

**FACULTY OF MEDICINE - BENGHAZI UNIVERSITY**

**DERMATOLOGY DEPARTEMENT**

**كلية الطب - جامعة بنغازي**

**قسم الأمراض الجلدية والتناسلية**

Quality of life in infants and children with atopic dermatitis in Benghazi

نوعية الحياة في الرضع و الأطفال المصابين بالاكزيما التآيبيية في بنغازي

by: Orieda Awad Gebreel EL.Awamei

Supervised by: Ibrahim Abdulgader Al-mukahal

Associate Professor of dermatology

This thesis is to be submitted to the dermatology department –faculty of medicine – Benghazi university as a partial fulfillment to the Master degree (MSC) in specialty of Dermatology and Venereology

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Dr. Ibrahim Almukahal

Many thanks to my husband who supported me all the time.

## **ABBREVIATION**

Atopic dermatitis (AD)

Infant dermatology quality of life(IDQOL)

Children dermatology quality of life(CDQOL)

Eczema area severity index (EASI)

Immunoglobulin E (IgE)

T Lymphocyte cell (Tcell)

Cutanouse Lymphocyte antigen (CLA)

T helper 1 lymphocyte (Th1)

Thelper2 lymphocyte (Th2)

Inter leukin 4(IL4)

Interleukin 13(IL13)

Intercelluler adhesion molecule (ICAM)

Staphlyococcus-aurous (S-aurous)

Radioallergosorbent tests (RasTs)

Human immunodeficiency virus (HIV)

Topical calcineurein inhibitor (TCI)

Human dust mite (HDM)

Food and drug administration (FDA)

Ulter violet B (UVB)

Psoralin –ulterviolet A (PUVA)

## **Certification**

This thesis entitled "Quality of life in infants and children with atopic dermatitis in Benghazi " , prepared by Dr. "OridaAwadgebreel " , under supervision of Dr. "IbrahimAlmukahal, MD" , has been approved for submission to the faculty of Medicine , Benghazi University of Medical Science , Benghazi – Libya in partial fulfillment for certification of degree of Master in Dermatology and Veneology (MSc)

### Supervisor

Assistant Professor Dr. Ibrahim Al mukahal , MD

Dermatology department

Jamhoriya Hospital

Benghazi – Libya

## **Declaration**

This is to declare that I have not submitted the research work embodied in this thesis " Quality of life in infants and children with atopic dermatitis in Benghazi " to any other university before.

### **Candidate**

Dr.OridaAwadGebreel

Dermatology department

Jamhoriya hospital

Benghazi - Libya

January-2014

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# **SUMMARY**

## 1-SUMMARY

**Introduction:**skin disease can cause severe disability and handicap in children.

Atopic dermatitis (AD) has a major impact on patients quality of life (QOL), and it can affect the psychological health of families of patients with AD. However, to the best of our knowledge, this is the first study to assess the quality of life in Libyan children .

**Aim of the study:**the purpose Of this study was to determine the Qol of infant and children with AD and its correlation with disease severity.

**Material and methods:** a study was conducted on a total of 87 children aged between 6 months and 1years, attending out-patients clinic at dermatology department,Jomohoria hospital,BenghaziLibyaPeriod extending from November 2010 to 30 July 2013.detailed history was taken . children with help of their parents were asked to complete the translated Arabic version of infants dermatitis quality of life (IDQOL) and children ,s dermatology life quality index (CDLQI). The severity of eczema was assessed using the eczema area and severity index (EASI).

**Results:**Among the total patients, 41.4% of patients were male and 58.6% were females .The mean score of IDQOL was 17.6%and mean score of CDLQI was 17.8%. whereasthe range score of IDQOL was 8-26, and the range score of CDLQI was 6-28. 46% of patients were found to have a history of other allergic diseases , and family history of atopy was reported in 79.3%of the cases. IDQOLHigh scorewas for itching, scratching and mood disturbance (question 1 and 2)which the mean score was 2.45 and 2.28 respectively.

The high score of CDLQI was for itching ,scratching, mood and other people (behavior (question 1,2 and 8)in which mean score was 2.4, 2.3 and 2.2 respectively.

The EASI score revealed that majority of patients (74.7%)have moderate severity 18.4%have mild severity and 6% have severe severity .there was a correlation between the CDLQI and EASI ( $r=0.14$ ).

