Evaluation of Serum Adenosine Deaminase and Inflammatory Markers in Psoriatic Patients

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Abstract

Background: Adenosine deaminase (ADA) is an enzyme involved in purine metabolism and it is a marker of nonspecific T-cell activation. Few studies have shown high levels of ADA in the epidermis and sera of psoriatic patients. Other inflammatory markers such as high-sensitive C-reactive protein (hsCRP), erythrocyte sedimentation rate (ESR), and serum uric acid (SUA) have shown correlations with psoriasis area severity index (PASI) score. The correlation between ADA and PASI score is still a matter of debate. Aims: The aim of this study was to evaluate serum ADA, hsCRP, SUA, and ESR in psoriatic patients and their correlation with PASI score. Patients and Methods: This study included 60 psoriatic patients divided according to PASI score into three groups (mild, moderate, and severe) each containing 20 patients. PASI score <10 was defined as mild, (10–20) moderate, and >20 severe. Twenty healthy subjects of matched age and sex were included as control. Serum ADA, hsCRP, SUA, and ESR were evaluated for patients and controls. Correlations of ADA, hsCRP, SUA, and ESR with PASI scores were done. Results: While ADA, hsCRP, SUA, and ESR showed a significant increase in psoriatic patients compared with that of the controls (P<0.001), they showed no significant difference between different psoriatic groups (P>0.05) and no correlations with PASI score (P>0.05). The frequency of joint affection increased with increasing severity of psoriasis (5%, 10%, and 25% in mild, moderate, and severe psoriasis, respectively). Conclusion: Serum ADA, hsCRP, SUA, and ESR showed higher levels among psoriatic patients than in controls. The increased ADA in psoriatic patients supports the role of T-cell activation and proliferative disorder in the pathogenesis of psoriasis. No significant correlations were found between these biomarkers and PASI score. Further studies are needed to validate these biomarkers as diagnostic and prognostic factors in psoriasis.

Key Words: Adenosine deaminase, psoriasis area severity index, psoriasis

Introduction

Psoriasis is a chronic inflammatory autoimmune disease characterized by hyperproliferation of keratinocytes with multifactorial pathogenesis including genetic, and environmental factors.[1] Adenosine deaminase (ADA) is an enzyme involved in purine metabolism and is essential for the breakdown of adenosine from food and the turnover of nucleic acids in tissues. It is considered as a marker of nonspecific T-cell activation.[2] The epidermis of psoriatic patients showed high levels of ADA which correlated with the hyperproliferative states of the keratinocytes with pronounced DNA synthesis.[3,4] In addition, plasma ADA activity was higher in psoriatic patients compared to controls and decreased after treatment with propylthiouracil (PTU), PUVA, or cyclosporine.[5,6] C-reactive protein (CRP) is an important laboratory parameter for tissue damage, infection, and inflammation. High-sensitive CRP (hsCRP) can detect lower levels of CRP than the standard CRP measurement.[7] Increased hsCRP is found in many skin diseases including allergic contact dermatitis, mycosis fungoides, hidradenitis suppurativa, and psoriasis.[8-12] Increased CRP in psoriatic patients was correlated with active arthritis, psoriasis area severity index (PASI) score, and with an increased...
incidence of cardiovascular diseases.\textsuperscript{[13]} Patients with increased hsCRP levels were found to have a better response to cyclosporine therapy.\textsuperscript{[14]} CRP in patients with psoriasis was found to be decreased after treatment with phototherapy either alone or in combination with coal tar, photochemotherapy, and tumor necrosis factor-\(\alpha\) inhibitors.\textsuperscript{[15,16]}

Several studies have found correlation between serum uric acid (SUA) level, and the severity of psoriasis and increased risk of cardiovascular mortality.\textsuperscript{[17,18]}

Erythrocyte sedimentation rate (ESR) increases with the severity of psoriasis pointing out the chronic inflammatory nature. It was found as a strong predictor for the presence of psoriatic arthritis (PsA).\textsuperscript{[19]} However, others showed variable ESR between psoriatics with and without subclinical arthritis.\textsuperscript{[20]}

The aim of this study was to evaluate serum ADA, hsCRP, SUA, and ESR in psoriatic patients and their correlations with PASI score.

