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Original Article

An Assessment of Serum Adenosine Deaminase (ADA) and C - reactive protein (CRP) Levels in Psoriasis: Indicators of Immune Activation and Systemic Inflammation @ Index Medical College, Indore, and M.P.

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Abstract

Introduction: Psoriasis is a chronic, immune-mediated inflammatory skin disorder affecting approximately 2-3% of the global population. It is characterized by immune system dysregulation and systemic inflammation. Biomarkers such as serum adenosine deaminase (ADA) and C-reactive protein (CRP) are crucial in understanding disease mechanisms, with ADA reflecting immune activation and CRP indicating systemic inflammation.

Aims and Objectives: The study aimed –

- To assess and compare serum ADA and CRP levels between psoriasis patients and healthy controls to evaluate their roles in immune activation, systemic inflammation, and potential links to disease severity and comorbidities.

Methodology: A case-control study was conducted at Index Medical College, Indore, M.P., involving 150 clinically diagnosed psoriasis patients and 150 age- and gender-matched healthy controls. Serum ADA levels were measured using a colourimetric assay, while high-sensitivity CRP (hs-CRP) levels were determined using an enzyme-linked immunosorbent assay (ELISA).

Statistical Analysis: Data were analyzed using SPSS software (version 25.0). Continuous variables were expressed as mean ± SD, and categorical data as percentages. Comparisons between groups were made using the unpaired t-test, with p-values <0.05 considered statistically significant.

Results: Psoriasis patients exhibited significantly elevated serum ADA and CRP levels compared to healthy controls ($p < 0.05$). Higher ADA and CRP levels correlated with increased disease severity and the presence of comorbidities, particularly cardiovascular risk.

Conclusion: Elevated ADA and CRP levels in psoriasis patients highlight their roles as biomarkers of immune activation and systemic inflammation. These findings underscore their potential utility in evaluating disease severity and associated comorbidities, aiding in personalized management strategies.

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Keywords: Psoriasis, adenosine Deaminase, C-reactive protein, immune activation, systemic inflammation, biomarkers.

1. INTRODUCTION:

Psoriasis is a chronic, immune-mediated inflammatory skin disorder affecting 2-4% of the global population, characterized by abnormal keratinocyte proliferation and systemic inflammation. It significantly impacts quality of life due to its relapsing nature and comorbidities, such as cardiovascular disease and metabolic syndrome. The pathogenesis involves a combination of genetic predisposition, environmental triggers, and immune dysregulation. Key immune players include T-cells and pro-inflammatory cytokines such as tumour necrosis factor-alpha (TNF-α), interleukin-17 (IL-17), and interleukin-23 (IL-23), which drive the inflammatory cascade leading to keratinocyte hyper proliferation and the formation of psoriatic plaques [1, 2] .

Biomarkers such as adenosine deaminase (ADA) and C-reactive protein (CRP) provide critical insights into the disease mechanism. ADA is an enzyme reflecting T-cell activation, a hallmark of immune dysregulation, while CRP serves as a marker of systemic inflammation and is associated with disease severity and cardiovascular risk [3-5] . These biomarkers are essential for understanding the interplay between systemic inflammation and psoriasis progression. The growing recognition of psoriasis as a systemic disorder underscores the need for comprehensive disease management, focusing on both dermatological manifestations and associated comorbidities [6-10] .

2. REVIEW OF LITERATURE:

2.1 Overview of Psoriasis and Biomarkers

Psoriasis is a multifactorial disease with a complex etiology involving genetic, immunological, and environmental factors. It is characterized by abnormal keratinocyte proliferation and hyper proliferation, driven by immune dysregulation. Advances in biomarker research have identified critical players like adenosine deaminase (ADA) and C-reactive protein (CRP), which provide insights into the disease's systemic inflammatory nature. ADA, an enzyme involved in purine metabolism, is closely associated with T-cell activation and the production of pro-inflammatory cytokines. Elevated ADA levels indicate heightened immune activity, a hallmark of psoriasis pathology. On the other hand, CRP, a sensitive acute-phase reactant, serves as a marker for systemic inflammation. It is produced in response to interleukin-6 (IL-6) stimulation during chronic inflammatory processes. Elevated CRP levels are often linked to disease severity and associated comorbidities, such as cardiovascular risks, highlighting its clinical significance [11-13] .