**Conclusion** : Atopic dermatitis has a profound effect on many aspects of a patient's life . quality of life issue is now recognized as an important outcome in medical care in Libya : therefore by interpreting these results

We can aim to improve the QOL of AD patient'sby targeting the prominent issues of itching , scratching and mood disturbance .

Education and psychological support for patient and their family need more than physical treatment of symptom to improve . their long term Physical outcome

# **INTRODUCTION**

## 2-Introduction

Atopic dermatitis (AD) - also known as atopic eczema - is a chronically relapsing, highly pruritic, inflammatory skin disease that affects 2-5% of the general population. AD has the largest impact on infants and children, affecting an estimated 10-20% or more, but is also believed to affect 1-3% of adults (1) Its precise definition, in relationship to other immunologic terms such as “allergy” and “hypersensitivity”, has been the source of and remains unresolved. The ambiguity in its current definition has contributed to difficulty in reaching a consensus in the diagnosis of AD (2-4)

Atopic dermatitis (AD) is a chronic and recurrent dermatosis characterized mainly by episodic pruritis and skin dryness. Its prolonged evolution, intensity of symptoms and discomfort may cause serious implications on the patients and their families' life.<sup>(5-7)</sup>

Atopic dermatitis most often begins in infancy or early childhood, with about 90% of cases appearing in the first five years of life, which is a critical period for psychosocial development. Both physiological and psychological abnormalities have been documented in young children with atopic dermatitis as well as in their families. Psychosocial abnormalities in infants, preschool children as well as school children with atopic dermatitis include behavioral problems, excessive dependency, clinginess, and fearfulness. In addition to daytime scratching, sleep in children with atopic dermatitis is marked by overall reduced sleep efficiency with frequent awakenings,<sup>(8)</sup> which for many children persist even during disease remission<sup>(9)</sup> Other reported sleep-related abnormalities include difficulty falling asleep, diminished total sleep greater difficulty awakening, daytime tiredness, and irritability. Difficulty falling asleep and night awakenings in children with atopic dermatitis correlate with behavior and discipline problems.<sup>(10)</sup>

Itching and scratching as well as difficulties with falling asleep and problems with bathing, show very great impact on the quality of life in affected children.<sup>(11)</sup>

Atopic dermatitis is one of the commonest skin diseases in children that has been proved to cause psychological, social and functional disability to them and their families and these effects are shown to be positively correlated with severity of illness.<sup>(12-13)</sup>

In general, chronic diseases are the primary focus of quality of life assessments, since they have a major impact on patients' lives.<sup>(14-15)</sup> Prominent among these are dermatological diseases, since they have a profound effect on people's lives, whether due to the itching, the anti-esthetic appearance or to restrictions caused by lesions or by treatment. It has been proven that they have a significant impact on social relations, on psychological status and the day-to-day activities of patients.<sup>(16)</sup> As a result, many different instruments for measuring quality of life have been developed specifically for patients with dermatological diseases, aimed at both the adult and pediatric populations.<sup>(17-18)</sup>

Increasingly, a demonstration of improvement in the quality of life of patients forms part of the assessment of new therapies.<sup>(19)</sup>

# **REVIEW OF LITERATURE**



### 3-REVIEW OF LITERATURE

Atopic dermatitis is a difficult condition to define, because it lacks a diagnostic test and shows variable clinical features. The following definition seems to be in accord with most consensus groups. Atopic dermatitis (which is synonymous with atopic eczema) is an itchy, chronic or chronically relapsing, inflammatory skin condition. The rash is characterized by itchy papules (occasionally vesicles in infants), which distribution. The eruption is frequently associated with other atopic conditions in the individual or other family members(20-22)

Atopic dermatitis (AD) - also known as atopic eczema- is a chronically relapsing, highly pruritic, inflammatory skin disease that affects 2-5% of the general population. AD has the largest impact on infants and children, affecting an estimated 10-20% or more, but is also believed to affect 1-3% of adults (1)

Its precise definition, in relationship to other immunologic terms such as “allergy” and “hypersensitivity”, has been the source of and remains unresolved. The ambiguity in its current definition has contributed to difficulty in reaching a consensus in the diagnosis of AD (23-25)

#### 3.1 Epidemiology

The prevalence of AD has increased over the past 30 years. It is currently estimated that 10-20% of children and 1-3% of adults in developed countries are affected by the disorder (26). AD often starts in early infancy; approximately 45% of all cases begin within the first 6 months of life, 60% during the first year, and 85% before 5 years of age. Up to 70% of these children outgrow the disorder before adolescence (27).