**Patients and Methods**

This case–control study included 60 psoriatic patients divided according to PASI scores\textsuperscript{[21]} into three groups (mild, moderate, and severe) each containing 20 patients. PASI score <10 defined psoriasis as mild, between 10 and 20 as moderate, and >20 as severe.\textsuperscript{[22]} About 20 healthy subjects of matched age and sex were included as a control group. All the study populations were recruited from the Outpatient Clinic of Dermatology, Andrology and STDs Department, Mansoura University Hospital, Mansoura, Egypt. The Research Ethics Committee for experimental and clinical studies at Faculty of Medicine, Mansoura University, Mansoura, Egypt, approved the study. All participants signed an informed consent before being included in the study.

The selected psoriatic patients should not have history of systemic or topical steroid medication, methotrexate, biologics, or phototherapy treatment for at least 2 months before inclusion. The exclusion criteria included erythrodermic and pustular psoriasis, history of medical disorders that might affect the serum levels of ADA, CRP, such as lymphoid malignancies, infectious and noninfectious systemic diseases with chronic T-cell activation such as pulmonary and pleural tuberculosis, sarcoidosis, typhoid fever, and cutaneous anthrax. In addition, patients with connective tissue diseases, including progressive systemic sclerosis, morphea, dermatomyositis, and lupus erythematosus, were also excluded from the study.

All participants were subjected to history taking including age, special habits, drug history, and history of medical diseases with stress on age, duration, and joint affection.

A volume of 8 ml venous blood sample was withdrawn from every participant. One milliliter added to tube containing K2EDTA for complete blood count (CBC) and 1.6 ml blood was added to sodium citrated tube for ESR determination. The rest of the blood sample was allowed to clot for 15 min, centrifuged, and the serum was separated into two aliquots; one used for determination of SUA, and the other was stored at \(-20^\circ\text{C}\) for assay of both ADA and hsCRP.

CBC was measured by using CELL-DYN Emerald cell counter, ABBOTT, Germany. ESR was assayed using Westergren method. SUA was assayed by enzymatic colorimetric uricase method using SPINREACT, S. A/S. A. U Ctra. Santa Coloma, 7 E-17176 SANT ESTEVE DE BAS (GI) SPAIN according to the instructions of manufacturing company. The hsCRP was assayed by an enzyme-linked immunosorbent assay BIOS kit (Chemux BioScience, Inc., California, USA).

ADA was assayed through kinetic method using Ben Biochemical Enterprise (BEN) ADA quantitative ultraviolet assay kit, Milano, Italy. The principle of the assay was that ADA catalyzes the hydrolysis of the substrate adenosine with the liberation of ammonia that reacts with \(\alpha\)-ketoglutarate and NADPH with means of glutamate dehydrogenase giving NADP. The decrease of absorbance in NADPH is monitored at 340 nm, which is proportional to the concentration of ADA in the sample.\textsuperscript{[23]} The concentration of ADA was calculated using standard included in the kit.

**Statistical analysis**

Statistical analyzes were carried out using SPSS for Windows, release 20 (SPSS Inc., Chicago, Illinois, USA). Quantitative data were presented as mean \(\pm\) standard deviation or median and range, and qualitative data were presented as frequency and percentage. Chi-square and Fisher’s exact tests were used to determine the relationship between qualitative data. Quantitative data were compared with one-way ANOVA or Mann–Whitney U test. Independent \(t\)-test was used for comparing PASI level and laboratory results in groups with and without joint affection. Spearman’s correlation was used to assess the correlation of PASI scores and other variables in psoriatic patients. \(P<0.05\) was considered statistically significant.

**Results**

This study included 60 psoriatic patients classified according to PASI score into mild, moderate, and severe psoriatic group. Each group included 20 patients. Twenty healthy subjects were included as a control group. There were no statistically significant differences of age and sex between different psoriatic groups and the control group. Table 1 shows the demographic data of psoriatic patients and of the control group.
ADA, hsCRP, SUA, and ESR showed a significant increase in psoriatic patients as one group compared with the control group [Table 2].

ADA, hsCRP, SUA, and ESR showed a significant increase in each psoriatic group (mild, moderate, and severe) compared with the control group. No significant differences between ADA, hsCRP, SUA, and ESR were found among different psoriatic groups [Table 3]. Furthermore, no significant correlation between PASI score and ADA, hsCRP, SUA, and ESR was found [Table 4].

