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Serum FcεRI as a Putative Biomarker of Disease Severity and Immune Perturbation in Paediatric and Adult Atopic Dermatitis

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ABSTRACT

Objective: Atopic dermatitis (AD) is a chronic inflammatory skin disease characterised by immune dysregulation. Fc epsilon receptor I (FcεRI) plays a crucial role in AD pathogenesis by mediating IgE-dependent immune responses. This study aimed to investigate the role of free FcεRI in serum and its impact on white blood cell subsets in patients with AD.

Materials and Methods: A total of 110 AD patients aged 1–30 years were recruited from hospitals in Mosul between October 2024 and February 2025. Participants were divided into two age groups: children (1–15 years, n = 38) and adults (16–30 years, n = 32), with age-matched healthy controls (n = 40). Blood samples were analysed for FcεRI levels using enzyme-linked immunosorbent assay and for white blood cell counts via CBC analysis.

Results: FcεRI levels were significantly higher in AD patients compared to controls ($P \leq 0.05$), with children exhibiting higher concentrations than adults. Regression analysis revealed significant correlations between FcεRI levels and white blood cell subpopulations, including neutrophils, lymphocytes, and eosinophils.

Conclusion: These findings suggest that FcεRI contributes to the inflammatory profile of AD, with potential implications for autoimmune disease development. FcεRI may serve as a valuable biomarker for AD severity and immune dysregulation. Future studies should explore its therapeutic targeting to mitigate chronic inflammation in AD patients.

Keywords: Atopic dermatitis, FcεRI, IgE, immune response, inflammation, biomarker

INTRODUCTION

Atopic dermatitis (AD) represents a chronic inflammatory skin disease characterised by severe itching, disruption of the epidermal barrier, and immune, genetic and environmental dysregulation (1). The high-affinity IgE receptor Fc epsilon receptor I (FcεRI) has emerged as a key molecular component in the pathophysiology of AD, serving as a critical interface between allergens and inflammatory cascades (2).

High-affinity FcεRI receptors are central mediators of allergic inflammatory responses, acting as the primary surface receptors for immunoglobulin E on various immune cells. FcεRI is expressed on the surface of human Langerhans epidermal cells, where it facilitates the absorption of allergens associated with IgE and plays a key role in the pathogenesis of AD (3). The receptor consists of an alpha chain responsible for IgE binding, a beta chain involved in signal amplification and two gamma chains necessary for signal transmission (4). When allergens bind to IgE molecules associated with the FcεRI receptor, this leads to cellular activation and the subsequent release of inflammatory mediators that trigger allergic symptoms (5, 6).

The expression of FcεRI extends beyond Langerhans cells to other immune cell populations relevant to the pathophysiology of AD (7). Notably, research has shown

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regulated expression of FcεRI receptors on eosinophils in AD patients compared to other inflammatory skin diseases (8). It is likely that this enhanced receptor expression across multiple cell types amplifies allergic inflammatory emissions in the affected tissues, contributing to the persistence of the inflammatory state characteristic of chronic AD lesions (9). The relationship between FcεRI surface expression and disease activity suggests that these receptors act not only as passive components but as active contributors to the pathogenesis of AD (10) and its onset and progression.

The presence and pathophysiological significance of antibodies against FcεRI receptors in AD presents a complex and contradictory picture based on current research (11). Unlike chronic spontaneous urticaria (CSU), where a large body of evidence supports the presence of antibodies against functional FcεRI receptors, the situation in AD appears more ambiguous (12). Several research groups have demonstrated the presence of antibodies against IgE and/or FcεRI in patients with CSU, with approximately 37% of CSU patients exhibiting spontaneous IgG activity associated with FcεRI receptors. These autoantibodies can activate mast cells and basophils, directly contributing to the pathogenesis of urticaria (13). However, when examining patients with AD specifically, the evidence for similar antibodies targeting the FcεRI receptor is less conclusive (14). One key study investigating IgG antibodies against the FcεRI receptor in various patient groups found anti-FcεRI antibody cross-reactivity in the IgG fraction of AD patients. This study correlated the appearance of FcεRI antigens in serum with the emergence of IgG autoantibodies (15). Soluble FcεRI (sFcεRI) levels are markedly elevated in atopic individuals, including those with AD. Elevated sFcεRI levels may serve as a biomarker for IgE-mediated diseases such as AD, reflecting ongoing immune activation and the development of autoantibodies against sFcεRI (16).

For this reason, the present study aimed to examine the development and appearance of free FcεRI receptors in the serum of patients with AD, investigate the effects of these antigens on different subsets of white blood cells – including lymphocytes, neutrophils, and others – and finally analyse the relationship between FcεRI receptor concentration and changes in white blood cell count to determine the correlation between these variables.

MATERIALS and METHODS

Study Area

Clinical samples were collected from patients attending consultations at Al-Salam, Ibn-Sina and Mosul General Hospitals in the city of Mosul during the period from 10 October 2024 to 1 February 2025. The required tests and practical experiments were carried out in the laboratories of the Department of Life Sciences at the Faculty of Science – University of Mosul and in advanced immunology laboratories in the aforementioned hospitals.

Study Cases

A total of 110 blood samples were collected from individuals aged 1–30 years and divided into four groups: two patient groups (children and adults) and two control groups (children and adults). The reason for dividing the study samples into these groups is that the CBC test showed differences in the control group for children compared to adults, as children are still in the developing stages of their immune systems.