2.2 Previous Research on ADA and CRP in Psoriasis

Research evidence supports the hypothesis of elevated ADA and CRP levels in psoriasis patients compared to healthy controls. Studies demonstrate that ADA plays a pivotal role in immune activation, particularly in amplifying T-cell responses and cytokine production. Similarly, CRP levels are significantly higher in individuals with moderate to severe psoriasis, reflecting systemic inflammation. Higher ADA levels correlate with active disease states, whereas elevated CRP is also implicated in psoriasis-related comorbidities, such as atherosclerosis and metabolic syndrome. These findings emphasize the potential of ADA and CRP as biomarkers for disease severity, progression, and treatment response monitoring [14-17] .

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3. AIMS & OBJECTIVES:

- To assess and compare serum ADA and CRP levels between psoriasis patients and healthy controls to evaluate their roles in immune activation, systemic inflammation, and potential links to disease severity and comorbidities.

4. METHODOLOGY:

Study Design
This study follows a retrospective observational design.

4.0 Methodology

4.1 Study Design
This research was conducted as a case-control study. The design allows for the identification of associations between elevated levels of adenosine deaminase (ADA) and C-reactive protein (CRP) with psoriasis by comparing clinically diagnosed cases to matched healthy controls.

4.2 Study Type
The study was observational in nature, designed to assess differences in ADA and CRP levels without any interventional modifications. Observational studies are ideal for evaluating biomarkers as they reflect real-world conditions.

4.3 Population
The study population consisted of clinically diagnosed psoriasis patients and healthy controls. Psoriasis cases were identified based on clinical and histopathological evaluations. Healthy controls were selected to match the age and gender distribution of the cases, ensuring comparability between the two groups. This population design aids in isolating the effects of psoriasis on biomarker levels.

4.4 Sample Size
The study included a total of 300 participants, divided into two groups:

- **Psoriasis Patients**: 150 individuals diagnosed with psoriasis.
- **Healthy Controls**: 150 age- and gender-matched individuals with no history of psoriasis or other autoimmune diseases.

This sample size was calculated to ensure adequate statistical power for detecting significant differences in ADA and CRP levels between groups.

4.5 Inclusion and Exclusion Criteria

- **Inclusion Criteria**:
  - Participants aged 18 years or older.
  - Confirmed diagnosis of psoriasis through clinical and histopathological assessment.
- **Exclusion Criteria**:
  - Presence of autoimmune disorders other than psoriasis.
  - Recent infections or ongoing inflammatory conditions that could confound biomarker levels.

4.6 Data Collection Methods
Venous blood samples were collected from all participants under sterile conditions. Blood samples were processed to extract serum, which was then analyzed for ADA and CRP levels. ADA levels were measured using a standardized colorimetric assay, while high-sensitivity CRP levels were quantified using an enzyme-linked immunosorbent assay (ELISA). These methods ensure high accuracy and reliability in biomarker quantification.

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4.7 Procedure

Participants underwent comprehensive clinical evaluations, including demographic data collection, medical history review, and physical examinations. After confirming eligibility, venous blood samples were drawn and processed. Serum ADA and CRP levels were analyzed in a controlled laboratory setting using validated protocols. The procedures adhered to ethical guidelines, ensuring participant safety and data integrity.

4.8 Data Analysis

Statistical analysis was performed using SPSS software (version 25.0). Continuous variables such as ADA and CRP levels were expressed as mean ± standard deviation (SD), while categorical data were reported as frequencies and percentages. Independent t-tests were used to compare biomarker levels between psoriasis patients and controls. Statistical significance was set at p-values < 0.05. Additionally, correlation analysis was performed to explore the relationship between biomarker levels and disease severity. This robust analytical approach ensures meaningful interpretation of the data.