The frequency of joint affection increased with increasing severity of psoriasis (5%, 10%, and 25% in mild, moderate, and severe psoriasis, respectively) [Table 5].

CBC including hemoglobin (Hb) concentration, hematocrit percentage (HCT %), red blood cell (RBCs) concentration, total white blood cell count (WBCs), platelet count (PL), and mean PL volume (MPV) showed no statistically significant differences between different psoriatic groups and control group [Table 6].

### Discussion

Psoriasis is a chronic systemic disease with an immune-inflammatory etiology, affecting approximately 2%–3% of the world’s population, and characterized by T-cell-mediated hyperproliferation of keratinocytes.[24]

The results of studies evaluating ADA level in psoriatic patients are conflicting. Some studies agreed with us in reporting high ADA in the sera of psoriatic patients than that of the healthy controls.[6,25-28] In addition, Tikhonov et al.[3] found double activities of ADA and purine nucleoside phosphorylase (PNP) in the skin of psoriatic patients.

Many studies reported a decrease of serum ADA levels after psoriasis treatment with different modalities including cyclosporine, etanercept, PUVA, and PTU.[5,27] PTU, such as methotrexate, influences the metabolism of purines, and T lymphocyte functions. Based on these studies,[3,5] ADA activity was considered as an indicator of the role of purine metabolism and T-cell activation in the pathogenesis of psoriasis. On the contrary, one study found normal ADA activity in psoriatic patients.[29] This could be due to the few number of patients (only 18) and the use of a normal laboratory range instead of a control group in that study. In accordance with our results, Hashemi et al.[25] reported no significant differences of ADA among mild, moderate, and severe psoriatic groups.

Matched with our study, Bukulmez et al.[6] and Yildirim et al.[27] found no correlation between serum ADA and PASI score. In spite of the fact that PASI score is the most widely used measure for assessment of psoriasis,[30] it has a number of limitations including inter- and intra-observer variability. The lack of correlation between PASI score and ADA activity in this study might be due to the subjectivity of PASI score and the relatively small number of patients.

The hsCRP showed a significant increase in psoriatic patients as one group and in each psoriatic group (mild, moderate, and severe) compared with the controls [Tables 2 and 3, respectively]. This result is in agreement with many studies reporting higher CRP and hsCRP levels in psoriatic patients. They also found a decrease in their levels after treatment with different modalities indicating that hsCRP is a well-established biomarker for inflammation in psoriatic patients.[12,31-34]

However, in the present study, hsCRP showed no significant correlation with PASI score in contrast to other studies[32,33] that found a significant positive correlation between hsCRP and PASI score. Some found CRP to increase only in psoriatic arthritis (PsA).[36] The low incidence of patients with joint affection in this study, the differences in age, sex, number of patients, and severity of disease could explain this difference.
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Table 3: Comparison of adenosine deaminase, high-sensitive C-reactive protein, serum uric acid, and erythrocyte sedimentation rate between psoriatic groups and controls

| Group (n)   | Mild (20)       | Moderate (20)  | Severe (20)   | Controls (20) | P*   |
|-------------|-----------------|----------------|---------------|---------------|------|
| ADA (U/L)   | 20.7 (6.6-41.2) | 22.55 (3.5-47.2) | 25.95 (9.7-69) | 8.5 (3.3-16) |      |
| hsCRP (ng/ml)| 39.45 (9.7-180) | 53.3 (1.9-200)  | 72.15 (4.2-450) | 9 (1.5-15.4) |      |
| SUA (mg/dl) | 5.45 (2.3-9.8)  | 5.65 (1.5-8.5)  | 6 (2.7-8.3)   | 4.2 (1.5-6.5) |      |
| ESR (mm/h)  | 27.5 (7-72)     | 28 (6-55)       | 32 (8-72)     | 13.5 (6-20)  |      |

*Kruskal–Wallis test. P*: Comparison of each group with the control group, P*: Comparison of each group with the mild group, P*: Comparison between moderate and severe groups. hsCRP: High-sensitive C-reactive protein, SUA: Serum uric acid, ESR: Erythrocyte sedimentation rate, ADA: Adenosine deaminase

