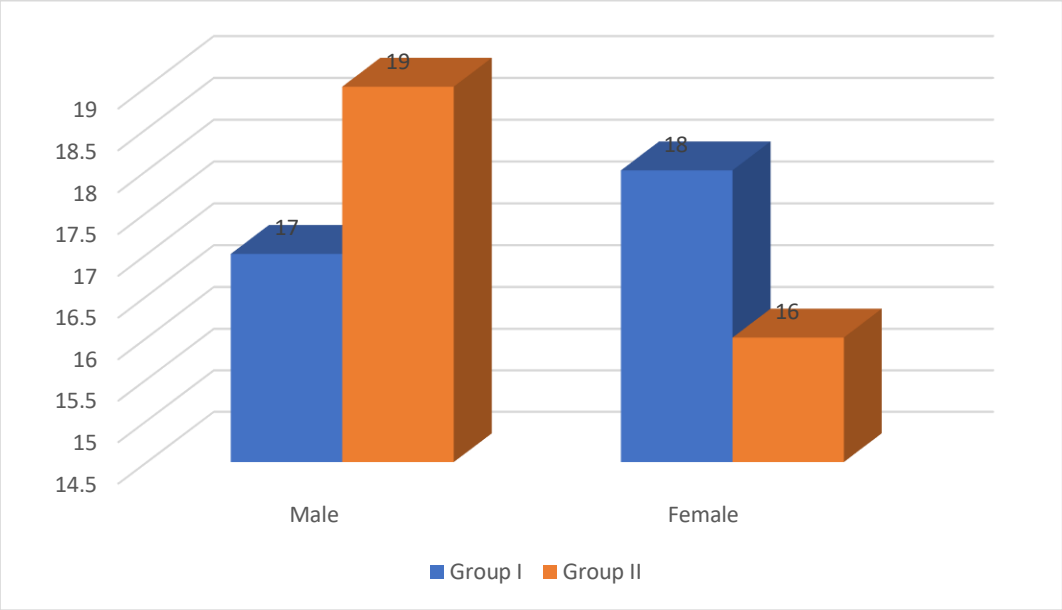
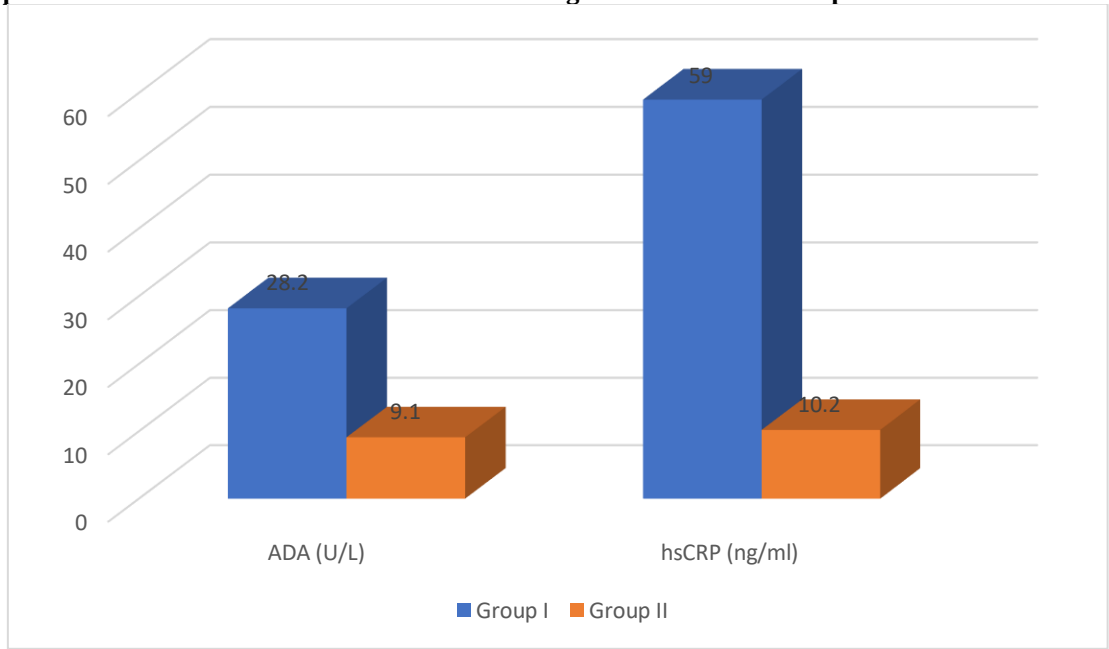


Table II Measurement of adenosine deaminase and high-sensitive C-reactive protein level

Parameters	Group I	Group II	P value
ADA (U/L)	28.2	9.1	0.02
hsCRP (ng/ml)	59.0	10.2	0.01

Table II, graph II shows that ADA level was 28.2 U/L in group I and 9.1 U/L group II. The mean hsCRP level in group I was 59.0 ng/ml and 10.2 ng/ml in group II. The difference was significant (P< 0.05).

