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Profile of Lymphocytic and Granulocytic Activities in Senegalese Patients with Atopic Dermatitis According to Disease Duration

Frederic Christian Diaz[1], Bambi Ngom[1], Khadim Diop[2,3], Mame Téné Ndiaye[2,3], Moustapha Mbow[1,4], Tandakha Ndiaye Dieye[1,5], Maguette Deme Sylla Niang[1,6], Alioune Dieye[1], Oumou S. Niang[2,7], Boubacar Ahy Diatta[2,7], Babacar Mbengue[1,7]

[1]Laboratoire d'Immunologie, Faculté de Médecine, Pharmacie, Odonto-Stomatologie, Université Cheikh-Anta-Diop, Dakar, Senegal
[2]Service de Dermatologie, Université Cheikh-Anta-Diop, Dakar, Senegal
[3]Service de Dermatologie, Hôpital Albert Royer, Dakar, Senegal
[4]Laboratoire de Biologie Médicale, Hôpital Militaire de Ouakam, Dakar, Senegal
[5]Laboratoire de Biologie Médicale, Centre National de Transfusion Sanguine, Dakar, Senegal
[6]Laboratoire de Biologie Médicale, Hôpital Général Idrissa Pouye, Dakar, Senegal
[7]Service de Dermatologie, Hôpital Aristide le Dantec, Dakar, Senegal
Email: Fredericchristian.diaz@ucad.edu.sn, christiangivenchy@gmail.com

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Abstract

Atopic dermatitis is an inflammatory skin disease with a juvenile tendency (15% - 20%) of cases with a chronic component that could justify the prevalence in adults (7%). Thus, eczematous-type lesions characterize it across various ethnic phenotypes. This pathology is well known in Europe, America, and Asia, but in Africa, especially in sub-Saharan Africa, poor data are available about atopic dermatitis immunopathology. In this African context, we proceeded to study the physiopathology of this pathology by evaluating the activation levels of immune cells, more specifically the level of lymphocyte activation and the membrane expression levels of the FcεRI-IgE complex on granulocytic cells. Our study is conducted from a descriptive and analytical case-control perspective over a period of 3 months, from December 2, 2021, to March 2, 2022, at Le Dantec medical center. The study population was diagnosed in the dermatology department by specialists based on the analysis of their clinical symptoms with the United Kingdom Working Party (SCORAD, CLDQI/DLQI); Statistical data were carried out using non-parametric tests with significance relief at $p < 0.05$. Cell analysis was done by flow cytometry with the SYSMEX Cube 8 to assess their activation through membrane expression of the complex FcεRI-IgE on granulocytes and lymphocytes. Overall, neutrophils are the most important cells in patients and healthy subjects, and normal blood lymphocyte lev-

els were observed; no significant differences were observed between patients and controls in these two cell populations. TCD8+ populations are the most blood lymphoid cells isolated ($p < 0.001$). The levels of eosinophils and basophils obtained in the patients were found to be the highest (p: 0.018; p: 0.149); however, they presented low levels of activation characterized by low membrane expression of FcεRI-IgE on granulocytes. Correlations between disease evolution and IgE/RfcE1 membrane expressions on basophils were found (Ro: 0.640; $p < 0.05$; Ro: 0.603, p: 0.027). Indeed, basophils and eosinophils expressing membrane IgE had a similar trend (Ro: 0.634, p: 0.0231). Our results are in contradiction with studies conducted on Afro-Americans and South Africans, in which the Th2 immune response was dominant. This is a preliminary study on A.D in Senegal, so we need to be cautious with these results, given the small number of patients and the need for further immunological and genetic investigations.

Keywords

Atopic Dermatitis, Lymphocytes, Granulocytes, IgE, FcεRI, Immuno-Allergy

1. Introduction

Atopic dermatitis is an inflammatory skin disease characterized by a relative degree of chronicity. It is highly prevalent in children (10% - 20%) and is also observed in adults (1% - 5%), a pattern that, according to several authors, underscores the chronic nature of this predominantly childhood condition [1]-[4].

This inflammatory skin disease is highly complex from a clinico-biological perspective, which underlies the difficulties observed in its immunological classification. Indeed, several classification systems have been developed, of which the endotype classification is the most widely used. The phenotypic classification is based on two components: extrinsic and intrinsic. The extrinsic form posits an external origin linked to environmental factors and is characterized by normal to subnormal IgE levels [5]-[9]. In addition to these phenotypic classification challenges, there is a considerable diversity in the immunological reactivities involved across different races and ethnic profiles [10].