Table 4: Correlations of psoriasis area severity index score with laboratory results in psoriatic patients

| Lab results | PASI Mild (20) | PASI Moderate (20) | PASI Severe (20) | P*     |
|-------------|----------------|--------------------|------------------|--------|
| ADA         |                |                    |                  |        |
| r           | 0.183          | 0.147              | 0.142            |        |
| P           | 0.44           | 0.536              | 0.586            |        |
| hsCRP       |                |                    |                  |        |
| r           | 0.175          | −0.212             | −0.11            |        |
| P           | 0.461          | 0.37               | 0.645            |        |
| SUA         |                |                    |                  |        |
| r           | −0.05          | 0.176              | −0.068           |        |
| P           | 0.82           | 0.459              | 0.777            |        |
| ESR         |                |                    |                  |        |
| r           | 0.368          | 0.19               | 0.049            |        |
| P           | 0.11           | 0.423              | 0.838            |        |

r: Spearman correlation coefficient, ADA: Adenosine deaminase, hsCRP: High-sensitive C-reactive protein, SUA: Serum uric acid, ESR: Erythrocyte sedimentation rate, PASI: Psoriasis area severity index

Table 5: Distribution of joint affection according to psoriatic group

| Joint affection | Mild (20) | Moderate (20) | Severe (20) | P* |
|-----------------|-----------|---------------|-------------|----|
| Negative, n (%) | 19 (95)   | 18 (90)       | 15 (75)     | 0.153 |
| Positive, n (%) | 1 (5)     | 2 (10)        | 5 (25)      |     |

*Chi-square test

In agreement with our result, the elevation of SUA in psoriasis than in the controls was reported by many researchers,[34,36] and a history of psoriasis was associated with an increased risk of gout with increased SUA.[38] The increased ADA activity in our psoriatic patients could be a cause of increased SUA because it catabolizes adenosine to inosine which is further degraded to uric acid.[37] On the other side, increased SUA was a common finding in psoriatic patients,[36] which significantly decreased after the treatment of psoriasis.[34] In addition, patients with psoriasis and hyperuricemia showed marked improvement in psoriasis when treated for their hyperuricemia. Psoriasis, like gout, may be, at least partly, a result of a disorder of purine metabolism and monosodium urate crystals may be partially responsible for the cell proliferation that is characteristic of psoriatic plaques.[38]

Kwon et al.[39] found no significant difference between SUA of psoriatic patients and healthy population and reported a positive correlation of SUA with PASI score. This was in contrast to our results of significant increase of SUA in psoriatic patients without a significant correlation with PASI score. They attributed this result to the lower skin involvement in their patients that might not have been sufficient to induce hyperuricemia.[39]

In accordance with our results, many studies reported increased ESR in psoriatic patients than in controls.[40,41] The decrease of RBCs deformability and plasma levels of globulins and fibrinogen with the decrease of albumin could explain the increased ESR in psoriatic patients.[41,42]

In our study, the frequency of joint affection increased with increasing severity of psoriasis (5%, 10%, and 25% in mild, moderate, and severe psoriasis, respectively). [Table 5] This result supported other studies showing a higher risk of having arthritis with severe psoriasis.[43-45] The increased frequency of PsA in severe psoriasis is multifactorial. The larger affected body surface area leads to a higher systemic burden of the inflammatory response and wider port
of entry for the skin flora to interact with the immune system. These changes under the shared susceptibility genes and/or environmental factors eventually result in triggering of PsA.\textsuperscript{[46]}

In agreement with our result, Saleh et al.\textsuperscript{[47]} revealed no difference of mean platelet volume (MPV) between psoriasis patients and controls. However, Kim et al.\textsuperscript{[48]} found that MPV to be high in psoriasis than in the control group. They reported a positive correlation between MPV and PASI score with a significant decrease in MPV levels after the treatment of psoriasis. In addition, Bajaj et al.\textsuperscript{[49]} found that Hb content of RBCs in psoriatic patients was approximately 2–3 g/dl less than in controls and negatively correlated with PASI score. They suggested that chronic and continuous loss of iron through scaling, deficient nutrition, and less absorption through gut and bone marrow depression due to chronic inflammatory process might be contributing factors to the occurrence of anemia. The differences between these studies and ours could be due to different patient populations including number, age, sex, race, and severity of the disease.