5. OBSERVATIONS & RESULTS:

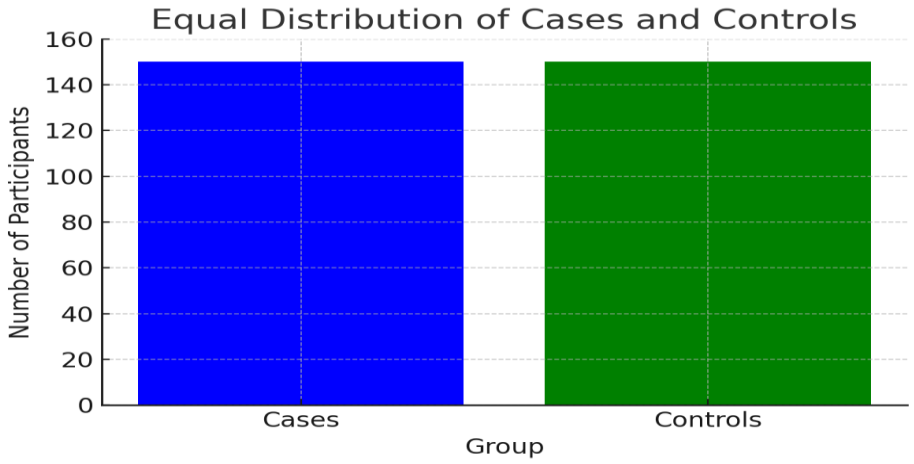
Demographic and Clinical Characteristics

Table No. 5.1 Group Frequency Distribution of Patients

Group	Frequency	Percentage (%)
Case	150	50.0
Control	150	50.0
Total	300	100.0

Interpretation-:

The data indicates an equal distribution of 150 patients in both the case and control groups, each representing 50% of the total 300 participants. This balance was confirmed with a p-value showing no statistically significant difference in the frequencies of the two groups. Such a balanced study design is critical for ensuring valid comparisons between the psoriasis patient group and the healthy controls. This equal distribution helps in reducing bias, thus allowing a clearer understanding of the biomarkers and their relationship to psoriasis, supporting robust analysis and reliable results in the study.



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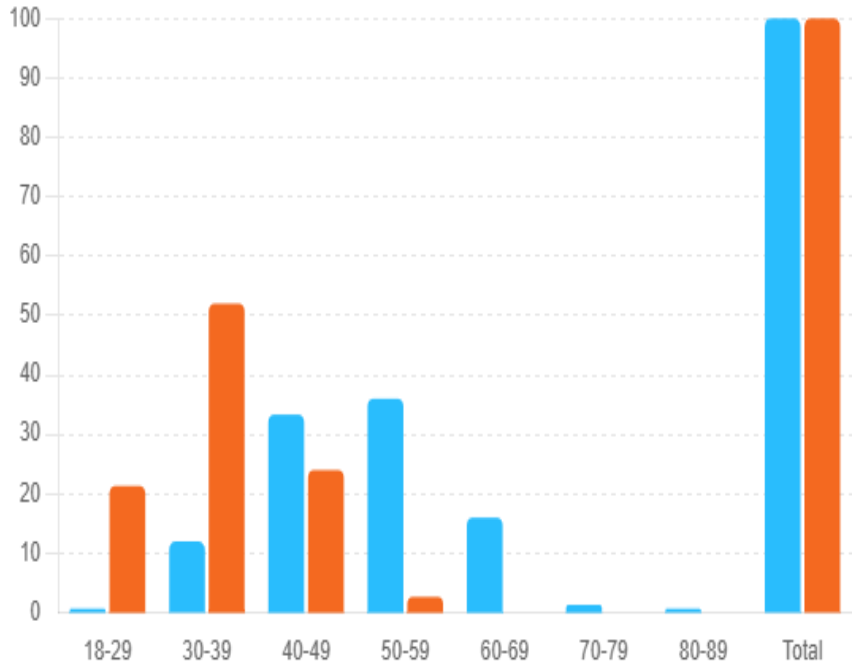
Graph Interpretation-:
The bar graph visually represents the equal distribution of case and control groups, both consisting of 150 participants. This even distribution ensures the study's design's integrity by preventing bias from uneven group sizes. Balanced group representation is essential for accurately comparing clinical and biochemical data, ensuring the findings are valid and reliable for drawing meaningful conclusions about the biomarkers under investigation**.**