Beyond these clinico-biologica parameters, atopic dermatitis also constitutes a major public health problem. This is due, on one hand, to its significant economic impact in terms of healthcare costs and occupational burden, amounting to 5 billion dollars in the US, and on the other hand, to the ensuing psycho-social consequences, such as social stigmatization, depression, and suicide [11]-[14]. While this skin condition is well known in Western countries and Asia, where it is the subject of extensive clinical and immunological studies, it remains under-recognized and underestimated in Sub-Saharan Africa, particularly in Senegal, as evidenced by the very limited number of studies reported in the literature [15]. Furthermore, in Senegal, atopic dermatitis is confronted with deep-seated traditional

beliefs from both clinical and therapeutic perspectives (involving traditional practitioners), which delay and complicate patients' diagnosis and treatment management.

Within this context of clinical and biological uncertainty, it has become essential to investigate the pathophysiology of atopic dermatitis in Sub-Saharan Africans, specifically Senegalese patients. This will be achieved by studying the activation profiles of immune cells in Senegalese individuals with atopic dermatitis, with the ultimate goal of optimizing therapeutic management for these patients [16].

2. Materials and Methods

1) Recruitment and Clinical Management

This study included 13 patients and 23 healthy individuals without a history of atopic skin conditions, such as atopic dermatitis, recruited between December 2, 2021, and March 3, 2022, from the dermatology department of the Aristide le Dantec Hospital in Dakar, Senegal. All the patients except one had a long therapeutic history managed by a dermatologist from the dermatology department. The clinical diagnosis is based on the United Kingdom Working Party criteria [17]-[19]. Recruitment included all patients with an atopic history, presence of itching and eczematous lesions in the folds, on the extensor surfaces of the limbs, and in the convex areas, and presence of xerosis! Patients with an incorrect medical document were excluded. Atopic dermatitis therapy depends on the patients' age, the severity of their symptoms (local or systemic), and treatment resistance. In Senegal, therapeutic management of atopic dermatitis follows international recommendations, such as European guidelines on atopic eczema [20] [21] or French guidelines [22] from the French Society of Dermatology and Venereology. Atopic dermatitis baseline therapy focuses on the use of emollients, avoidance of allergens and educational programs [20] [21] for adults and children/adolescents; Subsequently, for mild to moderate forms, treatment is based on the use of topical corticosteroids associated with wet wraps for the acute forms, topical calcineurin inhibitors for the reactive forms, Furthermore, an intense psychosomatic counseling is highly recommended [20] [21]; In severe and recurrent cases, therapeutic management may involve methotrexate or azathioprine; systemic glucocorticoids should be used only as a rescue therapy [20] [21]; Biotherapy (Dupilumab, Upadacitinib) aren't use due their expensive price in Senegal. Healthy controls were recruited at the University Cheikh Anta DIOP based on their age class; indeed, people with a history of atopy or recent infection were excluded! A medical check associated with a hemogram was done for each individual.

2) Sampling

A venous blood sample was collected into three EDTA tubes. One was used for a complete blood count (CBC) in hematology, and the other was used for immunophenotyping by flow cytometry. Following blood collection, the EDTA tubes were

placed in a cooler with dry ice to maintain a temperature between 15˚C and 25˚C prior to immediate CBC analysis in hematology for one of the tubes. A cell separation by Ficoll-Triosil gradient of 5 ml blood EDTA mixed 1:1 with PBS was used for the isolation of mononuclear cells (lymphocytes) from the whole blood by sedimentation. After a 400 g centrifugation for 30 minutes at room temperature, mononuclear cells formed a thin layer at the interface, and denser cells (granulocytes, red blood cells) passed through the Ficoll solution.

One tube was dedicated to numeration by Blue tryptan 0.4% which differentiate viable and non-viable cells. An equal volume of cell suspension and 0.4% Blue tryptan, have been gently shake and let stand at room temperature for 1 - 3 minutes. 10ul of the mix was putted on the hematocytometer for reading on microscope (×10 - 20). The living cells have an intact membrane, appear bright and colorless; Dead cells have a damage membrane, absorb the dye and appear dark blue.