Atopic dermatitis is more common in females, with a 1.5:1 female to male ratio(28)..

, in the literatures 45.5% were males and 54.5% were females.(29)But in other study the males were more than females, 55% male and 45%female.(30)

AD may affect all races and ethnicities, as the studies performed donot reveal significant differences between the various ethnic or racial groups(31).

In about 70–80% of patients, AD is associated with increased total IgE serum levels. Atopic diseases have different peaks of incidence at different ages during childhood. AD and food allergies have the highest incidence in the first 2 years of life. Sensitization to inhalant allergens is rare at that time of life. In later childhood, the prevalence of AD, food allergies, and food allergen sensitization decreases and the prevalence of asthma, allergic rhinitis, and sensitization to inhalant allergens rises (32).

### **3.2AETIOPATHOGENESIS**

#### **3.2.1Non-immunologic factors**

*Genetics.* The genetic susceptibility to suffer respiratory atopy has been linked to chromosome 11 (11q13)(33).the authors concluded that the risk of evidencing the disease was 57% when the mother was affected and 46% when the father was the affected parent; attempts have been made to correlate these findings with modifications of the intra-utero immune response or with breastfeedi.(34)

#### **3.2.2Epidermal Barrier Dysfunction**

More recently, a theory involving the role of Epidermal Barrier Dysfunction has been proposed as an explanation on the physiopathology of atopic dermatitis. Changes in at least 3 groups of genes encoding structural proteins, epidermal proteases and protease inhibitors predispose to a defective epidermal barrier and increase the risk of developing atopic dermatitis. The strong association between both genetic barrier

defects and environmental insults to the barrier with atopic dermatitis suggests that epidermal barrier dysfunction is a primary event in the development of this disease.(35)

### **3.2.3 Immunopathogenesis**

In both acute and chronic lesions, a prominent lymphocytic infiltrate is seen. Immunohistochemical staining has demonstrated that the infiltrate is comprised predominantly of activated T cells bearing CD3, CD4 and CD45 RO antigens(36,37). Essentially all T lymphocytes migrating into the skin bear the 'cutaneous lymphocyte antigen' (CLA), which functions as a skin-homing receptor for T cells(38). Vascular endothelial cells in atopic dermatitis lesions express elevated levels of the receptor, VCAM-1(39), which plays an important role in targeting the homing of CLA+ T cells to sites of skin inflammation(40). VCAM-1, which is induced by interleukin (IL)-4 and IL-13(41), also plays an important role in the migration of eosinophils and mononuclear cells into sites of allergic inflammation. Increased numbers of Langerhans cells are found in chronic atopic dermatitis lesions and have been shown to have high-affinity [FcεRI] and low-affinity [FcεRII] receptors and surface bound IgE molecules(42). Mast cell numbers are also increased in chronic atopic dermatitis lesions. Typically mast cells bearing both tryptase and chymase (MCTC) are predominant in normal skin, but in eczematous skin increased numbers of mast cells bearing just tryptase (MCT-generally seen in the lung and gastrointestinal tract) are present in large numbers(43). The development of MCT cells is dependent upon functional T cells, suggesting that infiltrating T cells are responsible for their presence in the eczematous lesions. Keratinocytes also show evidence of cytokine-induced activation. ICAM-1 is expressed on keratinocytes from atopic dermatitis lesions(44),

Atopic dermatitis has been classically associated to IgE hyperproduction; the latter is, as already stated, due to an imbalance between IL-4 and IFN- $\gamma$  production with increased levels of the former, as it has been shown that IL-4 is a potent stimulus of IgE production via the B-cells. The IL-4 levels are increased in patients with severe AD, and thus an increase in the IL-4 levels also correlates with the severity of the condition<sup>(45)</sup>

