Journal of Health and Technology
Volume 1, Issue 2, June 2025; pp: 59-68
eISSN: 2455-2852 DOI: 10.65129/health.v1i2.26

REVIEW ARTICLE

Assessment of Skin Barrier in Atopic Dermatitis

H. R. Suba, S. Subathra, T. Lovie Beneta and A. Parimala

Department of Physiology, Stanley Medical College, Chennai – 600001, Tamil Nadu, India;
dr.subakarthi@gmail.com

Abstract

The skin barrier is a complex protective system that helps maintain many essential functions, including immunology. Atopic Dermatitis (AD) or Atopic Eczema (AE) is a chronic, relapsing, complex inflammatory dermatosis that affects both children and adults, in which the skin's protective role is disturbed by factors such as genetic predisposition, epidermal barrier disruption, immune regulation abnormalities, and environmental influences. The objective of this study is to assess the severity of barrier disruption in the Skin and to investigate the relationship between moisture level and severity of lesions in patients with Atopic Dermatitis. This cross-sectional analytical study included 30 patients with Atopic Dermatitis (new and known cases) of both genders, aged 18 to 40 years, at the Department of Dermatology, Stanley Medical College Hospital. Examination of Skin with the help of SCORAD score assessment. Digital Moisturometer (To measure the Moisture level of skin by utilizing Bioelectric Impedance Analysis (BIA)) was done and the results were recorded.The participants are categorized as Dry skin and Normal skin based on the Moisture score and categorized as Moderate Atopic Dermatitis and Severe Atopic Dermatitis based on SCORAD score. Among participants with Dry skin, 43.8% had a moderate SCORAD score, while 85.7% had a severe SCORAD score. Among participants with Normal skin, 56.3% had a moderate SCORAD score, while only 14.5% had a severe SCORAD score. Among participants with Normal skin, 14.5% had a severe SCORAD score. The P value is found to be significant (0.017). This research describes the statistically significant relationship between skin moisture level and Atopic Dermatitis severity. This can prove to help investigate interventions to control the barrier integrity to enhance the Quality of life in patients with Atopic Dermatitis.

Keywords: Barrier-targeted Therapy, Digital Moisture Monitor, Epidermal Integrity, Lichenification, SCORAD Index, Transepidermal Water Loss (TEWL)

1. Introduction and Background

The skin barrier, also known as the epidermal barrier, is a complex and protective integumentary system that protects against chemicals, UV radiation, preserves a balanced internal environment, regulates body temperature, acts as a shock absorber, and provides lubrication.

A defective skin barrier leads to exposure of the integument to various exogenous substances, including allergens and microbes. Thus, an inflammatory cascade is triggered.

Atopic Dermatitis (AD) or Atopic Eczema (AE) is a chronic, relapsing, complex inflammatory dermatosis that affects both children and adults, in which various factors, including genetic predisposition, epidermal barrier abnormalities, immune regulation abnormalities, and environmental influences, disrupt the skin's barrier function.

This study explores the connection between dry skin and the severity of Atopic Dermatitis cases in comparison to individuals with normal skin. A primary factor contributing to both new and recurring lesions in Atopic Dermatitis is skin dryness (xerosis). This dryness leads to the itch-scratch cycle, which triggers an immunological response resulting in erythema, edema, and oozing in the affected areas. Oozing lesions may subsequently become secondarily infected. The repeated flare-ups of Atopic Dermatitis lesions lead to thickening and lichenification of the affected skin.

**Author for correspondence*

Article Received on: 22 March 2025 Revised on: 16 May 2025 Accepted on: 27 June 2025

Assessment of the severity of dryness helps in the management and improves the quality of life of patients with Atopic Dermatitis.

2. Review of Literature

Atopic Dermatitis (AD) is a chronic, relapsing inflammatory skin disease characterized by pruritus, eczematous lesions, and significant impairment in quality of life. The understanding of AD has evolved considerably, with epidemiological, clinical, and pathophysiological studies highlighting its complexity.

2.1 Epidemiology and Clinical Profile

Epidemiological investigations have demonstrated wide variability in prevalence across geographical regions. A South Indian hospital-based study highlighted distinct clinical patterns and demographic trends, underscoring the regional variation in disease expression[1]. Global epidemiological data emphasize the increasing burden of AD in both children and adults[2,] while adult AD is often underrecognized, presenting with different morphological patterns than childhood disease[3] with the prevalence rate of AD-0.58%[1].

2.2 Disease Severity and Assessment Tools

Accurate evaluation of AD severity is crucial for both clinical care and research. The Patient-Oriented SCORAD (PO-SCORAD), a validated self-assessment scale, enables patients to record symptoms such as pruritus and sleep loss, thereby enhancing patient engagement in disease monitoring[4]. Disease severity has also been correlated with the presence of asthma and rhinitis, reinforcing the concept of the "atopic march"[5]. Such tools provide reliable measures of disease burden and allow for better standardization in clinical practice.