3) Immunophenotyping by Flow Cytometry

Analysis of the leukocyte population in the samples by flow cytometry was performed using a SYSMEX CUBE 8 instrument. To optimize the study of leukocytes, cell labeling was performed using fluorochromes, the detection of which, upon UV exposure, revealed cellular characteristics. Four fluorochromes, Fluorescein isothiocyanate-5 (FITC), phycoerythrin (PE), peridinin-chlorophyll-protein (PerCP), Allophycocyanin (APC) were used in this study to characterize lymphoid cells and granulocytes. Consequently, various panels were developed based on the specific labeling of target cells, enabling their precise isolation and identification (Table 1 and Table 2). Gating strategies are shown in Figure 1 and Figure 2.

Table 1. Groupe 1: P1 to P7.

	FITC	PE	PeRCP	APC
L1	CD3	CD8	CD45	CD4
L2	CD25	CD19	CD45	CD4
G1	CD49d	Rfcε1	CD203c	CD16g
G2	CD49d	IgE	CD203c	CD16g

Table 2. Groupe 2: P8 to P13 + health controls.

	FITC	PE	PeRCP	APC
L1	CD3	CD4	CD45	CD8
L2	-	CD3	CD45	CD25
L3	CD10	CD19	CD45	CD3
G1	CD49d	CD16g	CD203c	Rfcε1
G2	CD49d	CD16g	CD203c	IgE

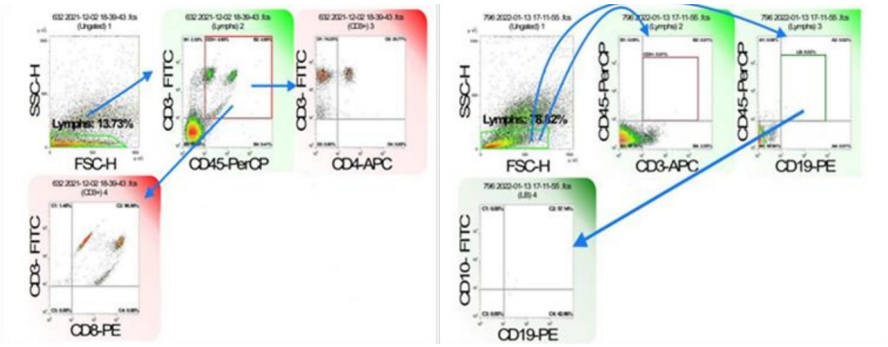

Figure 1. Gating strategy for T-lymphocyte population by immunophenotyping.

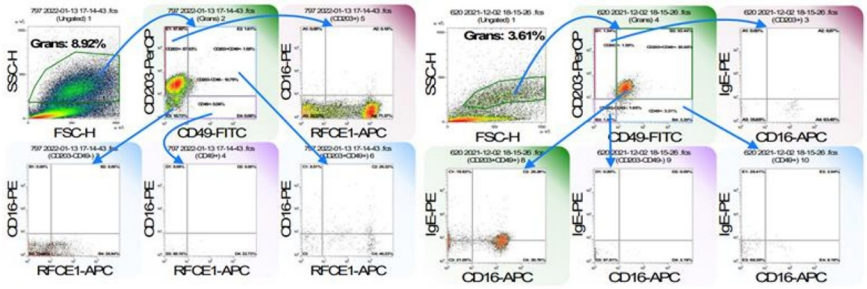

Figure 2. Gating strategy for granulocyte population by immunophenotyping patients and controls.

4) Statistical Analysis

Data were collected using Excel, and statistical analyses were performed with SPSS version 27.0 (IBM) and GraphPad Prism version 11.0.2. Owing to the small sample size (n = 13) in our study, non-parametric tests were employed. These included the Wilcoxon, Mann-Whitney, and Kruskal-Wallis tests for comparisons between variables, as well as Spearman's rank correlation test. Only results with a p-value of less than 0.05 ($p < 0.05$) were considered statistically significant.

3. Results

1) Epidemiological and Clinico-Biological Data

Baseline hematological data were determined for both patients and healthy individuals. The comparison of median ages between patients and healthy controls indicated that patients were generally older, with a median age of 33 years compared with 27 years in healthy individuals (p: 0.010). The analysis of lymphocytic and granulocytic populations by immunophenotyping in Figure 1 and Figure 2 revealed that only the eosinophil count was found to be significantly higher in patients with atopic dermatitis (0.24 vs 0.16, p: 0.0193). The other leukocyte concentration showed no significant differences between patients and healthy individuals ($p > 0.05$) (Table 3).