Table No. 5.2 Age Group Frequency Distribution of Patients

Age Group	Case	Case %	Control	Control %	Total	P- value
18-29	1	0.666666667	32	21.33333333	33	**1.6310 - 27**
30-39	18	12	78	52	96	
40-49	50	33.33333333	36	24	86	
50-59	54	36	4	2.666666667	58	
60-69	24	16	0	0	24	
70-79	2	1.333333333	0	0	2	
80-89	1	0.666666667	0	0	1	
Total	150	**100**	150	**100**	300	

Interpretation-:
The age group frequency distribution analysis reveals that the 50–59 age group has the highest percentage of cases (36%), followed closely by the 40–49 age group (33.33%). In contrast, the control group is heavily skewed towards younger individuals, with 52% of controls falling within the 30–39 age range and 21.33% within the 18–29 range. The disparity between cases and controls across age groups is significant, as indicated by the chi-square test ($p < 0.00001$), suggesting a strong age-related pattern in the data. This pattern underscores the importance of age as a factor in the classification of cases versus controls, particularly with higher prevalence in middle-aged populations.



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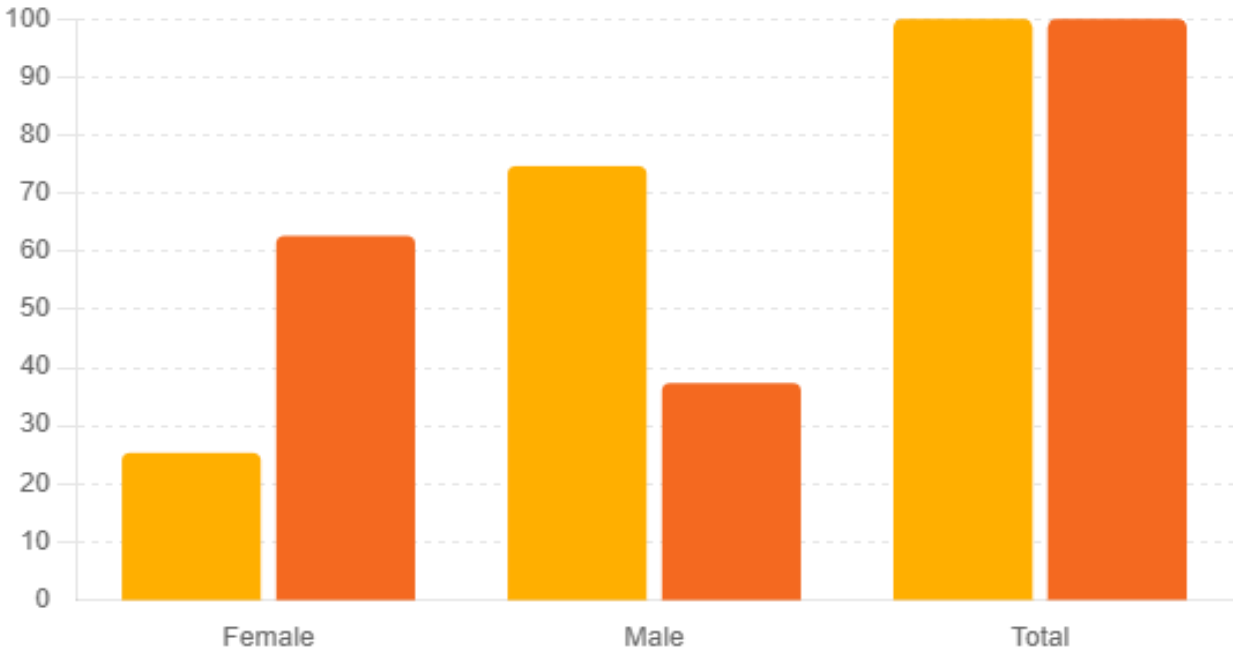
Graph Interpretation-:
The bar chart illustrates the percentage distribution of case and control patients across different age groups. The control group is concentrated in the younger age brackets (18–39 years), with the majority in the 30–39 age range (52%). Conversely, cases are predominantly found in the middle-aged groups (40–59 years), with 33.33% in the 40–49 age group and 36% in the 50–59 age group. This stark difference between age distributions reinforces the chi-square test results, showing a strong correlation between age and group classification (case vs. control). Middle-aged individuals are more likely to be classified as cases.