2.3 Structure of the Skin Barrier

2.3.1 Stratum Corneum

The Stratum Corneum (SC) is the outermost layer of the epidermis and the most significant in barrier function. It is structured as corneocytes embedded in a lipid matrix, often likened to a "brick and mortar" model. Corneocytes (the bricks) are filled with keratin and surrounded by a cornified envelope, while intercellular lipids (the mortar) consist of ceramides, cholesterol, and free fatty acids.

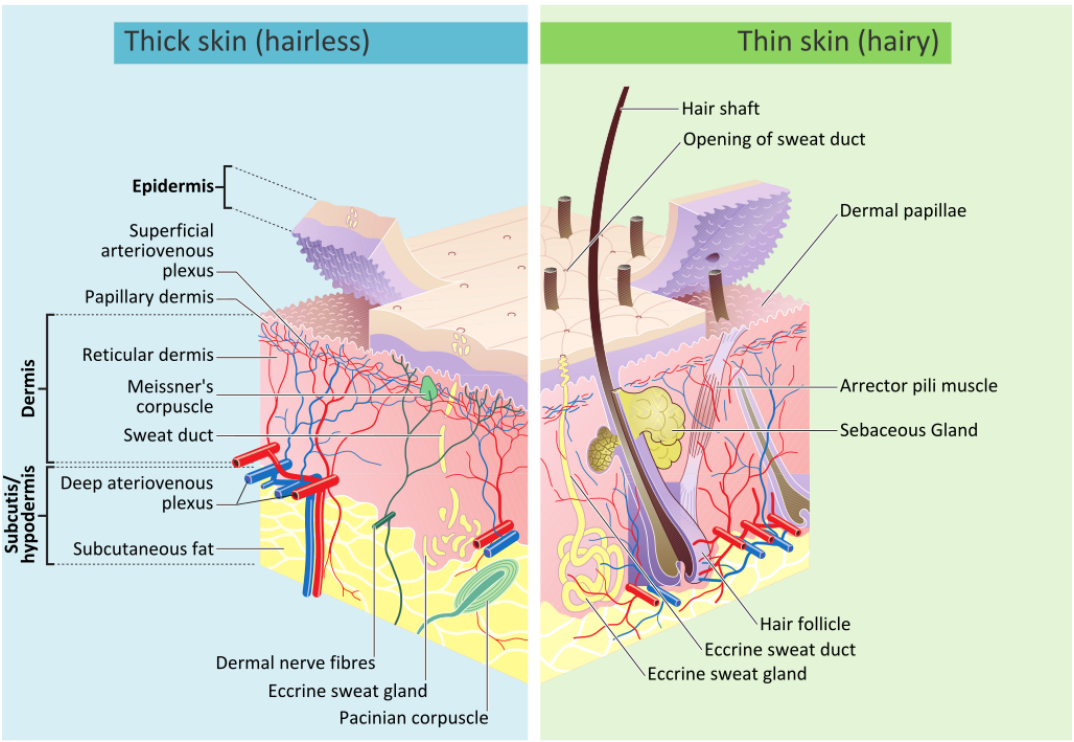

Figure 1. Components of skin.
IADVL'S Concise Textbook of Dermatology; 2019.

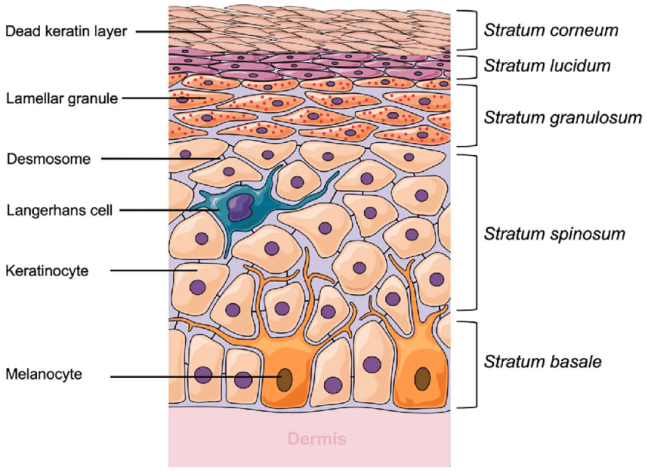

Figure 2. Layers of epidermis.

2.4 Filaggrin and Cornified Envelope Proteins

Filaggrin plays a vital role in keratin aggregation and is degraded into Natural Moisturizing Factors (NMFs) such as pyrrolidone carboxylic acid and urocanic acid. These maintain hydration and pH. Loss-of-function mutations in the FLG gene compromise barrier integrity and are linked with severe and early-onset Atopic Dermatitis[6].

2.5 Lipid Barrier

The lipid matrix in the SC is critical for barrier function. In AD, there is a significant reduction in ceramide content, especially ceramide 1 and ceramide 3, leading to increased TEWL and permeability.

2.6 Microbiome and Immune Interactions

The skin barrier also encompasses microbial and immunological components. Dysbiosis, particularly colonization by Staphylococcus aureus, is commonly seen in AD and exacerbates inflammation and barrier dysfunction.