From a clinical perspective, the mean disease duration observed in patients was 28 years, with a minimum duration of 29 days in one patient and a maximum of 46 years. Furthermore, the majority of patients (77%) presented with

an identical symptomatology, comprising pruritus and even eczematous lesions. Half of the patients (50%) exhibited allergic rhinitis as a comorbid sign; asthma and conjunctivitis were poorly represented in our study population, at 21% and at 0%, respectively. Additionally, a family history of atopic dermatitis was reported at 61.5% of patients. The investigation into the etiological factors of atopic dermatitis in patients revealed that food was the primary trigger for atopic eczema flares (35.5%, p: 0.581), followed by dust mites (23.1%, p: 0.022) and pollens (15.4%, p: 0.013). Other diverse factors contributed to the onset of flares in 15.4% (p: 0.092).

Table 3. Epidemiological and biological characteristics of patients and controls.

	Patients (n = 13)	Healthy Controls (n = 23)	p-Value
Age (Years), Median	33 (27 - 45)	27 (24 - 29)	**0.010**
Leukocyte Populations, Median			
Neutrophils ($\times 10^9$/L)	1,9677 (1,24 - 2,84)	2,2264 (1,14 - 3,17)	0.7979
Lymphocytes ($\times 10^9$/L)	2,262 (1,55 - 3,15)	2,2836 (1,05 - 6,63)	0.1565
Eosinophils ($\times 10^9$/L)	0,24 (0,1 - 0,46)	0,1614 (0,02 - 0,7)	**0.0193**
Basophils ($\times 10^9$/L)	0,0269 (0,01 - 0,04)	0,0218 (0,01 - 0,06)	0.122

2) Variations in Lymphocytic Cellularity between Patients and Healthy Controls

Analysis of the data revealed a significantly higher proportion of cells expressing the CD3 marker (T lymphocytes) in patients with atopic dermatitis (p: 0.01), whereas cells expressing the CD19 marker were more prevalent in healthy controls ($p < 0.001$), as shown in **Figure 3.** Furthermore, lymphoid cells co-expressing the CD3 and the CD8 markers (T cytotoxic) were significantly more abundant in patients with atopic dermatitis ($p < 0.001$). The inverse was observed in healthy individuals regarding cells co-expressing the CD3 and CD4 markers, characteristics of T-helper lymphocytes (p: 0.022) (**Figure 4**). The investigation of lymphocyte markers in relation to disease duration revealed a single significant negative correlation with CD3 markers, characteristic of the T-lymphocyte population in patients (Ro: −0.686, p: 0.020). No other marker showed a significant correlation in this context ($p > 0.05$). A positive correlation was observed between lymphocytes expressing the CD3 marker (T-lymphocytes) and lymphocytes co-expressing the CD3 and CD8 markers (Ro: 0.417; $p = 0.013$). Conversely, a negative correlation was found between lymphocytes expressing the CD3 marker and those expressing the CD19 marker (B cell lymphocytes) (Ro: −0.352; $p = 0.038$); Also, T-helper lymphocytes (LTCD3+CD4+) exhibited a negative correlation with cytotoxic T lymphocytes (CD3+CD8+) (Ro: −0.356; $p = 0.038$). In addition, LTCD3+CD8+ showed a negative correlation with CD19+ lymphocytes.

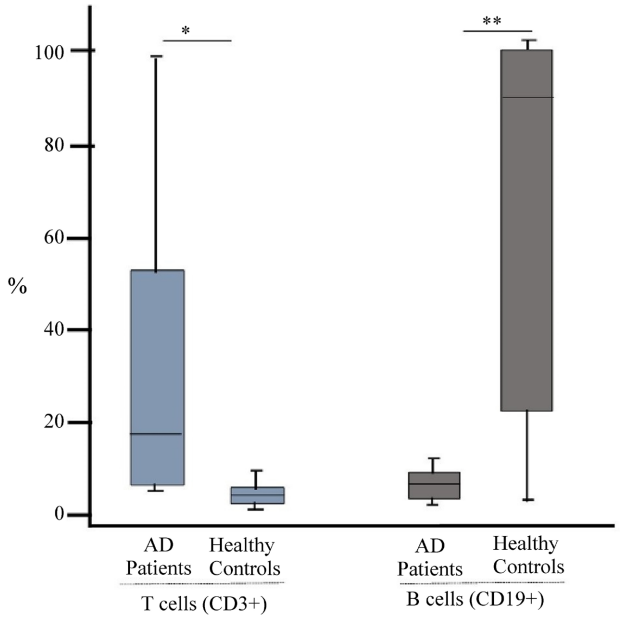

Figure 3. Comparison of CD3+ and CD19+ levels expression on lymphocyte populations in patients and healthy controls.