The first group, Group A (the paediatric patient group), included 38 patients of both sexes (men and women) aged 1–15 years who were diagnosed with chronic AD based on the SCORAD test. This patient group corresponded to a control group which included 20 children of both sexes aged 1–15 years, who were healthy and free from any chronic or immune diseases or other medical or health symptoms, and were not receiving any antibiotics, other medications or nutritional supplements.

The second group, Group B, included 32 adult patients of both sexes aged 16–30 years, diagnosed with AD. This patient group corresponded to a control sample of 20 healthy individuals of both sexes aged 16–30 years, who were free from any chronic or immune-mediated diseases, or any other morbid symptoms or health conditions, and were not receiving any treatment with antibiotics, other medications or nutritional supplements

Blood and Serum Collection

Venous blood (5 ml) was withdrawn from all study participants using sterile medical syringes of 5 ml capacity and distributed into two test tubes. The first test tube was of the anticoagulant EDTA type, and 2 ml of the blood sample was placed in it. It was then gently shaken to homogenise the blood and perform a complete blood

count (CBC). The remaining 3 ml of the blood sample was placed in the second test tube, which was of the gel tube type used to separate serum using a centrifuge at 4500 rpm for 10 minutes. The serum was then distributed into small plastic Eppendorf tubes and frozen at −20 °C to measure the concentration of free FcεRI of IgE in serum.

Measurement of the Concentration of Free FcεRI of IgE in Serum

This immunological examination was carried out using the American-made Enzyme-linked immunosorbent assay (ELISA) microplate reader device from the BIOTEK company.

Principle: ELISA is a pre-prepared kit that utilises the sandwich-ELISA dual principle. An antibody against human FcεRI was already added to the ELISA microplate provided with the kit. After the samples (or standards for all control and patient subjects, respectively) were added to the ELISA plate wells, they were combined with the specific antibody. Then, biotinylated antibody specific for human FcεRI, biotin and avidin–horseradish peroxidase (HRP) detection antibody were added to each well sequentially and incubated. The unbound components were washed off. Each well was filled with the standard solution. Only the wells containing human FcεRI, biotin reagent antibody and avidin–HRP developed a blue colour. When the stop solution was added, the enzyme–substrate reaction was terminated, and the colour changed to yellow. A spectrophotometer was used to measure the optical density (OD) at a wavelength of 450 nm. The OD value corresponded to the concentration of human FcεRI. The concentration in the samples was calculated by comparing the OD values with the standard curve.

Complete Blood Count

The Japanese-made (SYSMEX-KX/21N) device was used to perform a CBC test and measure the percentage of white blood cell counts in peripheral blood. The total number of white blood cells and the differential number of each of the cells (neutrophils, lymphocytes, monocytes, eosinophils, basophils) were measured.

Principle: Blood samples were drawn in this study for all patient and control groups, placed in EDTA tubes, mixed well and the device was turned on. The patient's information was confirmed through the electronic panel (name, age and type of examination required). The EDTA tube containing venous blood was placed under the device's needles, and a certain amount of blood was withdrawn by the device for the required measurement. The result was displayed on the screen and then printed on paper.

Ethical Considerations

Before conducting the study, ethical permission was obtained from the Nineveh Health Department / Research and Development Division, preceded by an official request approved by the Scientific Council at the Faculty of Science / University of Mosul. The study adhered to the requirements of public ethics stipulated internationally and in accordance with the Helsinki Convention. Before participating, the participants signed a permission form, stating that all their information would remain completely confidential and be used only for the purpose of scientific research. The tests mentioned in this study were provided free to the participants.

Statistical Analysis

The ANOVA variance analysis test was conducted to test the level of significance among the study groups and compare them with the control sample, in addition to conducting a regression coefficient test to find the impact of some factors on the study variables using the SPSS programme, version 24, at a significance level of $P \leq 0.05$.

RESULTS

The results of the study showed the relationship between age and the prevalence of the chronic phase of AD, where the ages of affected children in the first three years were more frequent than other ages, as shown in Figure 1:

The results also showed the relationship between the age of AD patients and the severity of AD, based on the global SCORAD scale used to determine disease severity, as shown in Figure 2:

The results of the study showed a significant increase in the concentration of FcεRI of IgE in AD patients in the age group A compared to the equivalent control group at a significant difference of 0.000 when $P \leq 0.05$ and as shown in Table (I):

The results of the study also showed a significant increase in the concentration of FcεRI receptor in the serum of IgE in AD patients in the age group B compared to the equivalent control group at a significant difference of 0.032 when the value of $P \leq 0.05$ and as shown in Table (II):