2.7 Pathogenesis of Barrier Disruption in Atopic Dermatitis

- The pathogenesis of Atopic Dermatitis (AD) has been attributed largely to abnormalities in the adaptive immune system, with key roles played by T-helper 1(Th1)/Th2 cell dysregulation, IgE production, dendritic cell signaling, and mast-cell hyperactivity, resulting in the pruritic, inflammatory dermatosis that characterizes AD[7].
- A defective skin barrier leads to exposure of the integument to various exogenous substances, including allergens and microbes. Thus, an inflammatory cascade is triggered.
- The inflammatory cytokines in AD comprise the T Helper (TH) cell 2 cytokines such as interleukins (ILs) 4, 5, 13,31, 33 and thymic stromal lymphopoietin (TSLP).
- IL-4 and IL-13 downregulate filaggrin expression in keratinocytes, further disrupting the epidermal barrier. IL-4 also downregulates the expression of cutaneous defensins and upregulates the expression of bacterial adhesion molecules, both of which promote the colonization of Staphylococcus aureus on the AD skin.
- There are decreased levels of lipids, ceramides, cholesterol and free fatty acids in the lipid bilayer and lamellar granules, resulting in increased TEWL (Trans Epidermal Water Loss)[8].
- There is a significant decrease in tight junction (particularly claudin 1), which leads to increased susceptibility to infections and increased allergen entry, resulting in immune dysregulation.
- Reduced Antimicrobial Peptides (AMPS) (ß-defensins, cathelicidins) in the stratum corneum and granulosum lead to increased susceptibility to infections. Increased expression of proteases (kallikreins, cathepsins, caspase-14) leads to abnormal desquamation and breakdown of structural proteins and lipids[1].

2.8 Clinical Burden and Management

The impact of AD extends beyond the skin. The burden of disease includes sleep disturbance, psychosocial stress, and comorbidities such as asthma, allergic rhinitis, and depression. Managing adult AD is particularly challenging, especially in refractory cases where a stepwise approach involving topical therapy, systemic immunomodulators, and biologics is required[9]. Standard textbooks, including the *IADVL's Concise Textbook of Dermatology*[6] and *Atopic Dermatitis: Text and Atlas*[8], provide consolidated insights into therapeutic strategies and clinical care pathways tailored for the Indian context. The literature indicates that AD is a multifactorial disorder with substantial heterogeneity across age groups and geographic regions. Epidemiological studies stress its rising global burden[2,3] while clinical research underscores the importance of validated severity indices

like SCORAD for uniform assessment[4,5]. Advances in understanding barrier dysfunction and immune dysregulation have reshaped pathogenic models[7,10,11], offering novel therapeutic targets. Nonetheless, AD continues to impose a significant psychosocial and clinical burden[9,12], necessitating comprehensive and individualized management strategies.

3. Aim of this Study

- To assess the severity of Atopic Dermatitis.
- To correlate the severity with skin barrier dysfunction using SCORAD scoring and digital skin moisture monitoring.

4. Objectives

- To evaluate the clinical severity of Atopic Dermatitis using the SCORAD Index.
- To assess skin hydration levels using a digital moisture monitor as an objective marker of barrier integrity.
- To analyze the correlation between disease severity and skin hydration status.
- To start early management.

5. Methodology

Data collection was done after obtaining Institutional Ethical Committee clearance. **Study Design**: Cross-sectional analytical study.

Place of Study: Department of Physiology and Department of Dermatology, Stanley Medical College.

Study Population: Patients diagnosed with Atopic Dermatitis of both genders (New and Known cases after obtaining informed consent) among 18 to 45-year-olds from the Dermatology OPD in Stanley Medical College.

Sampling Method: Convenience Sampling.

Sample Size: 30 Atopic Dermatitis patients (new and known cases) who are fulfilling the inclusion criteria according to the prevalence rate of AD-0.58%[1].

Study Tools: General Examination and Systemic examination were done.

- Examination of Skin with SCORAD score Assessment (To assess the level of severity in Atopic Dermatitis).
- Digital Dermoscope (For more accurate visual inspection of lesions).
- Digital Moisturometer (To measure the Moisture level of skin by utilizing Bioelectric Impedance Analysis (BIA)) was used and the results were recorded.

6. Scorad Score Assessment

The SCORAD score range is between 0 and 103 points and defines three classes of AD severity (i.e. mild if SCORAD <25, moderate if 25 ≤ SCORAD ≤ 50 and severe if SCORAD > 50).

7. Results

7.1 Severity Assessment

Table 1. Lesional involvement in percentage

Descriptives (n =30)	Percent of area involved
Mean	19.8
Median	18.0
Standard deviation	12.6
Minimum	4.50
Maximum	54.0

7.2 Plots

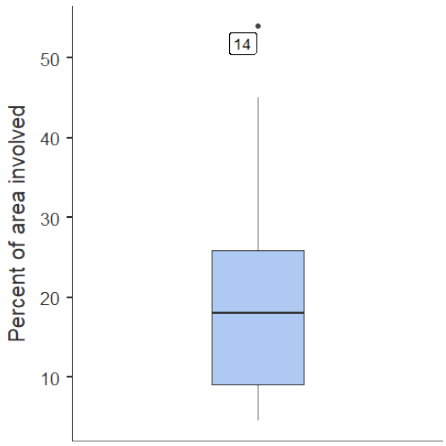

Chart 1. Percent of area involved.