### **3.2.4 Food allergy**

has its greatest incidence in infancy and early childhood.<sup>(46)</sup> It is estimated that about 8% of children will develop adverse reactions to food, most of them in the first year of life.<sup>(47)</sup> The prevalence of food allergy varies between regions and appears to be influenced by cultural and genetic factors.<sup>(48)</sup>

### **3.2.5 Airborne allergens.**

The fact that AD shows seasonal variations and that the patients improve when transferred from a given environment to a different one have led to a long-standing suspicion of a role of environmental factors in the development of the condition. The association of AD to atopic airway disease has been a reason for considering that airborne allergens (pollens, house dust, mites, animal danders and hairs, ...) in the context of the aetiopathogenesis of AD. Many studies have addressed this subject<sup>(49\_51)</sup>

**3.2.6 Infectious agents.**— The altered skin barrier of atopic patients provides a portal of entry for various pathogens, which are weakly deterred by their aberrant immune systems.<sup>(52)</sup>

1. Bacteria. Increased staphylococcal skin colonization of affected and normal skin has been noted in patients with AD compared with controls.<sup>(53)</sup> *S. aureus* colonization can be demonstrated in over 90% of lesions with AD. The density of *S. aureus* on inflamed AD lesions without clinical superinfection can reach up to 10(11) colony-forming units per cm on lesional skin. Although the majority of patients with AD are colonized by *S. aureus*, its presence does not necessarily indicate that it acts as a pathogen; antibiotic treatment is indicated only when there is evidence of overt clinical infection or a superantigen effect is suspected.<sup>(54)</sup>

2. Other bacteria, such as streptococcal species, may be important, but little clinical or investigative information exists to document their role.

3. Yeast. *Candida albicans* is the most common yeast, found in the mouth of 20%–25% of healthy adults, and regarded an opportunistic pathogen, or secondary invader of impaired skin, but there are no reports of increased or more severe *C. albicans* infections in patients with AD. However, positive skin prick tests with *C. albicans* occur more frequently in patients with AD than in normals. Yet it is unclear whether this response contributes to inflammation in AD.<sup>(55)</sup>

4. Dermatophytes. Dermatophytes (e.g., Trichophyton) are suspected of occurring more frequently in atopic patients, a fact ascribed to their relative Th1 cell defect. However, there is only a single report suggesting that colonization can act as a trigger factor for AD.<sup>(56)</sup>

5. Viruses. Patients with AD do not have a major deficiency in defending against viruses. However, some viral skin infections can have a dramatic course. Kaposi's

herpetiform and varicelliform eruptions, caused by spread of herpes and varicella viruses, are recognized as potentially dangerous complications of AD.<sup>(57)</sup>

### **3.3 Clinical features**

The distribution of skin lesions can be highly variable but is generally age-related<sup>(59)</sup>.

#### **3.3-1 Infantile Phase (0–2 Years)**

The earliest clinical features are dryness and roughness of the skin. Distinct eczematous lesions usually do not appear before 2 months of age. In infants, the dermatitis commonly affects face and scalp and spreads to involve the neck and trunk. Typically, lesions are erythematous and have highly pruritic, moist, oozing papulovesicles that may crust and scale. Secondary impetiginization may occur. The nasolabial and napkin areas are often spared. In children 1–2 years of age, the distribution of lesions moves from the face to the antecubital and popliteal fossae, neck, wrists, ankles, and retroauricular folds. Due to the developing ability to scratch, the primary lesions are altered and a more variable clinical picture develops with papules, poorly demarcated scaly patches, excoriations and haemorrhagic crusts. While the truncal lesions are often diffuse, on the extremities localized patches prevail that tend to involve both extensor and flexor aspects and commonly the wrists and ankles <sup>(60,61)</sup>.

#### **3.3-2 Childhood Phase (2–12 Years)**

During childhood, polymorphous manifestations with different types of skin lesions at different locations are common. At sites of chronic involvement, thickened plaques with excoriation and mild lichenification develop. During phases of exacerbation, acute erythema, plaque-like infiltrations and weeping or erosive skin lesions may

occur. Other morphological variants of the childhood phase are nummular, papulovesicular or lichenoid lesions. Flexures and buttocks become the predominant predilection sites. The nails may become shiny and buffed from constant rubbing and long-lasting eczema of the periungual skin (“eczema nails”) (58,60,61).