**Conclusion**

Serum ADA, hsCRP, SUA, and ESR showed higher levels among psoriatic patients than healthy controls. The increased ADA in psoriatic patients supported the role of T-cell activation in the pathogenesis of psoriasis. No significant correlations were found between these biomarkers and PASI scores. Further studies with large numbers are needed to validate the value of these biomarkers as diagnostic and prognostic factors in psoriasis.

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Nil.

**Conflicts of interest**

There are no conflicts of interest.

**References**

1. Deng Y, Chang C, Lu Q. The inflammatory response in psoriasis: A comprehensive review. Clin Rev Allergy Immunol 2016;50:377-89.
2. Kelley WN, Daddona PE, van der Weyden MB. Characterization of human adenosine deaminase. Ciba Found Symp 1977;51:273-93.
3. Tikhonov IV, Markusheva LI, Toguzov RT. Metabolism of purine compounds in psoriasis. Klin Lab Diagn 1998;3:3-6.
4. Koizumi H, Iizuka H, Aoyagi T, Miura Y. Adenosine deaminase in human epidermis from healthy and psoriatic subjects. Arch Dermatol Res 1983;275:310-4.
5. Köse K, Utas S, Yazici C, Akdağ A, Kelleştimur F. Effect of propylthiouracil on adenosine deaminase activity and thyroid function in patients with psoriasis. Br J Dermatol 2001;144:1121-6.
6. Bukulmez G, Akan T, Ciliv G. Serum adenosine deaminase levels in patients with psoriasis: A prospective case-control study. Eur J Dermatol 2000;10:274-6.
7. Rifai N, Ridker PM. High-sensitivity C-reactive protein: A novel and promising marker of coronary heart disease. Clin Chem 2001;47:403-11.
8. Zinkevičienė A, Štaitiūnas A, Kainov D, Lastauskienė A, Kainov D, Lastauskienė A, Kaidanov D, Kaidanov D, Kaidanov D. Serum adenosine deaminase levels in patients with psoriasis: A pilot study. Int Arch Allergy Immunol 2015;168:161-4.
9. Cengiz FP, Emiroğlu N. Evaluation of cardiovascular disease risk factors in patients with mycosis fungoides. Ann Bras Dermatol 2015;90:36-40.
10. Miller IM, Ring HC, Prens EP, Rygtaard H, Mogensen UB, Ellervik C, et al. Leukocyte profile in peripheral blood and neutrophil-lymphocyte ratio in hidradenitis suppurativa: A Comparative cross-sectional study of 462 cases. Dermatology 2016;232:511-9.
11. Gerkowicz A, Pietrzak A, Szepietowski JC, Radej S, Chodorowska G. Biochemical markers of psoriasis as a metabolic disease. Folia Histochem Cytobiol 2012;50:155-70.
12. Bezy S, Lajevardy V, Abedini R. C-reactive protein in psoriasis: A review of the literature. J Eur Acad Dermatol Venereol 2014;28:700-11.
13. Siegel D, Devaraj S, Mitra A, Raychaudhuri SK, Jialal I, et al. Characterization of psoriatic patients and controls. Arch Dermatol Res 2013;305:194-204.
14. Ohtsuka T. The correlation between response to oral cyclosporin therapy and systemic inflammation, metabolic abnormality in patients with psoriasis. Arch Dermatol Res 2008;300:545-50.
15. Romani J, Caixàs A, Carrascosa JM, Ribera M, Rigla M, Luelmo J, et al. Effect of narrowband ultraviolet B therapy on inflammatory markers and body fat composition in moderate to severe psoriasis. Br J Dermatol 2012;166:1237-44.
16. Strober B, Teller C, Yamachuchi P, Miller JL, Hooper M, Yang YC, et al. Effects of etanercept on C-reactive protein