Figure 3. Digital Moisture Meter.

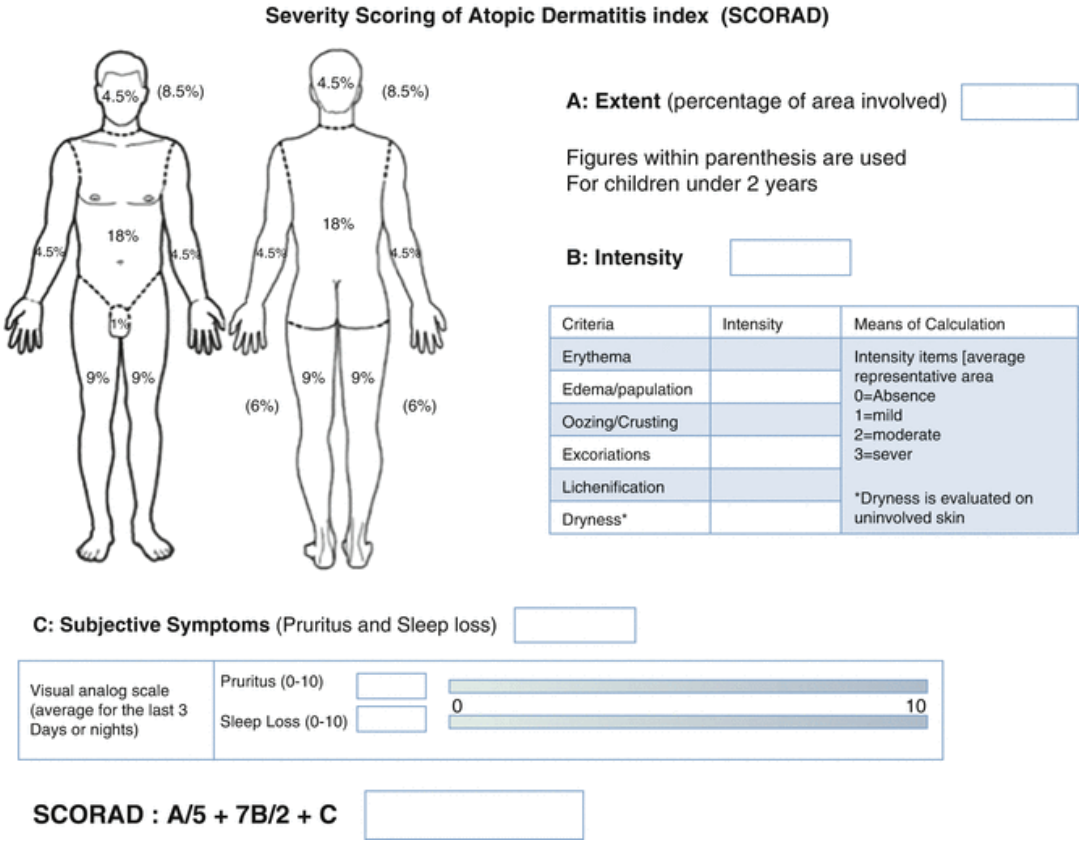

Figure 4. Severity scoring of atopic dermatitis index.

Figure 5. Digital dermoscope.

8. Results

- OPEN EPI software was used for the analysis of data.

Table 2. Assessment of pruritus and sleep loss

Descriptives (n =30)	Pruritus	Sleep loss
Mean	3.77	3.77
Median	3.50	3.50
Standard deviation	1.81	1.81
Minimum	1	1
Maximum	7	7

- The participants were categorized as Dry skin and Normal skin based on the moisture score and categorized as Moderate Atopic Dermatitis and Severe Atopic Dermatitis based on SCORAD score.

8.1 Demographic Characteristics

A total of 30 patients with clinically diagnosed Atopic Dermatitis (AD) were included in the study. The mean age of participants was 32.1 years (SD = 7.86), with ages ranging from 13 to 45 years. The age group distribution showed that 83.3% of the patients were in the 21 to 40 years age group, while 3.3% were aged 11–15 years, and 6.7% each were in the 16–20 years and above 41 years categories.