### **3.3.3 Adolescent Phase (12–18 Years) and Adulthood**

The main clinical picture of atopic eczema in adolescents are flexural lichenified and often excoriated skin lesions (62). In addition, wrists, ankles and eyelids are frequently affected (63). In more widespread disease, the upper trunk, shoulders and scalp may be affected.

Atopic eczema spontaneously clears in about 40% of children before or during adolescence but may remain quiescent in others until adulthood, when it most commonly shows facial and extensor involvement, lichenifications in the flexural areas, and involvement of wrists, hands, ankles, feet, fingers and toes. It may also reappear as hand eczema. A small subgroup of patients exhibits the first symptoms not before adulthood (58).

## **3.4 Associated disorders Other manifestations of atopy**

### **3.4.1 Dry skin**

This is a common feature of atopic dermatitis and figures prominently in its management. It is likely that it occurs because of increased transepidermal water loss through an abnormal stratum corneum (64,65).

**3.4.2 Dennie-Morgan skin fold.** This is simply the result of the presence of oedema in the lower eyelid that reveals the existence of a second or third palpebral crease. The

Dennie-Morgan fold was long held to be a diagnostic sign for the disease, but a number of studies have shown that it may be observed in 49% of the normal population, even though it is certainly more frequent among atopics (75%)(66).

### **3.4.3 Pityriasis Alba**

In areas of previous eczema, especially in the face, neck, and upper trunk, finely scaling and diffusely demarcated hypopigmented patches sometimes resembling tinea corporis or vitiligo may develop. The condition is most prominent after prolonged sun exposure and represents postinflammatory hypopigmentation. Pityriasis alba has been reported to occur in 35%–44% of eczema patients (67,68).

### **3.4.4 Hyperlinearity of the Palms/Soles**

Hyperlinearity of the palms or the soles is noted more often in atopic patients than in nonatopic patients and has been found in up to 88% of atopic eczema patients. It is regarded as increased expression of palmar and/or plantar creases and lines (69)

### **3.4.5 White Dermographism**

In nonatopic individuals, firm stroking of the skin causes a red line with a reflex erythema. In contrast, the majority of eczema patients shows a delayed white line, which replaces the initial erythematous reaction after 1 min (70).

### **3.4.6 (Peri-)Orbital Darkening**

Many eczema patients exhibit a blue-grey hue around the eyes with accentuation of the suborbital area. This condition is more frequent in the young (66).

### **3.4.7 Keratosis Pilaris**

Keratosis pilaris is a disorder of keratinization of the (xerotic) hair follicles characterized by tiny rough bumps on the skin (like “chicken skin”). Primarily, it



appears on the back and outer sides of the upper arms, but can also occur on thighs and buttocks or any body part except palms or soles. It is frequently associated with atopic eczema but can also be seen in other inflammatory dermatoses or occur in individuals without other skin lesions. It most often appears in childhood, reaches its peak incidence in adolescence, and becomes less apparent during adulthood (71).

#### **3.4.8 Palmar and plantar dermatitis.**

hand eczema has been noted to occur in 70% of children with AD.(72) Atopy has been reported to occur in up to 57% of patients with juvenile palmar-plantar dermatosis.(73)

#### **3.4.9 Nipple dermatitis.—**

Nipple dermatitis is noted in 12%–23% of patients with AD.(74)

### **3.5 Impact on quality of life**

Psychological characteristics of the atopic patient

It is difficult to define any change that is typical for AD patients, but an attempt can be made, in my opinion, to define a psychologic profile. It is impossible to establish whether the psychologic changes influence the disease or, quite to the opposite, whether it is the disease that conditions certain behavioural patterns in the atopic patients. Regarding the first possibility, the influence (positive or negative) of hospital admission on the evolutionary course of AD is well demonstrated(75), as well as the fact that patients who have improved again deteriorate when they are discharged and return to their home environment, in correlation to a number of social conditionants. Further factors such as stress, marriage, military service or pregnancy, among many others, may negatively influence the course of the disease. Restlessness, aggressiveness, anxiety, irritability and other manifestations are frequent findings

among atopics, and it is not uncommon for these patients to use these features to achieve what they want(76).

, and others found that 38% of siblings of children with AD also had disturbed sleep(77).