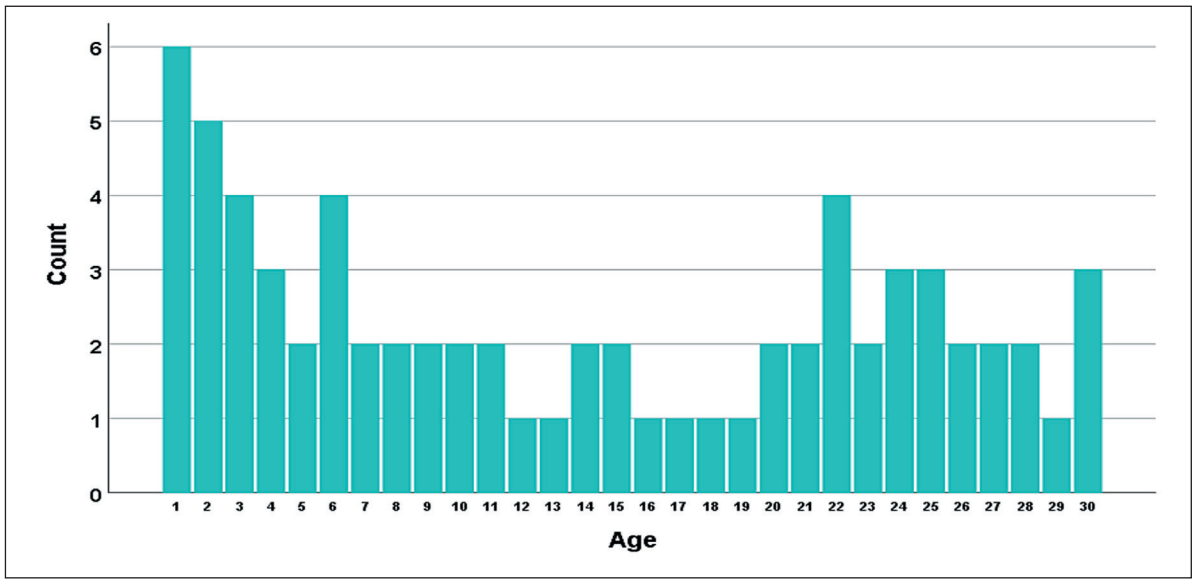

Figure 1: Relationship between the prevalence of AD and the age of patients.

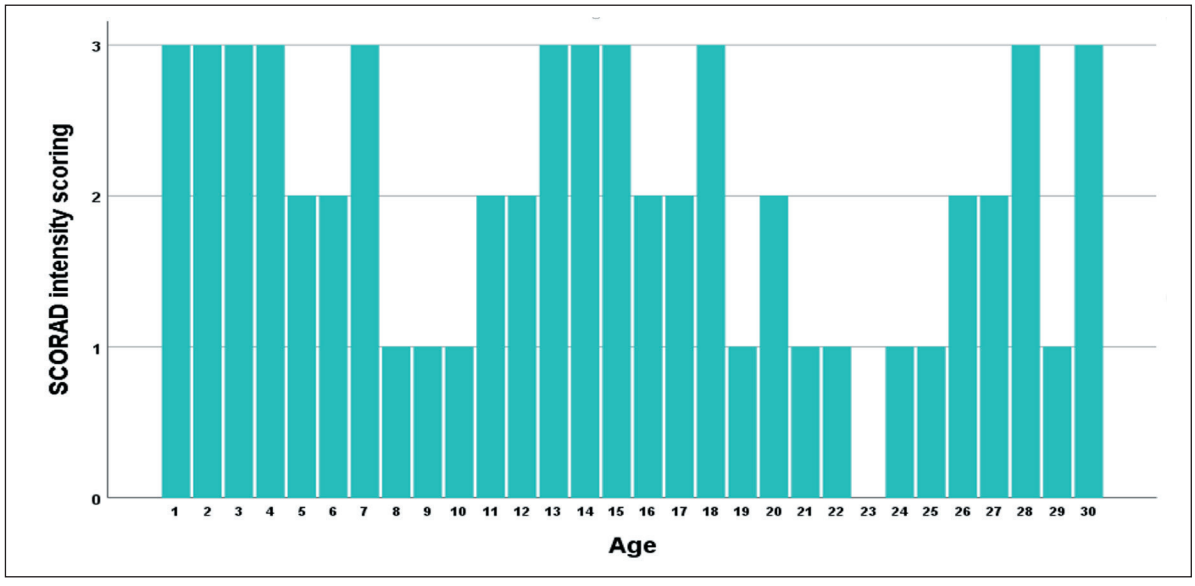

Figure 2: Relationship between injury severity and patients' ages based on the SCORAD scale.

Table I: statistical indicators represented by sample count, mean, standard deviation, for testing the concentration of the FcεRI of IgE in the age group A.

Variable	Group (1-15)	N. Sample	Mean± SD	Extreme value	p-value
FcεRI of IgE ng/ml	Patients	38	6.87 ± 5.14	0.79 -- 19.63	**0.000**
	Control	20	1.81 ± 0.50	1.22 -- 2.90	

$P \leq 0.05$

Table II: statistical indicators represented by sample count, mean, standard deviation, for testing the concentration of the FcεRI of IgE in the age group B.

Variable	Group (61-03)	N. Sample	Mean± SD	Extreme value	p-value
FcεRI of IgE ng/ml	Patients	32	4.10 ± 2.67	0.87 -- 12.07	**0.032***
	Control	20	2.50 ± 1.35	0.79 -- 4.95	

**P ≤ 0.05*

Table III: The regression coefficient independent variable FcεRI of IgE in the group B of study variables WBC count, Neutrophils, Lymphocytes, Monocytes, Eosinophils, and Basophils

Independent variable	Dependent variable	Relationship	Equation	Bi		p-value (b_1)
				b_0	b_1	
FCεRI of IgE ng/ml	WBCc cell/µl	Inverse	$Y = b_0 + (b_1 / X)$	9199.08	4381.05	0.047
	Neutrophils cell/µl	Compound	$Y = b_0 * (b_1 \wedge X)$	3903.93	1.008	0.000
	Lymphocytes cell/µl	Inverse	$Y = b_0 + (b_1 / X)$	3181.84	5368.87	0.003
	Monocytes cell/µl	Compound	$Y = b_0 * (b_1 \wedge X)$	809.92	0.997	0.000
	Eosinophils cell/µl	Compound	$Y = b_0 * (b_1 \wedge X)$	384.80	0.960	0.000
	Basophils cell/µl	Compound	$Y = b_0 * (b_1 \wedge X)$	57.75	0.972	0.000

Analysis of the Regression Coefficient

The benefit of the regression coefficient test lies in evaluating the ability of FcεRI of IgE to affect the white blood cell count by identifying a significant relationship through the regression equation that measures the extent of the effect. In addition, the test provides a value for each pair of variables, helping the researcher determine whether a significant relationship exists. The regression equation can also be represented graphically. This test further allows the extraction of the value of any variable in the study based on its counterpart, using a mathematical formula.