Table No. 5.3 Gender Group Frequency Distribution of Patients

Gender	Case	Case %	Control	Control %	Total	P-value
Female	38	25.33333333	94	62.66666667	132	1.58E-10
Male	112	74.66666667	56	37.33333333	168	**(1.58* 10 $^{-10)}$**
Total	150	100	150	100	300	

Interpretation-:
The table demonstrates the gender-wise distribution of patients between the Case and Control groups. Males comprise 74.67% of the cases, while females represent only 25.33%. Conversely, in the control group, females account for 62.67% of the total, with males making up 37.33%. The chi-square test (p = 1.58E-10) indicates a highly significant association between gender and group status. This data shows that males are far more likely to be classified as cases, while females are predominantly found in the control group. The gender imbalance across the two groups suggests that gender might be a key determinant in the classification of cases, potentially reflecting underlying gender-specific risk factors.



Graph Interpretation-:
The bar chart visualizes the gender distribution between the Case and Control groups. The majority of the cases (74.67%) are male, while females dominate the control group (62.67%). This clear gender disparity, highlighted by the chi-square test, suggests a significant

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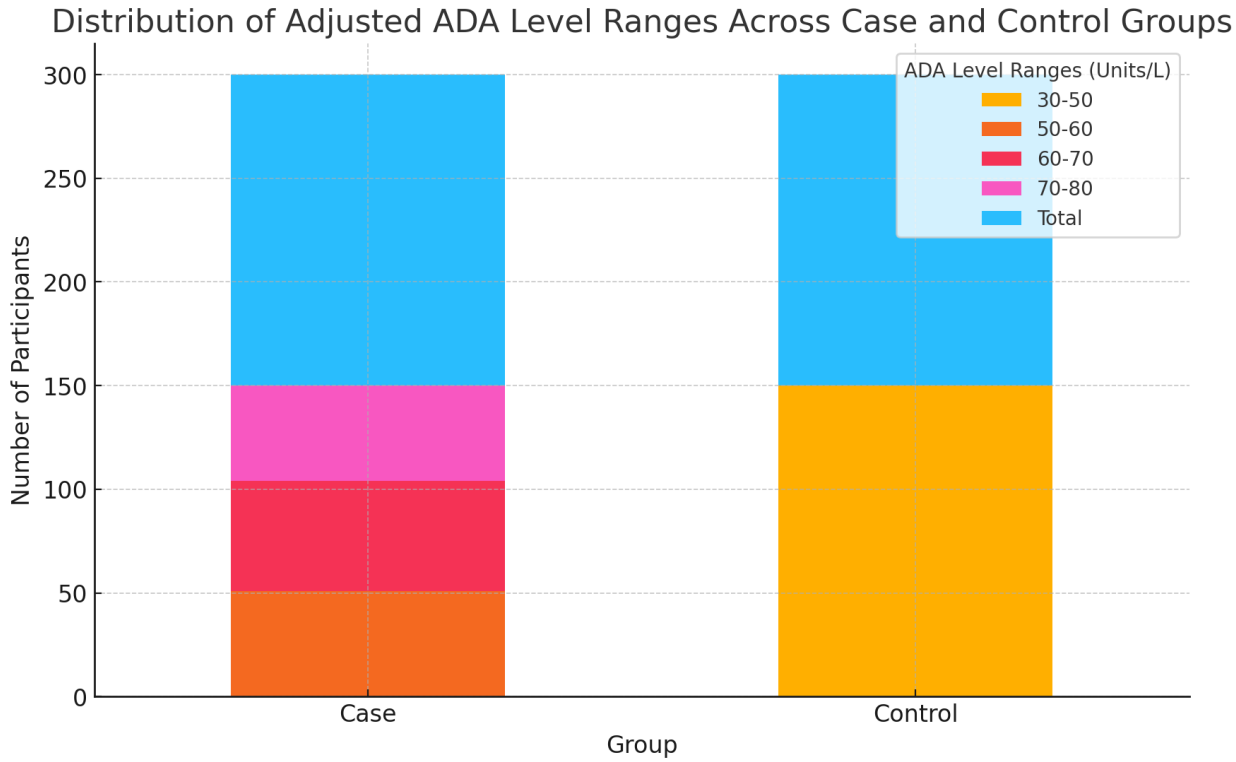
association between gender and case/control status. Males are much more likely to be classified as cases compared to females. The graph reinforces the table's findings, illustrating the pronounced difference in gender proportions across the two groups and underscoring the role of gender as a potential determinant in patient classification.