. Sleep loss leads to physical and mental exhaustion for part or all of the family causing mood disturbance, loss of concentration and impaired performance at school or work(78)

The atopic child is restless, with disordered sleep(79), mischievous, lively, "never stopping", and drives his/her parents to desperation. At school he is usually the leader and the first one in his class during the first few years; afterwards, his lack of concentration relegates him/her to the last places. Events in his immediate environment (new brethren, divorce of the parents, new environments) have a usually negative influence on the evolution of the disease. The atopic child's parents also suffer the consequences of the disease, as they have emotional difficulties with their offspring. Parents or other relatives that live with an atopic child are less spontaneous and evidence increased irritability(80).

### **3.6 DIAGNOSIS**

As no biochemical criteria exist that may firmly establish the certainty diagnosis of atopic dermatitis, clinical criteria must be reverted to. The universally accepted criteria are those postulated in 1983 by Hanifin and Rajka(81), which are presented in Table II. These criteria have been examined by a number of investigators, and their reliability has been fully validated(74).

#### **Diagnostic criteria for Atopic Dermatitis**

### **3.6.1 Major criteria**

- Pruritus
- Characteristic morphology and distribution
- Flexure lichenification in adults
- Involvement of face, flexures and extensor surfaces in children and adolescents
- Combination of the two patterns in children and adults
- Chronic and recurrent character
- Personal or familial history of atopy

### **3.6. 2 Miner criteria**

- Xerosis
- Ichthyosis / exaggerated palmar creases / keratosis pilaris
- Immediate (Type I) skin reactivity on skin testing
- Increased serum IgE levels
- Early onset age
- Tendency to skin infections and cell-mediated immunity deficiency
- Tendency to non-specific hand and foot dermatitides
- Nipple eczema
- Cheilitis
- Recurrent conjunctivitis
- Infraorbital (Dennie-Morgan) skin fold
- Keratoconus
- Anterior subcapsular cataract
- Eye rings ("shiners"), periocular darkening of the skin

- Facial pallor or erythema
- Pityriasis alba
- Skin folds on the anterior aspect of the throat
- Pruritus induced by sweating
- Intolerance to wool and to fat solvents
- Perifollicular enhancement
- Intolerance to some foods
- Course influenced by environmental and emotional factors
- White dermographism

Three or more major and three or more minor criteria must be met for diagnosis

### **3.7 Investigation**

The diagnosis of atopic dermatitis is rarely aided by investigations. Estimation of total serum IgE, specific radioallergosorbent tests (RASTs) and prick tests usually serve only to confirm the atopic nature of the individual. It is possible that such confirmation may be of value occasionally in adult-onset dermatitis. However, one must remember that 20% of patients with atopic dermatitis have normal total IgE levels and negative RASTs, whereas 15% of apparently healthy individuals have a raised IgE<sup>(82)</sup>.

The value of investigations in identifying trigger factors in atopic dermatitis is disputed. How helpful specific RASTs to foods or aeroallergens are for disease management is unclear (83–85), although it is suggested that if they are negative, allergy is unlikely (49). It may be that skin prick test positivity to food allergens in young children with severe atopic dermatitis and a high serum IgE indicates a high risk of developing later allergic respiratory disease (86).

If one suspects immunodeficiency with atopic dermatitis, then the appropriate investigations should be performed. For example, immunoglobulin levels and subclasses, IgE levels, white-cell count, platelets, complement levels and function, and T, B and phagocyte cell numbers and functions. If clinically appropriate, one may also consider testing for HTLV-I and human immunodeficiency virus (HIV).

Bacteriology and virology swabs may be helpful in identifying causes for deterioration of atopic dermatitis. Although atopic dermatitis skin is often colonized by *Staphylococcus aureus*, bacterial culture can identify antibiotic resistance and detect  $\alpha$ -haemolytic streptococci. Herpes simplex is usually readily cultured, but a Tzanck smear, an immunofluorescence slide test, or electron microscopy can also be helpful, and will provide more rapid confirmation of infection.

Patch testing may also help to identify a contact allergen responsible for deterioration of the skin condition, particularly in adults (87,88). The 'atopy patch test' for aeroallergens (85) and foods (89).

**Pathology** The histological changes are not specific, and are those of a subacute or chronic eczema. In infancy, early lesions show acanthosis and sometimes spongiosis, oedema of the dermis and infiltration with lymphocytes, histiocytes, plasma cells and eosinophils, sometimes in large numbers.