1. Sample patients for category A:

1. The regression coefficient of the independent variable (FcεRI of IgE ng/ml) in the dependent variable (WBCc cell/µL) is a nonlinear relationship of the inverse type (Inverse) and is significant in terms of the probability value, which amounted to (0.047) and is less than (0.05).

2. The regression coefficient of the independent variable (FcεRI of IgE ng/ml) in the dependent variable (Neutrophils cell/µL) is a nonlinear relationship of the combined type (Compound) and is significant in terms of the probabilistic value, which amounted to (0.000) and is less than (0.05).

3. The regression coefficient of the independent variable (FcεRI of IgE ng/ml) in the dependent variable (Lymphocytes cell/µL) is a nonlinear relationship of the inverse type (Inverse) and is significant in terms of the probabilistic value, which amounted to (0.003) and is less than (0.05).

4. The regression coefficient of the independent variable (FcεRI of IgE ng/ml) in the dependent variable (Monocytes cell/µL) is a nonlinear relationship of the combined type (Compound) and is significant in terms of the probabilistic value, which amounted to (0.000) and is less than (0.05).

5. The regression coefficient of the independent variable (FcεRI of IgE ng/ml) in the dependent variable (Eosinophils cell/µL) is a nonlinear relationship of the combined type (Compound) and is significant in terms of the probability value, which amounted to (0.000) and is less than (0.05).

6. The regression coefficient of the independent variable (FcεRI of IgE ng/ml) in the dependent variable (Basophils cell/µL) is a nonlinear relationship of the combined type (Compound) and is significant in terms of the probability value, which amounted to (0.000) and is less than (0.05). As shown in Table III and its dependent figures (3-8) respectively.

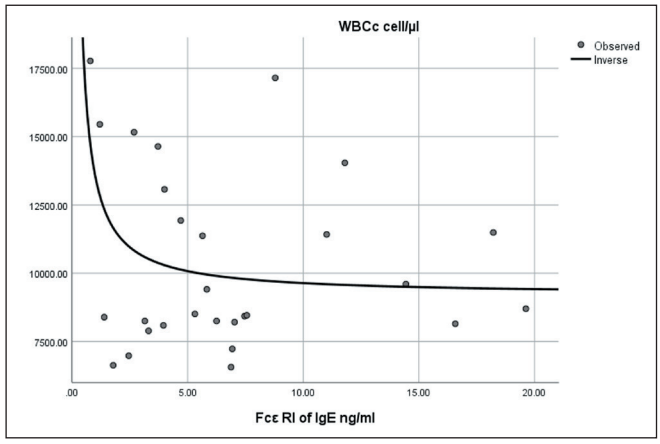

Figure 3: Linear regression coefficient between FCεRI and white blood cell count for category A.

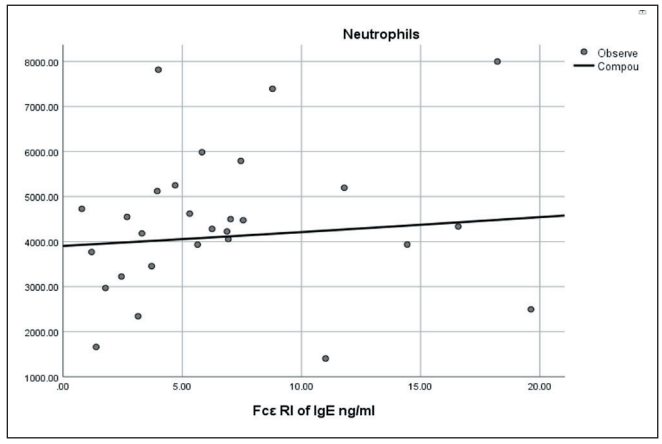

Figure 4: Linear regression coefficient between FCεRI and absolute numbers of neutrophils for category A.

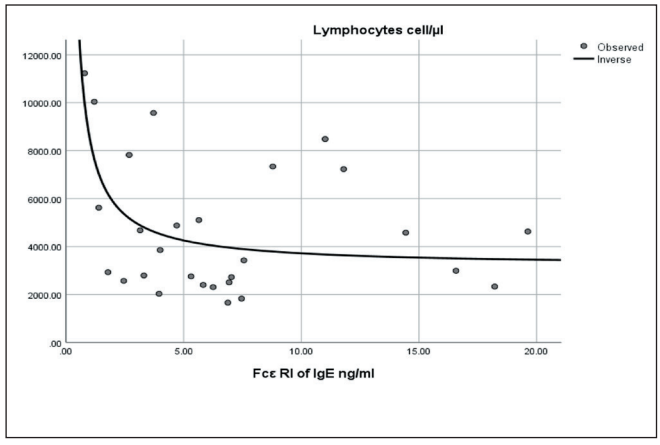

Figure 5: Linear regression coefficient between FCεRI and absolute numbers of lymphocytes for category A.