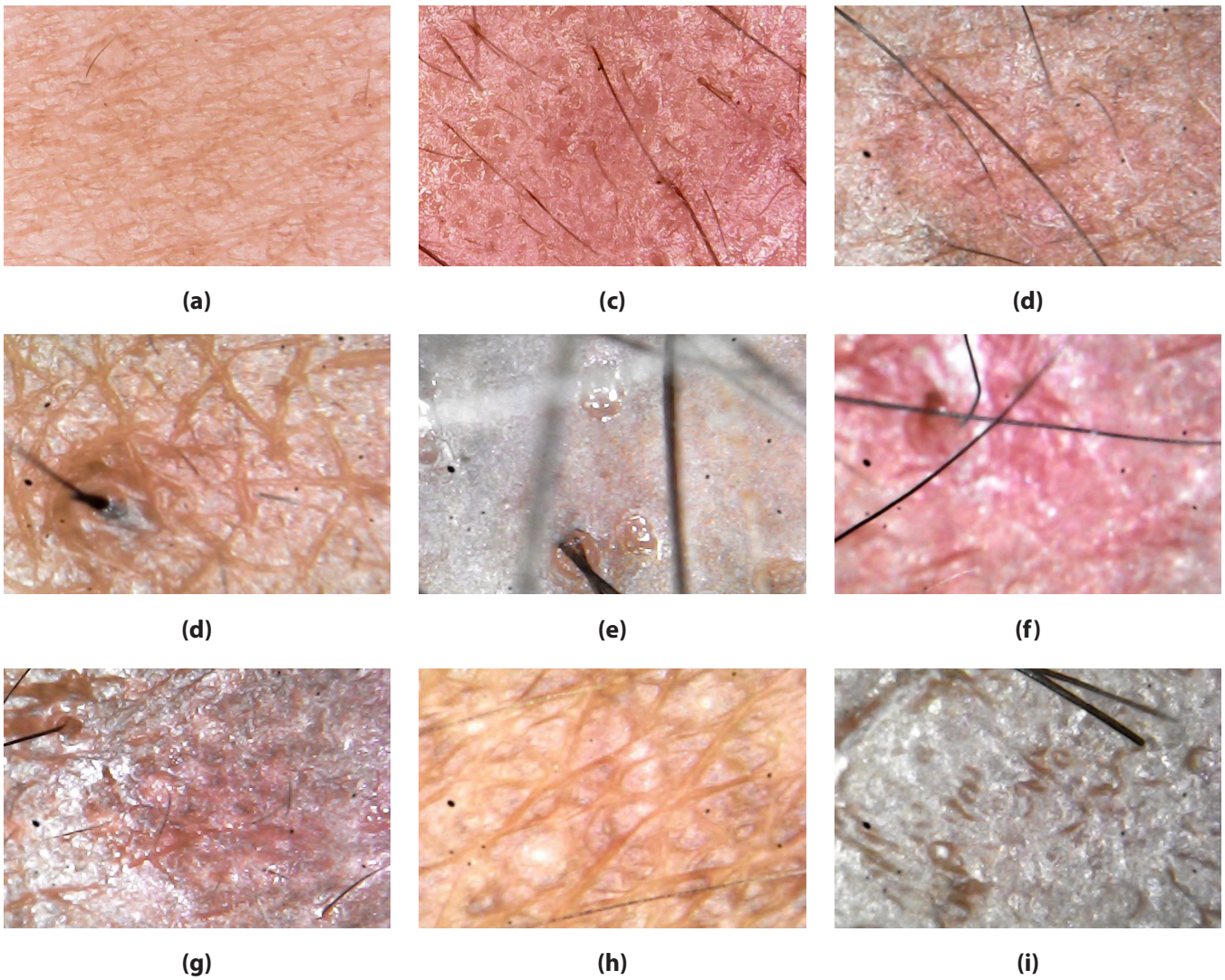

Figure 6. **(a)** Normal skin. **(b)** Xerotic skin. **(c)** Xerotic eczema. **(d)** Perifollicular inflammation. **(e)** Blistering dermatitis. **(f)** Vasculitis. **(g)** Scaling and crusting. **(h)** Keratosis pilaris. **(i)** Lichenification.

With respect to gender distribution, 63.3% of the participants were female (n=19) and 36.7% were male (n=11). Age stratification by sex revealed that the majority of the females (89.5%) and males (72.7%) were in the 21–40 years age bracket.

8.2 Clinical Parameters of Disease Severity

The mean percentage of body surface area involved was 19.8% (SD = 12.6), ranging from 4.5% to 4%.

8.3 Assessment of SCORAD Parameters

The severity of individual SCORAD parameters was distributed as follows:

- **Erythema:** The Most common grades were moderate (43.3%) and mild (40%); severe erythema was seen in 13.3% of participants.
- **Edema/Papulation:** Mild edema/papulation was the most frequent finding (60%), followed by none (23.3%), and moderate (13.3%).
- **Excoriations**: Mild (46.7%) and moderate (36.7%) excoriations were common, while severe excoriations were seen in 10%.
- **Oozing/Crusting:** A large majority (73.3%) exhibited no oozing/crusting, while 23.3% had mild findings.
- **Lichenification:** 46.7% had no lichenification, while 36.7% had mild and 13.3% had moderate.