In later age groups, the histology is that of lichenification, sometimes with more eosinophils in the dermis than are found in lichen simplex, and with an increase in the number of Langerhans' cells. Eosinophilia in tissues as well as in peripheral blood is found in many cases of atopic dermatitis, but is of no help in determining the role of allergy in producing the symptoms (90,91).

### **3.8 Complications**

#### **3.8.1 Impact on quality of life**

Psychosocial aspects

Atopic dermatitis has a profound effect on many aspects of patients' lives and the lives of their families<sup>(92-95)</sup>.

In children, one of the most disturbing impacts of the disease is on the sleep pattern. This can lead to behavioural difficulties in the most severely affected children <sup>(10,96)</sup>.

The patient's dermatitis can also interfere with the functioning of the family <sup>(97)</sup>.

Increasingly, a demonstration of improvement in the quality of life of patients forms part of the assessment of new therapies <sup>(19)</sup>.

In other study the mean score of CDLQI was  $12.83 \pm 6.52$ .<sup>(98)</sup>

and the mean score for IDQoL3 was 4.52 (SD 3.67, range 0–20).<sup>(99)</sup>

In other study found that atopic dermatitis can cause psychological difficulties for the child, parent(s) or other family members and may affect their interpersonal relationships.<sup>(100-104)</sup> Psychological disturbance may also promote disease flares or affect clinical management. Others found double the rate of psychological disturbance in school-aged children.<sup>(105)</sup>

#### **3.8.2 Growth delay**

Growth delay can be associated with atopic dermatitis <sup>(106)</sup>. It used to be seen in severe cases before the advent of corticosteroid therapy, and can therefore be attributed to the disease. However, suspicion must also clearly rest on both oral and topical steroid therapy, which can cause growth stunting in any child on long-term therapy <sup>(107)</sup>.

### **3.8.3 Bacterial infections**

Secondary bacterial infection with staphylococci or streptococci is virtually an integral part of the clinical picture (108).

### **3.8.4 Viral infections**

Patients with atopic dermatitis, both active and quiescent, are liable to develop acute generalized infections with herpes simplex (eczema herpeticum) and vaccinia (eczema vaccinatum) viruses, to produce the clinical picture of Kaposi's varicelliform eruption (109).

### **3.8.5 Ocular abnormalities**

A number of ocular changes can occur in atopic dermatitis (110). The Dennie–Morgan fold is often present as a fold of skin under the lower eyelids (111). However, this change is not specific to atopic dermatitis, and is commonly seen in non-atopic black children (112).

Conjunctival irritation is a common syndrome in atopic persons. As in hay fever, it may represent a true allergic reaction, or it may be due to a non-allergic irritability such as occurs in the nose or skin. Keratoconjunctivitis has been recorded (113). Keratoconus (114,115), or conical cornea, is a rare condition. It may occur in the absence of any other disease or in association with atopic dermatitis. It is due to a degenerative change in the cornea, which is forced outwards by the intraocular pressure, to give rise to marked visual disturbances. Onset is in childhood, and after some years progress of the disease becomes arrested. Contact lenses may be helpful.

Cataract associated with atopic dermatitis has certain peculiarities which distinguish it from other types of cataract (114)

### **3.9 Differential diagnosis**

In the individual patient, one must consider a number of other conditions. Scabies should always be excluded, and can cause confusion when superimposed on pre-existing atopic dermatitis. In the first few months of life, the differentiation of infantile seborrhoeic dermatitis from atopic dermatitis can be difficult (116).

### **3.10 Prognosis**

The prognosis for patients with AD is generally favourable, with most children outgrowing the condition by early adolescence. However, patients with severe, widespread disease and concomitant atopic conditions, such as asthma and allergic rhinitis, are likely to experience poorer outcomes (117).

### **3.11 Treatment**

The treatment of AD should be directed at limiting itching, repairing the skin and decreasing inflammation when necessary. Therefore, the successful management of AD requires a multifaceted approach that involves patient and caregiver education, optimal skin care practices, anti-inflammatory treatment with topical corticosteroids (first-line) and/or topical calcineurin inhibitors (TCIs), the use of first-generation antihistamines to help manage sleep disturbances, and the treatment of skin infections. Systemic corticosteroids may also be considered in severe cases that cannot be controlled with appropriate skin care and topical therapy (118,119,120,121,122).