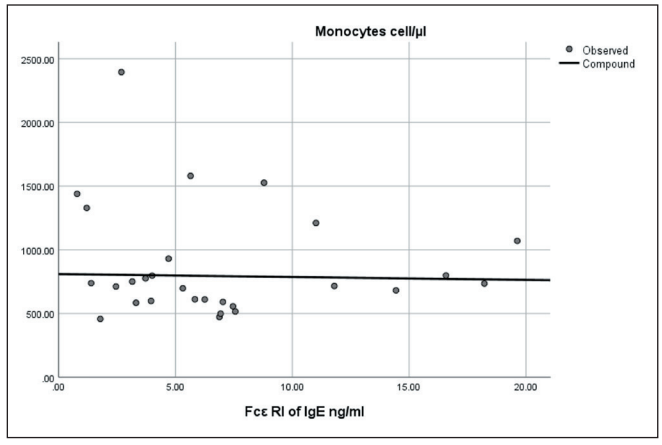

Figure 6: Linear regression coefficient between FCεRI and absolute numbers of monocytes for category A.

2. Sample patients for category B:

1. The regression coefficient of the independent variable (FcεRI of IgE ng/ml) in the dependent variable (WBCc cell/µL) is a nonlinear relationship of the Combined type (Compound) and is significant in terms of the probability value, which amounted to (0.000) and is less than (0.05).
2. The regression coefficient of the independent variable (FcεRI of IgE ng/ml) in the dependent variable (Neutrophils cell/µL) is a nonlinear relationship of the combined type (Compound) and is significant in terms of the probabilistic value, which amounted to (0.000) and is less than (0.05).
3. The regression coefficient of the independent variable (FcεRI of IgE ng/ml) in the dependent variable (Lymphocytes cell/µL) is a nonlinear relationship of the Combined type (Compound) and is significant in terms of the probabilistic value, which amounted to (0.000) and is less than (0.05).
4. The regression coefficient of the independent variable (FcεRI of IgE ng/ml) in the dependent variable (Monocytes cell/µL) is a nonlinear relationship of the combined type (Compound) and is significant in terms of the probabilistic value, which amounted to (0.000) and is less than (0.05).
5. The regression coefficient of the independent variable (FcεRI of IgE ng/ml) in the dependent variable (Eosinophils cell/µL) is a nonlinear relationship of the combined type (Compound) and is significant in terms of

the probability value, which amounted to (0.000) and is less than (0.05).

6. The regression coefficient of the independent variable (FcεRI of IgE ng/ml) in the dependent variable (Basophils cell/μL) is a nonlinear relationship of the combined type (Compound) and is significant in terms of the probability value, which amounted to (0.000) and is less than (0.05). As shown in Table IV and its dependent figures (9-14) respectively.

Table IV: The regression coefficient independent variable FcεRI of IgE in the group B of study variables WBCcount, Neutrophils, Lymphocytes, Monocytes, Eosinophils, and Basophils.

Independent variable	Dependent variable	Relationship	Equation	Bi		P- value (b_1)
				b_0	b_1	
FCεRI of IgE ng/ml	WBCs cell/μl	Compound	$Y = b_0 * (b_1 \wedge X)$	7557.99	1.00	0.000
	Neutrophils cell/μl	Compound	$Y = b_0 * (b_1 \wedge X)$	4479.37	0.976	0.000
	Lymphocytes cell/μl	Compound	$Y = b_0 * (b_1 \wedge X)$	2126.69	1.041	0.000
	Monocytes cell/μl	Compound	$Y = b_0 * (b_1 \wedge X)$	594.01	1.001	0.000
	Eosinophils cell/μl	Compound	$Y = b_0 * (b_1 \wedge X)$	201.42	0.972	0.000
	Basophils cell/μl	Compound	$Y = b_0 * (b_1 \wedge X)$	39.24	0.992	0.000

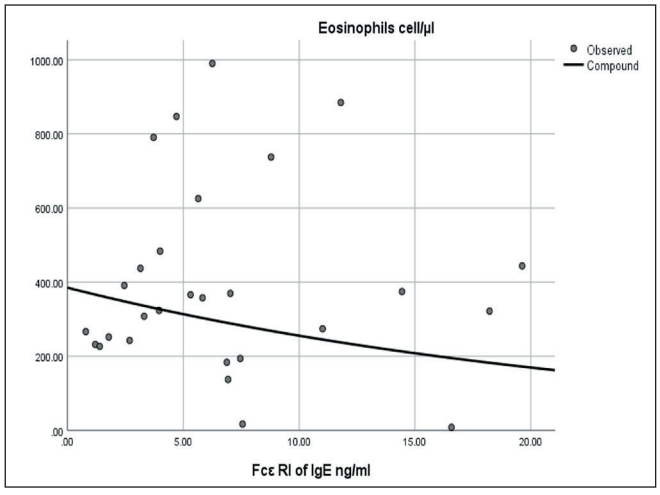

Figure 7: Linear regression coefficient between FCεRI and absolute numbers of eosinophils for category A.