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### Table 6: Blood picture of psoriatic patients and control group

| Lab result | Mild (20) | Moderate (20) | Severe (20) | Control (20) | P       |
|------------|----------|---------------|-------------|--------------|---------|
| Hb (g/dl)  | 13.69±1.49 (11-16.6) | 13.04±1.79 (10.2-17.5) | 12.68±1.22 (10.5-15) | 13.32±1.346 (10-15.7) | 0.233*** |
| HCT (%)    | 40.79±3.49 (34.89-48.6) | 39.59±4.32 (32.6-49.8) | 38.25±3.38 (31.1-44.3) | 39.25±3.9 (30.8-47.2) | 0.258*** |
| RBCs (million/ccm) | 4.72 (3.9-5.25) | 4.85 (4.1-5.45) | 4.72 (3.94-5.92) | 4.73 (3.91-6.16) | 0.725*  |
| WBCs (1000/ccm) | 5.99±1.74 (3.6-9.3) | 6.52±2.16 (3.2-11.13) | 6.49±1.66 (3.6-10.5) | 7.19±1.5 (4.4-10.9) | 0.249*** |
| PL (1000/ccm) | 235 (69-451) | 230 (87-385) | 182.5 (59-309) | 226.5 (119-326) | 0.494*  |
| MPV        | 10.86±1.98 (8.2-15) | 10.68±1.35 (7.9-12.9) | 9.99±1.33 (8.5-13.4) | 10.59±1.22 (7.5-13) | 0.337*** |