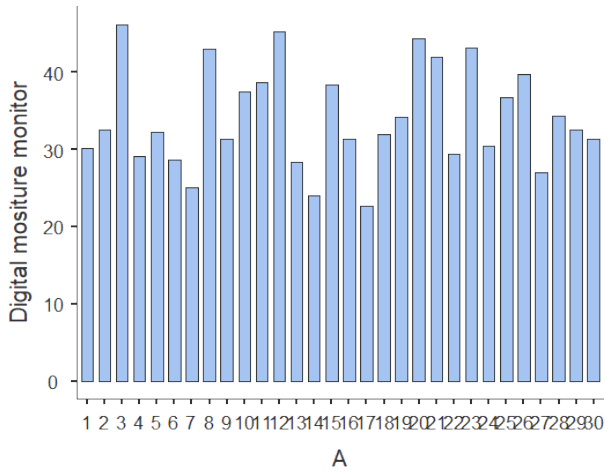

Chart 2. Score of moisture level

Table 3. Grading of SCORAD

Digital Moisture		SCORAD Score Grade		
		Moderate	Severe	Total
Dry	Observed	7	12	19
	% within column	43.80%	85.70%	63.30%
Normal	Observed	9	2	11
	% within column	56.30%	14.30%	36.70%
Total	Observed	16	14	30
	% within column	100%	100%	100%

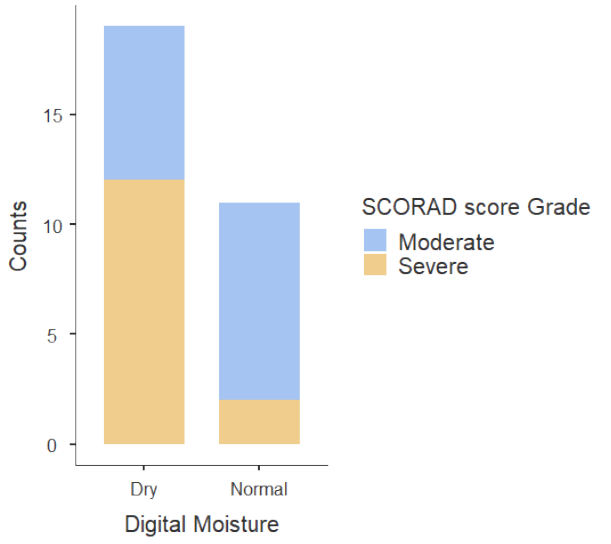

Chart 3. Grading of SCORAD

Table 4. P value

X2 Tests			
	Value	df	p
X2	5.66	1	0.017
N	30		

8.4 Subjective Symptoms

The mean pruritus score reported was 3.77 (SD = 1.81) on a visual analogue scale, with a range from 1 to 7. A similar pattern was seen for sleep loss, also with a mean of 3.77 (SD = 1.81).

8.5 SCORAD Index

The SCORAD index, which integrates objective signs and subjective symptoms, showed a mean score of 53.9 (SD = 17.1), with a minimum of 26.6 and a maximum of 88.4. Based on severity grading:

- **Moderate AD** was observed in 53.3% (n = 16).
- **Severe AD** in 46.7% (n = 14).

No participants were classified as having mild disease.

8.6 Skin Hydration Assessment

Digital skin moisture readings were recorded using a calibrated device. The mean value was 34.1 (SD = 6.5), with a minimum of 22.7 and a maximum of 46.2.

Based on hydration levels:

- 63.3% (n = 19) of participants had dry skin
- 36.7% (n = 11) had normal hydration levels

8.7 Association between Skin Hydration and Disease Severity

A cross-tabulation of SCORAD severity grades with digital moisture levels revealed:

- Among participants with severe AD, 85.7% (n = 12) had dry skin
- In contrast, among those with moderate AD, only 43.8% (n = 7) had dry skin, while 56.3% (n = 9) had normal hydration

A Chi-square test showed a statistically significant association between dry skin and severe disease severity ($\chi^2 = 5.66$, df = 1, p = 0.017), indicating that decreased skin hydration is significantly correlated with increased clinical severity in AD patients.

9. Discussion

This study aimed to evaluate the clinical profile, severity grading, and skin hydration status in patients with

Atopic Dermatitis (AD), using both objective clinical scoring (SCORAD index) and digital skin moisture assessment. The findings offer important insights into the relationship between skin barrier function, disease severity, and demographic patterns among adult AD patients.

9.1 Demographic Trends

The study population comprised predominantly female participants (63.3%), consistent with prior epidemiological data suggesting a higher prevalence of AD in females, especially in adulthood[1]. The majority (83.3%) of participants were aged between 21 and 40 years, highlighting that AD remains clinically significant beyond childhood, affecting young and middle-aged adults with substantial disease burden. These findings corroborate studies that emphasize adult-onset or persistent AD in this age group[4].

9.2 Clinical Severity and SCORAD Profile

The SCORAD (Scoring Atopic Dermatitis) index, a validated tool for assessing disease severity, revealed a mean score of 53.9, with nearly equal distribution between moderate (53.3%) and severe (46.7%) grades. This high burden of disease severity indicates inadequate disease control or chronicity in a significant proportion of patients. Notably, none of the participants had mild disease, reflecting the clinical need for more aggressive or tailored management approaches in the studied population.