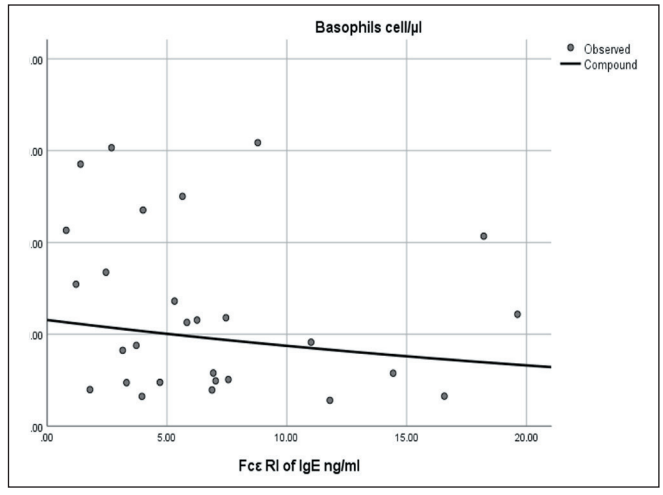

Figure 8: Linear regression coefficient between FCεRI with absolute numbers of basophils for category A.

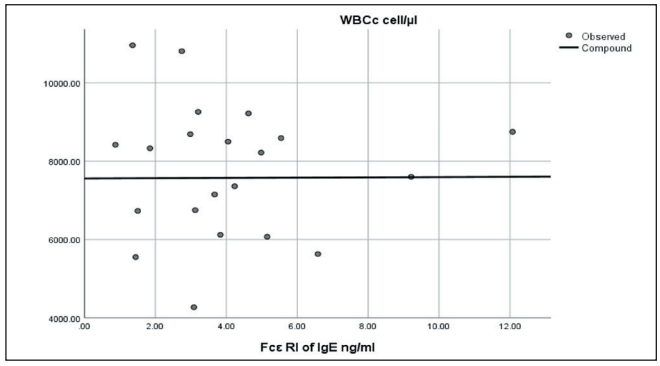

Figure 9: Linear regression coefficient between FCεRI and white blood cell count for category B.

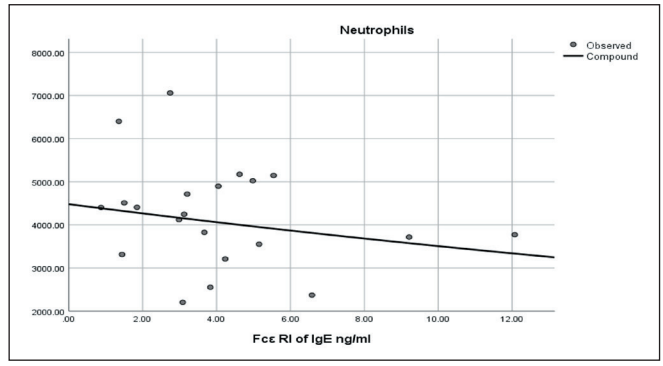

Figure10: Linear regression coefficient between FCεRI and absolute numbers of neutrophils for category B.

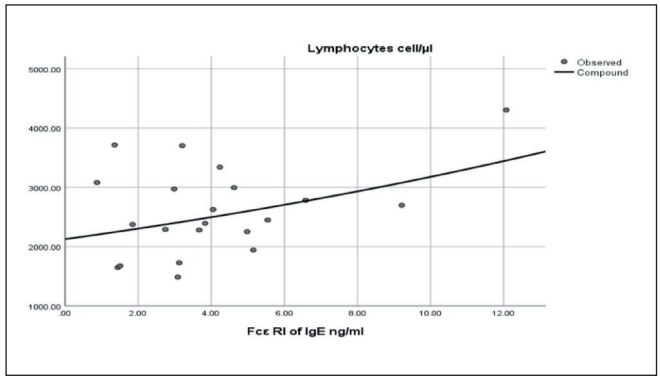

Figure 11: Linear regression coefficient between FCεRI and absolute numbers of lymphocytes for category B.

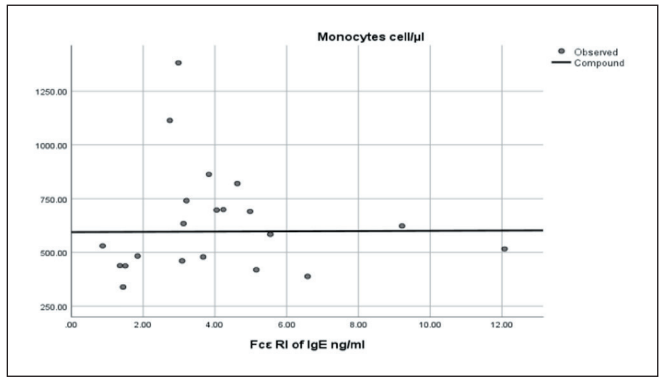

Figure 12: Linear regression coefficient between FCεRI and absolute numbers of monocytes for category B.

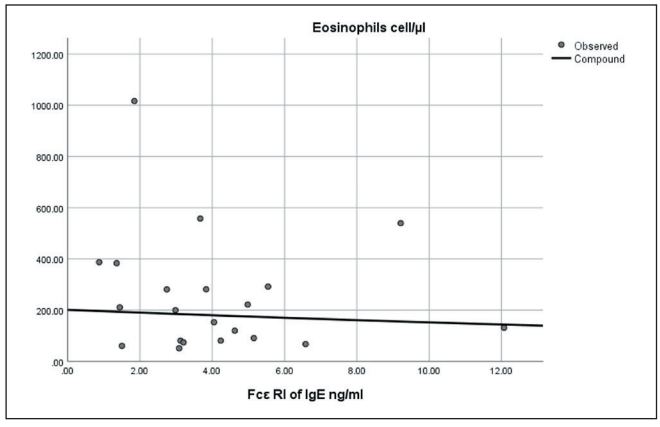

Figure 13: Linear regression coefficient Between FCεRI and absolute numbers of eosinophils for category B.