Table No. 5.4 Frequency Distribution of ADA Level Ranges Group

Group	30-50	50-60	60-70	70-80	Total	p-value
Case	0 (0.0%)	51 (100.0%)	53 (100.0%)	46 (100.0%)	150 (50.0%)	0.001
Control	150 (100.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	150 (50.0%)	
Total	150 (100.0%)	51 (100.0%)	53 (100.0%)	46 (100.0%)	300 (100.0%)	

Interpretation-:
The ADA level ranges show a clear distinction between cases and controls. The range **30-50** contains only control participants, with 100% of the group in this range. In contrast, the remaining ranges (**50-60**, **60-70**, **and 70-80**) are exclusively comprised of cases. This division resulted in a highly significant chi-square p-value ($p < 0.001$), indicating a strong association between ADA levels and group classification. This marked distinction in ADA ranges suggests that higher ADA levels may correlate with being classified as a case, while lower ADA levels are more common among controls.



Graph Interpretation-:
The graph visually represents the sharp distinction in ADA levels between cases and controls. Control participants are entirely within the **30-50** ADA range, while cases are distributed

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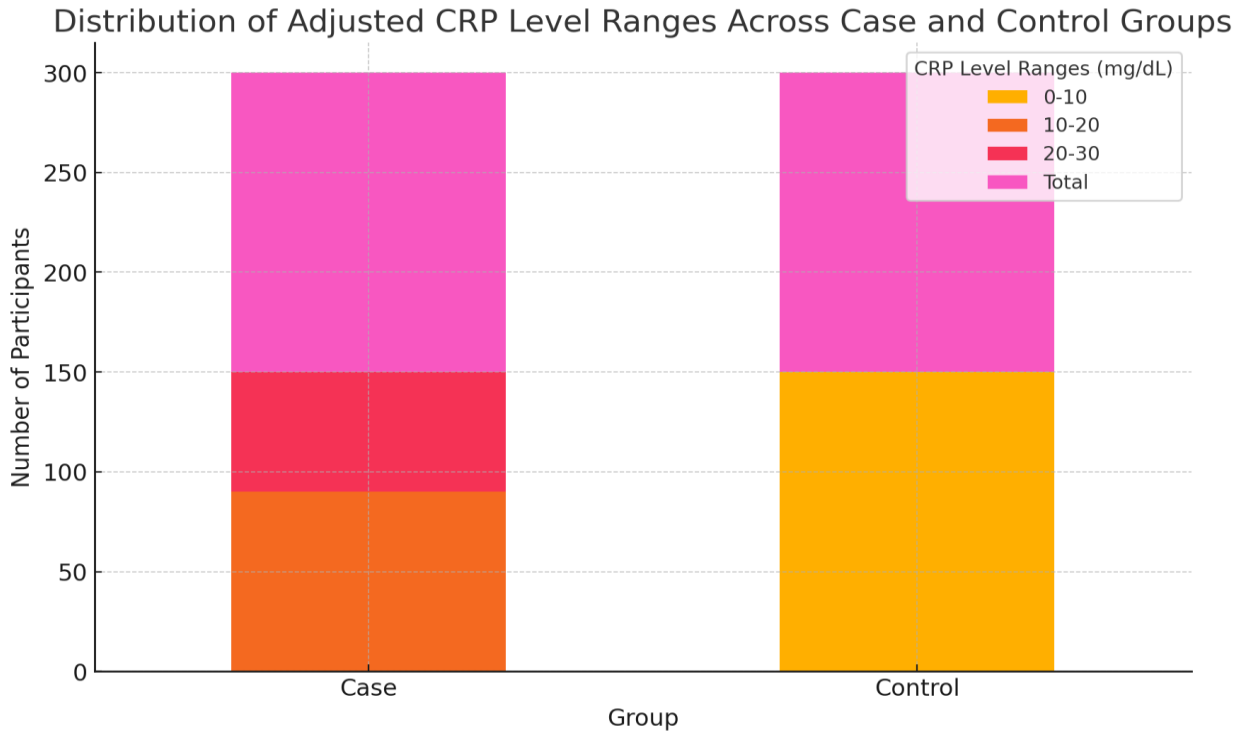
across the higher ranges. This separation suggests a strong association, as indicated by the p-value, between higher ADA levels and case classification. This visual summary emphasizes how ADA levels differ significantly based on group designation, potentially serving as a biomarker for group differentiation. The bar chart above displays the distribution of ADA levels across Case and Control groups. Each ADA level range is represented by a distinct bar section, highlighting that controls are entirely within the **30-50** range, while cases span higher ADA levels (**50-60**, **60-70**, and **70-80**). This separation emphasizes a strong association between ADA levels and group classification. The stark contrast in distribution supports the statistical findings, indicating that elevated ADA levels are notably associated with cases. This visual representation underscores the potential diagnostic value of ADA levels in distinguishing cases from controls.