Component-wise analysis of SCORAD parameters revealed that moderate erythema and edema/papulation were the most prevalent objective signs. Additionally, excoriations and lichenification were common, pointing toward chronic scratching and barrier disruption. Subjective symptoms such as pruritus and sleep loss, both scoring an average of 3.77 on a 10-point scale, underscore the impact of AD on patient quality of life and daily functioning—an aspect well-documented in earlier literature[8].

9.3 Skin Hydration and Barrier Function

A key objective of the study was to assess skin barrier integrity through digital moisture monitoring, a non-invasive and reproducible method for evaluating stratum corneum hydration. The mean hydration value was 34.1%, with 63.3% of patients classified as having dry skin. These findings align with the central pathophysiological hallmark of AD—epidermal barrier dysfunction, characterized by reduced natural moisturizing factors and impaired lipid profiles[6]. Importantly, a statistically significant association was found between low moisture levels and higher disease severity ($p = 0.017$), reinforcing the pivotal role of barrier impairment in disease exacerbation. Among patients with severe AD, 85.7% had dry skin, compared to only 43.8% among those with moderate disease. This suggests that hydration status may serve as a potential surrogate marker for disease severity, supporting earlier claims that skin hydration correlates inversely with trans epidermal water loss and inflammation[5].

9.4 Clinical Implications

The study emphasizes the need for barrier-targeted therapy in AD management, including emollients, ceramide-based moisturizers, and occlusive agents. While systemic treatments aim to control immune dysregulation, an adjunctive focus on restoring epidermal integrity is vital for achieving long-term remission and improving quality of life[9]. Moreover, routine use of digital moisture monitors in clinical settings may help personalize therapy and monitor treatment response in real-time.

Additionally, the high levels of excoriations and lichenification in patients with moderate and severe AD reflect the cyclical nature of pruritus and scratching, further aggravating barrier disruption. Patient education, antipruritic strategies, and behavioral interventions must be considered integral to comprehensive care.

10. Summary

Skin barrier dysfunction is central to the pathogenesis of atopic dermatitis. Accurate assessment using clinical, biophysical, and molecular methods is critical for diagnosis, treatment monitoring, and prevention. As technology advances, barrier-based personalized therapy holds great promise for improving AD management. The major factor contributing to new and relapsing lesions in Atopic Dermatitis is skin dryness (Xerosis), which initiates the itch-scratch cycle, followed by an immunological reaction characterized

by erythema, Edema, and oozing in affected areas. Secondary infection follows in oozing lesions. Recurrent flare-up cycles of Atopic Dermatitis lesions induce thickening and lichenification of the affected sites.

- Detailed patient awareness about the skin barrier is mandatory.
- Education regarding trigger avoidance, like food and airborne allergens, infections, stress, irritants and habitual scratching.
- The initial prime care for the xerotic skin with moisturizers is the cornerstone to restore the barrier and prevent trans epidermal water loss in Atopic dermatitis, thereby we can improve the Quality of life in patients with AD.

Future research should focus on integrating molecular, microbiological, and biophysical data to create comprehensive barrier assessment models.

11. Limitations and Future Scope

This study is limited by its small sample size and cross-sectional design, which restricts causal inferences. Moreover, environmental and seasonal factors, which can influence skin hydration, were not controlled. Future studies with larger cohorts, longitudinal follow-up, and interventional arms focusing on barrier repair therapies could further validate digital hydration measures as biomarkers for disease severity and therapeutic efficacy.

12. Conclusion

In conclusion, this study highlights the substantial clinical severity and impaired skin hydration among adult AD patients. The significant association between skin dryness and SCORAD grade underscores the centrality of barrier dysfunction in disease pathogenesis. Incorporating objective skin hydration assessment alongside conventional severity scales may enhance clinical monitoring and optimize personalized treatment strategies in Atopic Dermatitis.