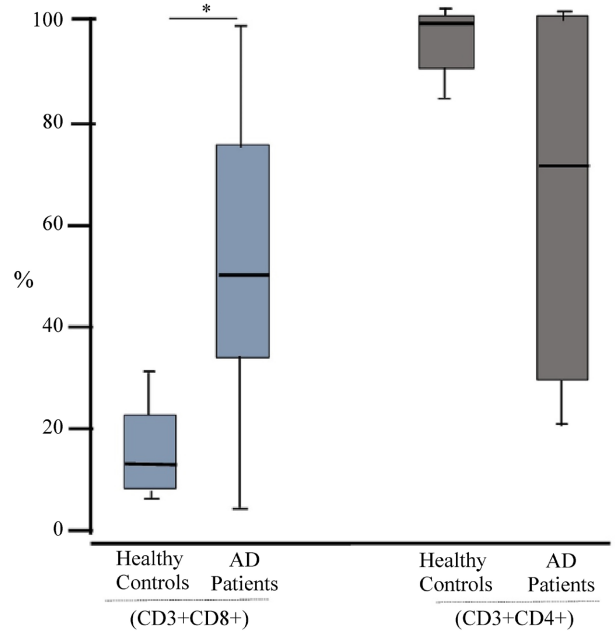

Figure 4. Variations in expression levels of CD3+CD4+ and CD3+CD8+ surface markers on lymphocyte populations in patients and healthy controls.

3) Variation in Granulocytic Cellularity in Patients and Healthy Controls

a. Expression of the FcεRI Receptor

Within the granulocyte population, the analysis revealed that, among basophils, cells that didn't express the surface receptor FcεRI were predominantly elevated in patients with atopic dermatitis, approaching statistical significance ($p = 0.06$). Furthermore, FcεRI expression was very low in patients with atopic dermatitis

($p > 0.05$). In healthy controls, the levels of blood basophil cellularity were low and non-significant ($p > 0.05$) (Table 3). Additionally, a weak positive correlation was observed between basophils that do not express the IgE-specific receptor and those that do (Ro: 0.372, p: 0.030). In the patient cohort, the investigation of basophil cellularity and FcεRI surface expression in relation to symptom periodicity demonstrated a strong association between atopic dermatitis duration and FcεRI expression (Ro: 0.6037, p: 0.020). In patient cohort, the investigation of basophil cellularity and surface expression of FcƐR1 in relation to symptom periodicity demonstrated a strong association between the duration of atopic dermatitis and FcƐR1 expression (Ro: 0.6037, p: 0.020) (Figure 5).

Regarding eosinophil and neutrophil populations, the expression levels of FcεRI were not significantly different in either patient. Furthermore, no significant correlation was observed within the eosinophil population of the patients ($p > 0.05$) (Table 3), nor between the duration of atopic dermatitis and the expression of the IgE-specific receptor (Ro: 0.165; p: 0.628). In healthy controls, the expression levels of the membrane receptor FcεRI on basophils, eosinophils, and neutrophils were found to be non-significant ($p > 0.05$).

b. Membrane Expression of IgE Antibody

Overball, similar to FcεRI expression, the majority of basophils obtained from patients did not express surface IgE ($p > 0.05$). A positive correlation between the expression of the specific surface receptor FcεRI and IgE antibody was observed in patients (Ro: 0.52, p: 0.03) (Figure 6). However, a very strong positive correlation was found between basophils not expressing surface membrane IgE and eosinophils expressing or not expressing surface IgE, with correlation coefficients of Ro: 0.709 (p: 0.007) and Ro: 0.808 (p: 0.001), respectively. Furthermore, eosinophils not expressing membrane IgE exhibited a parallel trend with neutrophils not expressing IgE (Ro: 0.706, p: 0.007). A strong positive correlation was observed between basophils expressing membrane IgE and eosinophils possessing membrane IgE (Ro: 0.63, p: 0.0231) (Figure 7). A positive correlation was observed between atopic dermatitis duration and the expression of IgE antibodies on basophil surfaces in patients (Ro: 0.640, p: 0.034) (Figure 8). In healthy controls, the membrane expression of IgE on basophils, eosinophils, and neutrophils was not significant ($p > 0.05$).