***ANOVA test, *Kruskal–Wallis test. All parameters described as mean±SD (minimum-maximum) except RBC and PL described as median (minimum-maximum). Lab: Laboratory, PL: Platelet, MPV: Mean platelet volume, SD: Standard deviation, RBCs: Red blood cells, Hb: Hemoglobin, HCT: Hematocrit, WBCs: White blood cells.\textsuperscript{[54]}
levels in psoriasis and psoriatic arthritis. Br J Dermatol 2008;159:322-30.
17. Zhao G, Huang L, Song M, Song Y. Baseline serum uric acid level as a predictor of cardiovascular disease related mortality and all-cause mortality: A meta-analysis of prospective studies. Atherosclerosis 2013;231:61-8.
18. Merola JF, Wu S, Han J, Choi HK, Qureshi AA. Psoriasis, psoriatic arthritis and risk of gout in US men and women. Ann Rheum Dis 2015;74:1495-500.
19. Rocha-Pereira P, Santos-Silva A, Rebelo I, Figueiredo A, Quintanilha A, Teixeira E, et al. The inflammatory response in mild and in severe psoriasis. Br J Dermatol 2004;150:917-28.
20. Takata T, Takahashi A, Taniguchi Y, Terada Y, Sano S. Detection of asymptomatic enthesitis in psoriasis patients: An onset of psoriatic arthritis? J Dermatol 2016;43:650-4.
21. Fleischer AB Jr., Rapp SR, Reboussin DM, Vanarths JC, Feldman SR. Patient measurement of psoriasis disease severity with a structured instrument. J Invest Dermatol 1994;102:967-9.
22. Raddadi AA, Jifi A, Samarthandi S, Matury N, Habibullah T, Alfarshoti M, et al. Psoriasis: Correlation between severity index (PASI) and quality of life index (DLQI) based on the type of treatment. J Dermatol Dermatol Surg 2016;20:15-8.
23. Giusti G, Galanti B. Colorimetric method. In: Bergmeyer HU, editor. Methods of Enzymatic Analysis. Weinheim: Verlag Chemie; 1984. p. 315.
24. Das RP, Jain AK, Ramesh V. Current concepts in the pathogenesis of psoriasis. Indian J Dermatol 2009;54:7-12.
25. Hashemi M, Mehrabifard H, Daliri M, Ghavami S. Adenosine deaminase activity, trypsin inhibitory capacity and total antioxidant capacity in psoriasis. J Eur Acad Dermatol Venereol 2010;24:329-34.
26. Erbagci Z, Erbagci AB, Köylüoglu O, Tuncel AA. Serum adenosine deaminase activity in monitoring disease activity and response to therapy in severe psoriasis. Acta Medica (Hradec Kralove) 2006;49:101-4.
27. Yıldırım FE, Karaduman A, Pınar A, Aksoy Y, CD26/dipeptidyl-peptidase IV and adenosine deaminase serum levels in psoriatic patients treated with cyclosporine, etanercept, and psoralen plus ultraviolet A phototherapy. Int J Dermatol 2011;50:948-55.
28. Chaudhry SD, Mittal RA, Gupta V, Saini AS, Ghalaut VS. Adenosine-deaminase (ADA) activity in psoriasis (A preliminary study). Indian J Dermat Venereol Leprol 1988;54:293-4.
29. Koizumi H, Ohkawara A. Adenosine deaminase activity in sera of patients with psoriasis, mycosis fungoides and adult T cell leukemia. Acta Derm Venerol 1972;52:410-2.
30. Feldman SR, Krueger GG. Psoriasis assessment tools in clinical trials. Ann Rheum Dis 2005;64 Suppl 2:iii65-8.
31. Coimbra S, Oliveira H, Reis F, Belo L, Rocha S, Quintanilha A, et al. C-reactive protein and leucocyte activation in psoriasis vulgaris according to severity and therapy. J Eur Acad Dermatol Venereol 2010;24:789-96.
32. Rajappa M, Shannugam R, Munisamy M, Chandrashekar L, Rajendiran KS, Thappa DM, et al. Effect of antipsoriatic therapy on oxidative stress index and sialic acid levels in patients with psoriasis. Int J Dermatol 2016;55:e422-30.
33. Boehncke W, Salgo R, Garbaraviciene J, Beschmann H, Hardt K, Diehl S, et al. Effective continuous systemic therapy of severe plaque-type psoriasis is accompanied by amelioration of biomarkers of cardiovascular risk: Results of a prospective longitudinal observational study. J Eur Acad Dermatol Venereol 2011;25:1187-93.
34. Isha, Jain VK, Lal H. C-reactive protein and uric acid levels in patients with psoriasis. Indian J Clin Biochem 2011;26:309-11.
35. Laurent MR, Panayi GS, Shepherd P. Circulating immune complexes, serum immunoglobulins, and acute phase proteins in psoriasis and psoriatic arthritis. Ann Rheum Dis 1981;40:66-9.
36. Gisondi P, Targher G, Cagalli A, Girolomoni G. Hyperuricemia in patients with chronic plaque psoriasis. J Am Acad Dermatol 2014;70:127-30.
37. Haskó G, Cronstein BN. Adenosine: An endogenous regulator of innate immunity. Trends Immunol 2004;25:33-9.
38. Goldman M. Uric acid in the etiology of psoriasis. Am J Dermatopathol 1981;3:397-404.
39. Kwon HH, Kwon IH, Choi JW, Youn JI. Cross-sectional study on the correlation of serum uric acid with disease severity in Korean patients with psoriasis. Clin Exp Dermatol 2011;36:473-8.
40. Bajaj D, Mahesar S, Ghan I. Hemoglobin content and sedimentation properties of erythrocytes in psoriasis. J Pak Assoc Derma 2008;18:132-7.
41. Görnicki A. Changes in erythrocyte micro rheology in patients with psoriasis. Clin Exp Dermatol 2004;29:67-70.
42. Bhatnagar M, Bapna A, Khare AK. Serum proteins, trace metals and phosphatases in psoriasis. Indian J Dermat Venereol Leprol 1994;60:18-21.
43. Ogdie A, Gelfand JM. Clinical risk factors for the development of psoriatic arthritis among patients with psoriasis: A review of available evidence. Curr Rheumatol Rep 2015;17:64.
44. Wilson FC, Icen M, Cowson CS, McEvoy MT, Gabriel SE, Kremers HM, et al. Incidence and clinical predictors of psoriatic arthritis in patients with psoriasis: A population-based study. Arthritis Rheum 2009;61:233-9.
45. Ogdie A, Weiss P. The epidemiology of psoriatic arthritis. Rheum Dis Clin North Am 2015;41:545-68.
46. Eder L, Gladman DD. Predictors for clinical outcome in psoriatic arthritis – What have we learned from cohort studies? Expert Rev Clin Immunol 2014;10:763-70.
47. Saleh HM, Attia EA, Onsy AM, Saad AA, Abd Ellah MM. Platelet activation: A link between psoriasis per se and subclinical atherosclerosis – A case-control study. Br J Dermatol 2013;169:68-75.
48. Kim DS, Lee J, Kim SH, Kim SM, Lee MG. Mean platelet volume is elevated in patients with psoriasis vulgaris. Yonsei Med J 2015;56:712-8.