13. References

1. Swamy AV, Surendran KAK, Swamy NBL, Bangaru H. Epidemiological profile and clinical pattern of atopic dermatitis in South Indian teaching hospital. IP Indian Journal of Clinical and Experimental Dermatology. 2019; 5(2):146-153. https://doi.org/10.18231/j.ijced.2019.032
2. Silverberg JI, *et al.* Epidemiology of atopic dermatitis: clinical implications. Allergy Clin Immunol Pract. 2021.
3. Barbarot S, et al. Epidemiology of atopic dermatitis in adults. J Eur Acad Dermatol Venereol. 2018.
4. Stalder JF, Barbarot S, Wollenberg A, *et al.* Patient-oriented SCORAD (PO-SCORAD): A new self-assessment scale in atopic dermatitis validated in Europe. Allergy. 2011; 66(8):1114-21. https://doi.org/10.1111/j.1398-9995.2011.02577.x PMid:21414011.
5. Celakovska J, Bukac J. The severity of atopic dermatitis evaluated with the SCORAD index and the occurrence of bronchial asthma and rhinitis, and the duration of atopic dermatitis. Allergy Rhinol (Providence). 2016; 7(1):8-13. https://doi.org/10.2500/ar.2016.7.0144 PMid:27103554 PMCid:PMC4837137.
6. IADVL'S Concise Textbook of Dermatology; 2019.
7. Elias PM, Steinhoff M. Outside-to-inside (and now back to "outside") pathogenic mechanisms in atopic dermatitis. J Invest Dermatol. 2008. https://doi.org/10.1038/jid.2008.88 PMid:18408746 PMCid:PMC2675555.
8. Sarkar R, Das A. Atopic Dermatitis: Text and Atlas; 2022.
9. Ellis CN, Mancini AJ, Paller AS, Simpson EL, Eichenfield LF. Understanding and managing atopic dermatitis in adult patients. Semin Cutan Med Surg. 2012; 31(3 Suppl):S18-22. https://doi.org/10.1016/j.sder.2012.07.006 PMid:23021781.
10. De Benedetto, Kubo A, Lisa A. Skin barrier disruption: A requirement for allergen sensitization. J Invest Dermatol. 2012; 132(3 Pt 2):949-63. https://doi.org/10.1038/jid.2011.435 PMid:22217737 PMCid:PMC3279586.
11. Kim BE, *et al.* Barrier abnormalities and immune dysregulation in atopic dermatitis. J Allergy Clin Immunol. 2019.
12. Drucker AM, *et al.* Atopic dermatitis: burden of disease and evidence-based treatment. Am J Clin Dermatol. 2017.

Annexure

MASTER CHART

ID.no	Age	Sex 1- M, 2- F	SCORAD SCORE PARAMETERS										SCORAD SCORE - A/5+7B/2+C (i.e. mild if SCORAD <25, moderate if 25 ≤ SCORAD ≤ 50 and severe if SCORAD > 50).	Digital Moisture monitor - % (0-35=dry,35 - 60 =normal ,60 to 100=excellent)
			A - Extent - percentage of area involved % (Ref Diagram)	B - Intensity (none-0,mild-1,moderate-2,severe-3)							C - Subjective symptoms (0 to 10)- Average for the last 3 days or			
				Erythema	Edema/ papulation	Oozing/ crusting	Excoriations	Lichenification	Dryness in uninvolved skin	Total	pruritus	sleep loss		
1	27	2	27	1	1	1	2	1	1	7	5	5	59.4	30.2
2	30	2	18	1	1	1	1	1	1	6	3	3	48.6	32.5
3	13	1	9	1	0	0	1	1	1	4	1	1	30.8	46.2
4	37	2	27	2	1	2	2	2	2	11	6	6	88.4	29.1
5	38	2	19	1	1	0	2	1	1	6	3	3	48.8	32.3
6	40	2	18	2	1	0	1	0	3	7	5	5	57.6	28.6
7	45	2	9	2	1	0	2	1	3	9	4	4	68.8	25.1
8	35	2	45	2	1	0	2	0	1	6	7	7	58	43
9	27	2	9	3	1	0	1	0	1	6	5	5	48.8	31.3
10	34	1	13.5	1	0	0	2	0	2	5	6	6	43.7	37.5
11	29	1	4.5	2	2	1	1	1	1	8	3	3	59.9	38.7
12	40	2	18	2	0	0	1	2	1	6	3	3	48.6	45.3
13	36	2	36	1	0	0	2	3	3	9	2	2	72.2	28.3
14	27	1	54	2	1	1	3	1	2	10	5	5	85.8	24
15	41	1	18	2	0	0	1	0	1	4	2	2	33.6	38.3
16	22	2	22.5	1	1	0	1	0	2	5	3	3	42.5	31.4
17	19	1	18	3	2	1	1	1	2	10	3	3	76.6	22.7
18	32	1	13.5	1	1	0	1	0	1	4	1	1	31.7	32
19	37	2	36	2	1	0	1	0	1	5	2	2	44.2	34.2
20	30	1	9	3	1	0	2	0	0	6	3	3	46.8	44.3
21	39	2	9	2	1	0	0	0	1	4	1	1	30.8	42
22	34	2	45	1	1	0	3	2	2	9	4	4	76	29.4
23	40	1	9	0	0	0	2	1	1	4	5	5	34.8	43.2
24	21	2	4.5	2	2	0	1	0	1	6	1	1	43.9	30.5
25	39	2	9	2	2	0	1	0	1	6	5	5	48.8	36.7
26	33	2	18	1	1	0	0	0	1	3	2	2	26.6	39.7
27	36	1	22.5	1	0	0	2	1	2	6	5	5	51.5	27
28	23	2	18	2	1	1	2	1	1	8	6	6	65.6	34.3
29	20	2	9	3	3	0	1	0	1	8	7	7	64.8	32.5
30	39	1	27	1	1	1	3	2	2	10	5	5	80.4	31.3