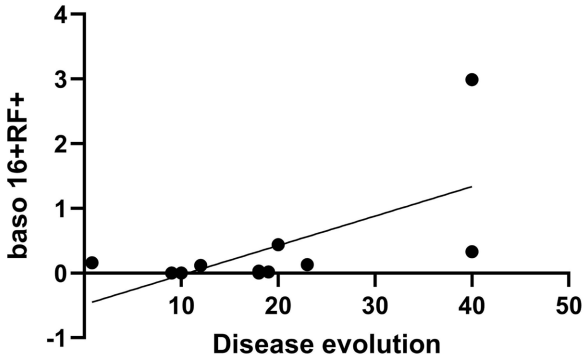

Figure 5. Variation in membrane RfcE1 expression on basophils based on disease evolution.

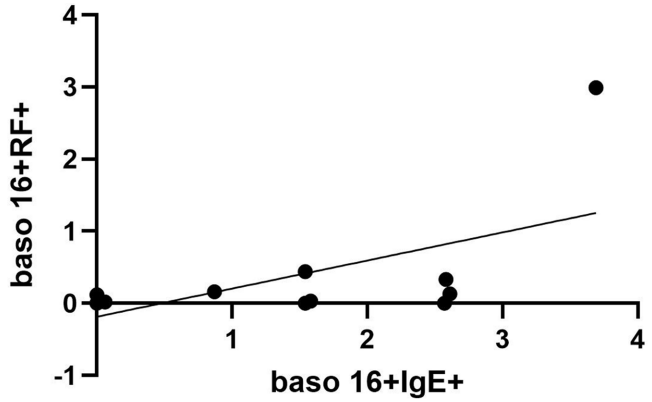

Figure 6. Correlation between membrane IgE and FcεRI receptor expression on basophils.

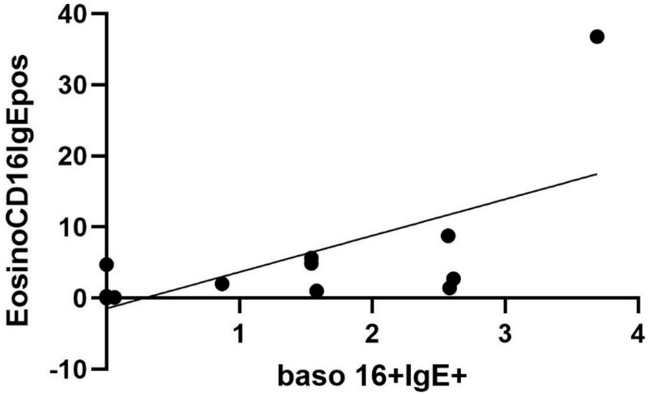

Figure 7. Correlation between membrane IgE and FcεRI receptor expression on eosinophils.

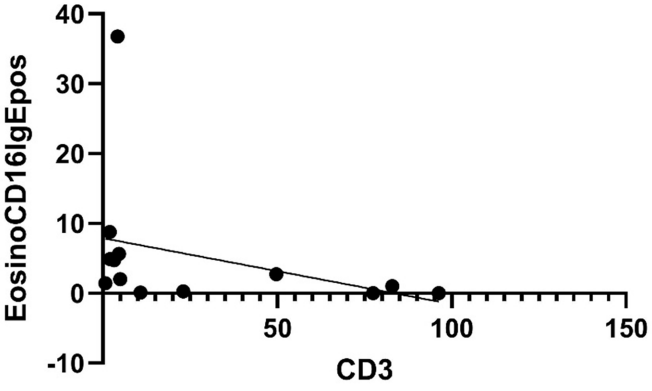

Figure 8. Correlation between T lymphocytes and eosinophils expressing membrane IgE.

Within the eosinophil population, cells exhibiting surface IgE were significantly more abundant in patients with atopic dermatitis ($p < 0.001$). However, no significant correlation was found between IgE expression and FcεRI on eosinophils; furthermore, disease evolution did not impact IgE expression on eosinophils (R1:

0.02, p: 0.95).