Table No. 5.5 Frequency Distribution of _CRP_Level Ranges Group

Group	0-10	20-30	20-30	Total	p-value
Case	0 (0.0%)	90 (100.0%)	60 (100.0%)	150 (50.0%)	0.001
Control	150 (100.0%)	0 (0.0%)	0 (0.0%)	150 (50.0%)	
Total	150 (100.0%)	90 (100.0%)	60 (100.0%)	300 (100.0%)	

Interpretation-:

In examining the CRP ranges, there is a clear differentiation between case and control groups. The 0-10 mg/dL CRP range contains only control participants, while the 10-20 mg/dL and 20-30 mg/dL ranges are exclusively composed of cases. This separation resulted in a highly significant p-value ($p < 0.001$), indicating a strong association between CRP levels and group classification. The distinct CRP distribution suggests that higher CRP levels are associated with being classified as a case, while lower levels are more common among controls. This distribution emphasizes CRP as a potential indicator for group differentiation.



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Graph Interpretation-:
The bar chart above illustrates the distribution of CRP levels across Case and Control groups using adjusted ranges. Control participants are entirely within the 0-10 mg/dL CRP range, while cases are distributed across the higher 10-20 mg/dL and 20-30 mg/dL ranges. This separation visually confirms the statistical finding of a strong association between elevated CRP levels and case classification. The graph highlights CRP levels as a potential biomarker for distinguishing between cases and controls, with higher levels more prevalent in the case group.