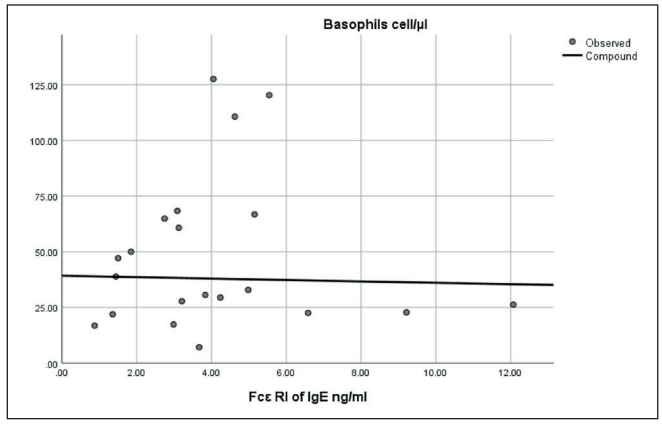

Figure 14: Linear regression coefficient between FCεRI with absolute numbers of basophils for category B.

DISCUSSION

The relationship between AD and patient age is complex, influenced by many factors including immune responses, genetic predispositions, environmental triggers and socioeconomic conditions. The results of the current study showed that most people with AD are neonates and infants between the ages of 1 and 3 years. They constitute the vast majority of the total cases, as AD appears mainly during early childhood, affecting about 20% of children, as shown in Figure 1. This finding is consistent with the study by (17). However, symptoms may persist in a large proportion of children into adulthood. It is estimated that AD persists in 40% to 60% of cases beyond childhood (18). In the second study group, comprising adults with AD aged 16–30 years, the prevalence is lower, estimated at 2%–7%. Nevertheless, it remains a major concern due to the possibility of recurrence when triggers are present, especially in individuals with a history of childhood AD, which aligns with the findings of the study (19). The current study found that adults with eczema show a tendency to develop other allergic diseases such as asthma and allergic rhinitis (6), which is described by the phenomenon known as the 'atopic trajectory' – the progression from childhood eczema to other allergic conditions (20).

The severity of injury and inflammation in the patients participating in the current study varied to varying degrees and showed an inverse relationship with age. The severity of the disease increased significantly in the first age group of children and gradually decreased with age until the second age group of adults, who exhibited a certain level of severity at a lower rate than in children. The severity of AD is often assessed using the SCORAD index, which is a measure of disease severity that categorises the condition into grades and assigns numerical values: 0 indicating no injury, 1 mild, 2 moderate and 3 severe injury. Children with early AD showed higher SCORAD scores, indicat-

ing more severe symptoms compared to those with later forms of the disease or older individuals (21), as shown in Figure 2.

The current study is consistent with another study suggesting that infants and young children tend to develop more severe forms of AD, often localised to the face and scalp, while adults may develop chronic dermatitis with ulcers and lesions commonly affecting the hands and feet (17).

High levels of circulating IgE antibodies appear in the serum of AD patients, binding to FcεRI on mast cells, basophils and dendritic cells. These ligands bind FcεRI on the cell surface, preventing its internalisation and increasing receptor expression (22,23). AD exhibits a Th2-type immune response, which promotes the production of cytokines such as IL-4 and IL-13. These, in turn, stimulate B cells to produce IgE and upregulate FcεRI expression on antigen-presenting cells (APCs) such as Langerhans cells (24). Persistent and chronic inflammation with skin lesions associated with AD may lead to localisation and activation of immune cells expressing FcεRI, increasing its concentration even in serum, especially in children under 10 years of age (25,26). This is consistent with the findings of the current study, which showed increased serum FcεRI levels in the 1–15 years age group.

Mutations in the function of the *Filaggrin* gene, a key skin barrier protein, are strongly associated with AD. This allows allergens to penetrate the skin, thereby leading to the production of IgE protein and up-regulation of FcεRI (27). Repeated and continuous exposure to environmental allergens (such as dust mites and pollen) leads to IgE sensitisation and FcεRI expression in individuals with AD (28). The data in one study (29), which is consistent with our findings, showed that the severity of the disease is directly proportional to the high levels of IgE and its FcεRI receptors on the surfaces of presenting cells in AD patients.

Foreign antigens penetrating the skin of AD patients and triggering allergies and itching lead to the production of FcεRI immune receptors by APCs in large and accumulated quantities, as a result of an error in the immune response, in an attempt to contain the immune complexes formed by the binding of foreign antigens with their antibodies. Consequently, these receptors fail to recognise the antigens correctly, resulting in excessive and inaccurate binding that intensifies itching and irritation. These findings were in agreement with those published in another study (30), conducted on patients with allergic diseases.

The increased concentration of FcεRI immune receptors in patients with chronic AD is related to the fact that these receptors are meant to be specific for IgE antibodies, owing to the high affinity and specificity between them. However, a similarly high affinity of these receptors for G-type autoantibodies has also been observed in AD patients. This leads FcεRI receptors to fail to distinguish between IgE and IgG antibodies, resulting in the formation of abnormal immune complexes that deceive the body's defences and ultimately contribute to immune failure or the development of autoimmune diseases, consistent with another study (31).