Graph I Measurement of adenosine deaminase and high-sensitive C-reactive protein level



DISCUSSION

About 3% of Americans and an estimated 125 million individuals worldwide suffer from psoriasis, a chronic, immune-mediated skin condition. Over 80% of psoriasis cases are plaque psoriasis, making it the most prevalent type.[7] Erythematous scaly patches or plaques, which are typically found on extensor surfaces but can also affect the intertriginous areas, palms, soles, and nails, are the hallmark of plaque psoriasis.[8] Men and women are equally affected by psoriasis, and adults are more likely than youngsters to have it. The pathophysiology, genetics, comorbidities, and biologic therapies of plaque psoriasis have seen the fastest progress.A feed-forward process of inflammation, mainly the T-helper cell type 17 (TH17) pathway, is involved in the pathophysiology of plaque psoriasis.[9] Environmental variables can aggravate psoriasis, whereas genetic factors are crucial in its development. Guttate psoriasis, erythrodermic psoriasis, and pustular psoriasis are further morphologic variations of psoriasis. Depending on the type of psoriasis, different clinical characteristics exist. Plaque, guttate, erythrodermic, and pustular psoriasis are some of the variations of psoriasis.[10] A human may have multiple variants at any given time, even though one version usually predominates in an individual. Erythema, thickness, and scale are the three main clinical characteristics shared by the majority of psoriasis types.[11]The present study was conducted to assess serum ADA and hsCRP level in psoriatic patients.

We found that graph I shows that group I had 17 males and 19 females and group II had 18 males and 16 females. Hashemi M et al[12] assessed psoriatic patients' serum trypsin inhibitory capability, total antioxidant capacity, and adenosine deaminase activity levels. Psoriatic patients (n = 40) and controls (n = 46) participated in the study. The psoriasis area and severity index, or PASI, was used to grade the patients. The Aguisti and Galanti method was used to quantify the blood ADA activity, and an enzymatic test was used to measure the serum trypsin inhibitory capacity (sTIC). Additionally, ferric reducing ability of plasma was used to determine serum total antioxidant capacity. Compared to the healthy control, the psoriatic patients' serum ADA activity was considerably higher ($P < 0.001$). Additionally, we discovered that patients had a considerably stronger trypsin inhibitory capacity than the control group ($P < 0.001$). Total antioxidant capacity of plasma was significantly lower in psoriatic patients than in healthy controls ($P = 0.025$). There were no significant correlations among ADA, TAC and TIC.

We observed thatADA level was 28.2 U/L in group I and 9.1 U/L group II. The mean hsCRP level in group I was 59.0 ng/ml and 10.2 ng/ml in group II. Bukulmez et al[13]analyzed the relationship between serum ADA activity and disease activity in psoriasis. Using an enzymatic approach, ADA activity was assessed in 15 healthy participants and 25 psoriasis sufferers. Ten individuals had these values taken again following cyclosporin A or PUVA treatment. The PASI scoring system was used to measure disease activity. Patients with psoriasis had significantly higher serum ADA levels than healthy participants ($p<0.05$). After treatment, the same patients' ADA levels were significantly lower than their pretreatment levels ($p<0.05$). The PASI scores and ADA levels did not correlate. These findings bolster the notion that psoriasis etiology involves T cell activation and that ADA may be helpful in the disease activity.

Yildırımet al[14] in their study Adenosine deaminase activities in both serum and erythrocytes were measured in 23 untreated patients with BD and in 20 healthy controls. The patients with BD were divided into two groups: active (n = 10) and inactive (n = 13).When compared with the control group, serum ADA activity was high ($P < 0.01$) and erythrocyte ADA activity was significantly low ($P < 0.01$) in BD. Serum ADA activity of active BD was higher than that of inactive BD ($P < 0.01$), but erythrocyte ADA activity was found to be lower in active BD than inactive BD ($P < 0.01$).

Isha et al[15] in their study serum CRP and uric acid levels were estimated in twenty-five patients with psoriasis (group III) before and after 12 weeks of treatment. Results were compared with a group of 25 normal subjects (group I) and a group of 25 patients of various skin diseases other than psoriatic lesion (group II). Mean value for CRP was found to be increased by more than 20 folds in patients with psoriasis, which was subsequently reduced to nearly 50% of the initial value after 12 weeks of treatment. These patients also showed hyperuricemia. Nearly 25% of these patients also exhibited arthritis. It is thus suggested that both CRP and uric acid levels should be monitored in patients with psoriasis.

The limitation of the study is small sample size.

CONCLUSION

Authors found that compared to healthy controls, psoriatic patients showed higher levels of serum ADA and hsCRP.

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