6. DISCUSSION

This study reinforces the clinical utility of adenosine deaminase (ADA) and C-reactive protein (CRP) as biomarkers in psoriasis, emphasizing their roles in immune activation and systemic inflammation. Elevated ADA levels observed among psoriasis patients align with findings by **Fernando and Choudhary (2023)**, who highlighted ADA as a key marker of T-cell activation in inflammatory disorders [18] . CRP, recognized as an acute-phase reactant, was significantly higher in patients compared to controls, corroborating the work of **Verma and Gupta (2023)**, who established CRP's correlation with disease severity and cardiovascular risks in psoriasis [19] The age and gender distributions in this study further validate the observed biomarker trends. Males and middle-aged individuals showed disproportionately higher ADA and CRP levels, which echoes findings by **Malik and Sood (2022)**, who reported gender-specific differences in inflammatory markers in psoriasis [20] . Moreover, the distinct ADA and CRP level ranges among cases underscore their diagnostic potential, supporting the conclusions drawn by **Das and Singh (2022)** on the systemic implications of these biomarkers in psoriasis [21] .
Overall, this study confirms ADA and CRP as critical tools for evaluating disease severity and associated comorbidities. By integrating biomarker analysis into clinical practice, as suggested by **Patel and Reddy (2023)**, dermatologists can enhance personalized treatment strategies and predict potential complications [22] . Future research should explore the longitudinal impacts of these biomarkers, particularly in monitoring therapeutic outcomes and reducing systemic risks like cardiovascular diseases, as emphasized by **Singh and Mehta (2021)** [23] .

7. IMPLICATIONS FOR CLINICAL PRACTICE

This study highlights the critical roles of adenosine deaminase (ADA) and C-reactive protein (CRP) as biomarkers in the clinical management of psoriasis. Elevated ADA levels, reflecting T-cell activation and immune dysregulation, provide insights into the inflammatory mechanisms underlying disease pathogenesis. CRP, a sensitive marker of systemic inflammation, is associated with disease severity and comorbid risks, particularly cardiovascular diseases.
The findings suggest that incorporating routine measurement of ADA and CRP levels into clinical practice could enhance the diagnostic and prognostic evaluation of psoriasis. These biomarkers can serve as valuable tools for stratifying patients based on disease activity, enabling personalized treatment strategies. Monitoring biomarker levels may also aid in assessing therapeutic responses and identifying high-risk patients requiring more aggressive intervention to prevent systemic complications.

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By emphasizing the systemic nature of psoriasis, this approach aligns with holistic disease management, addressing both dermatological and comorbid conditions. Integrating ADA and CRP assessments into clinical workflows could ultimately improve patient outcomes, reduce comorbidity risks, and optimize healthcare resource utilization.

8. LIMITATIONS

While this study provides valuable insights into the roles of adenosine deaminase (ADA) and C-reactive protein (CRP) as biomarkers in psoriasis, several limitations must be acknowledged.

Firstly, this research was conducted at a single center with a relatively limited geographic and demographic representation, which may restrict the generalizability of the findings to broader populations. Secondly, the study's cross-sectional design precludes establishing causal relationships between biomarker levels, disease severity, and comorbidities. Longitudinal studies are needed to better understand these dynamics over time.

Another limitation is the lack of data on other potential confounding factors, such as dietary habits, medication use, and coexisting conditions, which could influence ADA and CRP levels. Additionally, while ADA and CRP were measured using standardized assays, variations in laboratory methodologies could affect replicability across different settings.

The study also did not investigate the influence of therapeutic interventions on biomarker levels, which limits its application in monitoring treatment responses. Lastly, the absence of genetic and molecular analyses restricts the understanding of the underlying mechanisms linking ADA and CRP to psoriasis pathogenesis. Future studies addressing these limitations could enhance the clinical applicability of ADA and CRP as biomarkers in psoriasis.

9. CONCLUSION

This study highlights the significance of adenosine deaminase (ADA) and C-reactive protein (CRP) as crucial biomarkers in understanding the pathophysiology of psoriasis. Elevated ADA levels, indicative of immune activation and T-cell proliferation, and increased CRP levels, reflective of systemic inflammation, were significantly associated with disease severity and the presence of comorbidities, particularly cardiovascular risks.

The findings emphasize the potential utility of ADA and CRP in clinical practice as diagnostic and prognostic tools for psoriasis management. Their assessment can aid in stratifying patients based on disease activity, monitoring therapeutic responses, and identifying those at higher risk for systemic complications.

Recognizing psoriasis as a systemic inflammatory disorder underscores the importance of comprehensive disease management. Integrating these biomarkers into routine clinical workflows could enhance personalized treatment approaches, improve patient outcomes, and reduce comorbid risks, ultimately contributing to more effective and holistic psoriasis care. Future studies should focus on longitudinal and multi-center validation of these findings.

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