4) Lymphocytes and Granulocytes

Overall, no significant correlation was found between blood lymphocyte counts and blood basophil counts in either patients with atopic dermatitis or healthy controls. Within the different lymphocytic subpopulations, strong negative correlations were observed between CD3+T lymphocytes and basophil/eosinophil populations not expressing FcεRI, with correlation coefficients of Ro: −0.632, p: 0.0016 and Ro: −0.566, p: 0.044, respectively. Furthermore, the variation of CD3+T lymphocytes is inversely proportional to eosinophils expressing membrane IgE (Ro: −0.688, p: 0.00114), as shown in Figure 9. Also, the correlation between CD3+CD4+ T-helper lymphocytes and basophils not expressing membrane IgE revealed a negative relationship (Ro: −0.77, p: 0.003). A similar negative correlation was observed between CD3+CD4+T cells and eosinophils expressing membrane IgE (Ro: −0.77; p: 0.003), as well as those not expressing membrane IgE (Ro: −0.542, p: 0.001). Conversely, CD3+CD8+T cells exhibited a positive parallel trend with granulocyte populations, including eosinophils expressing or not expressing membrane IgE (Ro: 0.883, p: 0.000063; Ro: 0.77, p: 0.002) and neutrophils (Ro: 0.663, p: 0.014). CD19+ lymphoid cells (B lymphocytes) showed no significant correlative trend with any granulocytic population (p > 0.05).

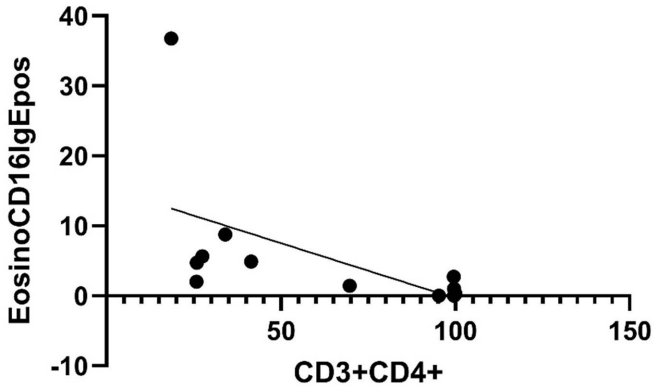

Figure 9. Correlation between membrane IgE on eosinophils and Thelper lymphocytes.

4. Discussion

According to the literature, atopic dermatitis is a common chronic inflammatory skin disease worldwide, characterized by a juvenile predominance that often persists in adulthood [13] [23]-[29]. It has been extensively studied in Western [30], Asian [31] [32], and American populations [33]. However, despite its high prevalence in West Africa, very few, if any, immunological studies have been conducted, leading to significant uncertainty in the diagnosis and therapeutic management of the condition. This study aimed to investigate the activity of various T and B lymphocytes populations as well as granulocytes, by analyzing the membrane expression of IgE and its surface receptor FcεRI in patients regularly monitored by specialists in the dermatology-venerology department. Our epidemiological findings

indicate that both patients and healthy controls were adults with relatively similar mean ages, which is ideal for assessing the chronic nature of the disease. Furthermore, the female predominance observed in our study (sex ratio: 0.6) has been reported by other research teams. Indeed, hormonal influences, particularly estrogens and progesterone, which are suggested to stimulate Th2-type immunity and suppress Th1 responses, have been implicated in atopic dermatitis [34]. The classic cutaneous symptomatology observed in the majority of patients, consisting of eczema associated with comorbidities such as allergic rhinitis and asthma, constitutes the Atopic March, a clinical theory supported by numerous scientific studies [35]-[38]. Food [8], dust mites, pollen, and other environmental factors were identified as the most recurrent triggers of atopic dermatitis flares in our patients, aligning with the most commonly documented etiological factors in the literature [39] [40]. From a biological perspective, patients exhibited isolated hypereosinophilia ($p < 0.05$), and the basophil population expressing neither FcεRI nor membrane IgE was low and non-significant in patients ($p > 0.05$) and absent in healthy controls ($p > 0.05$). A strong positive correlation was observed between the various granulocyte populations not expressing IgE and eosinophils that did ($p < 0.05$). This reflects the synchronized cellular immune dysregulation observed during atopic dermatitis flares, thereby characterizing the inflammation involved [41]. Furthermore, an increase in the expression of membrane IgE antibodies on blood basophils and eosinophils was correlated with disease duration among our recruited patients. In healthy subjects, the membrane expression of IgE and its specific receptor, FcεRI, on granulocytes was found to be absent. Analysis of lymphocytic population data revealed a predominance of the cytotoxic lymphocyte phenotype in patients, whereas T-helper lymphocytes were more abundant in healthy controls. Furthermore, the level of cytotoxic T lymphocytes was proportional to that of T lymphocytes, and both were correlated with the duration of atopic dermatitis among the recruited Senegalese patients. This consequently suggests a shift towards a Th1 immunological profile with increasing disease duration. In addition to the findings regarding membrane expression of IgE and its type 1-specific receptor on granulocytes, our work revealed an inverse relationship between T-lymphocyte levels and the expression of IgE on eosinophils and of its FcεRI on granulocytes. A similar inverse relationship was observed between T-helper lymphocytes (CD3+CD4+) levels and the expression of membrane IgE on granulocytes. Cytotoxic (CD3+CD8+) showed a parallel trend with blood eosinophil levels, particularly those expressing surface membrane IgE. These results, obtained from patients receiving regular and specialized follow-up by dermatologists in a hospital setting, reflect the impact of structured management in atopic dermatitis. Thus, although overall only hyperbasophilia was noted, we observed weak to non-existent granulocytic cellular activation (basophils, eosinophils, neutrophils) associated with a normal lymphocyte count, characterized by a predominance of cytotoxic blood lymphocytes. This contrasts with studies conducted on other populations, notably caucasians [42], asians [32], and African Americans [43] [44]. For instance, in Caucasians, a predominance of the adaptive Th2 immune responses