There is another study (32) that provides evidence similar to that obtained in the current study, showing that IgE can permeate damaged skin in AD patients and bind to its specific receptor FcεRI on the surface of mast cells, unlike in healthy individuals. This binding leads to the release of inflammatory mediators from these cells. The continued formation of such complexes contributes to the worsening of AD symptoms into a chronic condition. Although the proportion of FcεRI receptors in AD patients is abnormally elevated compared to healthy individuals, it is lower in this adult age group compared to children. It shows an inverse relationship with age, which is consistent with our study. The presence of free FcεRI antigens in serum reflects the complexity of the immune response at the skin surface. In addition to the immune response seen in type I allergy, a secondary autoimmune response involving the formation of IgG antibodies in parallel with the serum FcεRI receptor may occur, creating an inflammatory environment that may continue to manifest as chronic symptoms in AD patients, consistent with the research conducted elsewhere (33).

sFcεRI is a circulating form of the high-affinity IgE receptor, which is shed or secreted by cells such as mast cells, basophils and dendritic cells. It can bind to IgE in the serum, modulating IgE-mediated immune responses. In AD, sFcεRI levels are often elevated due to chronic inflammation and IgE dysregulation (34).

Neutrophils are not usually considered a major contributor to AD inflammation, but they may be recruited during secondary bacterial infections (e.g., *Staphylococcus aureus* colonisation). sFcεRI does not directly affect neutrophils but chronic inflammation in AD elevates neutrophil counts in severe or infected cases. In the current study, a significant correlation was observed between the

two variables due to the fact that the majority of patients exhibited chronic skin inflammation in AD, which in turn promotes secondary *S. aureus* infections (35). This demonstrates a direct correlation between secondary bacterial infection in AD patients and the neutrophil response in the bloodstream.

The relationship between sFcεRI and peripheral blood lymphocyte counts is clearly evident, as AD is associated with a dominant Th2-type immune response characterised by elevated IL-4, IL-5 and IL-13. sFcεRI may indirectly enhance Th2-type responses by increasing IgE production and B-cell activation (36). Monocytes and macrophages are indirectly influenced by sFcεRI but may be recruited to the inflamed skin in AD by cytokines (e.g., CCL2 and CCL17). These cells contribute to chronic inflammation, tissue remodelling, and the healing of surrounding tissues during inflammation in AD (37).

In addition to their role in the development of the inflammatory response, eosinophils in AD are characterised by eosinophilia (increased number of eosinophils), triggered by the activation of cytokines produced by Th2 cells (e.g. IL- 4, IL-5, IL-13). Furthermore, sFcεRI may promote eosinophil recruitment and activation by amplifying IgE-mediated responses and Th2 inflammation (38). Basophils are an essential component of IgE-mediated allergic responses.

Although basophils can express FcεRI, sFcεRI can modulate basophil activity by competing with membrane-bound FcεRI for IgE binding, which may reduce basophil lysis and the secretion of inflammatory factors. Chronic elevated IgE in AD may also lead to basophil activation and polarisation (39).

Anti-FcεRI antibodies may form insoluble immune complexes that accumulate in various tissues. The formation of these complexes may lead to the activation of the classical MIM system, which in turn activates complement or Fcγ receptors on immune cells, potentially exacerbating inflammation and complicating treatment. Other studies (40, 41) have demonstrated histamine release via an FcεRI antagonist in CSU.

The discrepancy in the type of relationships, despite their statistical significance as demonstrated by the regression coefficient test, between the two age groups in the current study may reflect the immune status of the first group, which includes individuals from the infant category. The immune system of this group is still developing, so it is natural for white blood cell counts to show higher levels compared to those in the second age group, whose immune system is mature and complete. Even when observing the clinical condition of the skin in the infant group, the ulcers may appear more advanced, penetrating the tissue and proving more difficult to heal compared to the second age group. This, in turn, is fully reflected in the immune response and the overall allergic state.

CONCLUSIONS

This study highlights the significant role of FcεRI in the inflammatory profile of AD. The findings indicate that FcεRI levels are markedly elevated in AD patients compared to healthy controls, with higher concentrations observed in children than in adults. This suggests that FcεRI plays a crucial role in disease severity and immune dysregulation. Regression analysis revealed a strong correlation between FcεRI levels and white blood cell subsets – particularly neutrophils, lymphocytes, and eosinophils – indicating its influence on immune cell activity in AD. The study further suggests that FcεRI may contribute to the development of autoimmune complications in AD patients by promoting chronic inflammation and immune dysregulation. These results support the potential of FcεRI as a biomarker for AD severity and disease progression. Its elevated levels in younger patients emphasise the need for early diagnostic markers and targeted interventions. Future research should investigate FcεRI as a therapeutic target to reduce inflammation and improve disease management. Overall, this study provides new insights into the immunopathology of AD, underscoring the importance of FcεRI in allergic inflammation and immune responses. A deeper understanding of its role may lead to improved diagnostic and treatment strategies, ultimately enhancing the quality of life for individuals with AD.

Conflict of Interest

The authors declare that there is no conflict of interest.

Author Contributions

Concept: **Rojan Ghanim Mohammed Al-Allaff,** Design: **Bandar Ibrahim Aayf Al-Shammari, Rojan Ghanim Mohammed Al-Allaff,** Data collection or processing: **Bandar Ibrahim Aayf Al-Shammari,** Analysis or Interpretation: **Bandar Ibrahim Aayf Al-Shammari, Rojan Ghanim Mohammed Al-Allaff,** Literature search: **Bandar Ibrahim Aayf Al-Shammari,** Writing: **Bandar Ibrahim Aayf Al-Shammari, Rojan Ghanim Mohammed Al-Allaff,** Approval: **Bandar Ibrahim Aayf Al-Shammari.**

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