associated with Th22 lymphocytes in acute forms [5] [45] is characterized by activation of B lymphocytes that produce massive amounts of IgE, which has a tropism for basophils, eosinophils, and mastocytes. Their cellular activation upon binding results in massive degranulation of proteinaceous and enzymatic substances with lytic effects on cells and connective tissues, leading to deleterious outcomes [44] [45]. In chronic forms, a Th2/Th1 balance characterized by the involvement of cytotoxic lymphocytes exerting lytic effects on connective tissue cells has been reported [7]. In Asian populations, the immune response is dominated by a cytotoxic profile characterized by Th17 lymphocytes, conferring a "Psoriasis-Like" phenotype [46]. African Americans who have been the subject of a few studies have presented a Th2 profile similar to that of Caucasians [47]. In Senegal, the management of atopic dermatitis involves several stages: for mild acute forms, administration of anti-inflammatory drugs associated with topical agents (superfatted soap and body milks) [11] [47] [48]. For severe acute forms, a combination of NSAIDs and corticosteroids supplemented by the cutaneous application of topical agents with local actions [49]. For severe and recurrent forms, management is based on methotrexate, with anti-inflammatory drugs and topical agents for localized action [50]. Upon recruitment, the majority of patients were on therapy with NSAIDs or even corticosteroids during episodes of cutaneous eczema flares, which may account for the low level of lymphocytic and granulocytic cellular activation. The data obtained in this study, although preliminary, suggest a therapeutic hypothesis involving immunotherapy targeting basophils and eosinophils with anti-IgE antibodies, combined with cytotoxic lymphocyte targeting. Alongside this therapeutic aspect, it's necessary to define a cytokine profile for patients with acute and chronic atopic dermatitis in Senegal to precisely optimize treatment and achieve an optimal and efficient resolution of the disease during flare episodes.

5. Conclusion

This preliminary study is the first step in the immunological exploration of atopic dermatitis in Senegal. This allowed us to assess the level of cellular activation in patients regularly followed by dermatologists in a specialized hospital department. The resulting implication is the prospect of targeting a specific cellular phenotype within the framework of personalized therapy. However, it appears essential to pursue further work, on the one hand, by studying cellular activation levels in blood and cutaneous lymphocytic and granulocytic phenotypes associated with the various cytokines produced in a larger patient population, and, on the other hand, by conducting genetic and proteomic profiling in these same patients.

Consent for Publication

All data collected were derived from the analysis of a file accompanied by a consent form that was freely read and approved by each patient and each healthy control.

Author Contributions

Conceptualization: Frédéric C. Diaz and Boubacar A. Diatta; Methodology: Frédéric C. Diaz; Software: Frédéric C. Diaz; Validation: Babacar Mbengue, Maguette D. S. Niang, Alioune Dieye, and Oumou S. Niang; Formal Analysis: Bambi Ngom; Investigation: Bambi Ngom, Khadim Diop, Mame Téné Ndiaye, and Frédéric C. Diaz; Resources: Babacar Mbengue, Alioune Dieye, Maguette S. Niang, and Oumou S. Niang; Data Curation: Frédéric C. Diaz and Bambi Ngom; Writing-Original Draft Preparation: Frédéric C. Diaz; Writing-Review and Editing: Frédéric C. Diaz, Babacar Mbengue, and Boubacar A. Diatta; Visualization: Moustapha Mbow; Supervision: Babacar Mbengue and Boubacar A. Diatta; Project Administration: Alioune Dieye, Babacar Mbengue, Maguette D. S. Niang, and Oumou S. Niang; Funding Acquisition: Babacar Mbengue and Alioune Dieye.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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