### 3.11.1 General hygienic measures

*Bathing.* The question whether the AD patient may bathe, and how, is obligate. According to Hanifin's recommendations<sup>(123)</sup>, and depending on the severity of the disease, bathing should be preferred, in the severe and pruriginous forms, to showering, the latter being reserved for those cases in which the skin has improved as to erythema, pruritus and desquamation. The patient should remain immersed in the bath for 20 minutes (until the fingertips become creased and prune-like); adding to the water emollient substances that mitigate pruritus and soften the skin is quite adequate. The most widely used emollients are oat-derived colloids, but some tar preparations (at present highly controversial) or mineral or vegetable oils may also be useful. The soap used should maintain an acidic pH and be devoid of irritant and sensitising substances; for these reasons, the so-called "syndets" (soapless soaps) are the most adequate ones. Soaps that have an antiseptic action help in preventing *Staphylococcus aureus* overpopulation, so that chlorhexidine soaps or preparations may be useful. The sponge or terry cloth used should be soft and cause no irritation of the skin.

After bathing, the skin should be dried carefully using always soft towels. Rubbing or hot air dryers should not be used, as they will further dehydrate the skin. An emollient cream should be applied after bathing, not immediately but some minutes later. A large variety of such

products are commercially available; the most useful ones are perhaps those containing □ fatty acids (primrose oil, linoleic and/or linolenic acid) or ceramides.

*General measures.* Clothing should be recommended that does not increase the itching sensation; the garments used should therefore be light and not tight fitting, and whenever possible should contain neither wool nor synthetic textile fibres; cotton and linen should be recommended.

Care should be taken to remove the labels stitched to the clothing, which have a strongly irritant action on the atopic skin.

Atopic patients do not tolerate heat, as they sweat abnormally and the itching sensation increases. They should therefore be advised to be moderate regarding physical exercise and exertion and to avoid warm and excessively dry environments. The adequate environmental conditions for these patients are 18°C temperature and 50% relative humidity<sup>(124)</sup>.

### **3.11.2 Reduction of trigger factors**

Atopic dermatitis can be aggravated by a variety of trigger factors. These will vary between patients, and may differ at various times in an individual patient. Most patients have dry skin, and soaps and detergents can irritate the dermatitis <sup>(125)</sup>.

A dispersible cream can be used as a soap substitute to cleanse the skin. Simple measures such as turning down the central heating, not heating the bedroom, avoiding contact of wool with the skin and wearing cotton clothing, may make life more comfortable for the patient. Formal dietary manipulation is really a second-line treatment, but if the patient clearly identifies aggravating foods then avoidance can be tried. Many patients have already started a restricted diet before seeing a doctor, so dietary assessment is important to confirm adequate nutrition. Airborne allergens are also aggravating factors, but formal manipulation of the environment is not required for most patients. Regular cleaning of the bedroom in particular, with Hoovering and damp dusting, may be helpful. Animal dander can aggravate atopic dermatitis and contribute to HDM antigen levels, and so the keeping of household pets should be discouraged. Flares of the dermatitis may be associated with the introduction of a new trigger factor into the environment, or even a new environment, such as a change of childminder, and so the trigger factor history should be reviewed frequently. Not only

can stress aggravate atopic dermatitis, but theseverely affected child is also a source of stress to the whole family (126,127). The doctor's role in giving simple reassurance and listening to family problems should not be underestimated. Stress can respond to treatment, and the dermatitis can be improved by using a variety of cognitive behavioural techniques and group therapy(128).

Dietary factors may aggravate the dermatitis of 15–35%(129) of children with atopic dermatitis, but in older children and adults this is far less common. Approaches to dietary management range from intensive investigation using double-blind food challenges (130) to empirical diets(131). The clinical benefit of dietary management is still debated, because there are few controlled studies of its value (127).

Even so, because some children benefit who have failed to respond to simple general and topical therapy,there is much to be said for a 3–4-week trial period on a modified elimination diet. Increased suspicion may be caused by a history of urticarial reactions or gastrointestinal reactions to foods. A positive specific raised IgE, particularly the specific circulating titres (IgE cap), may have a predictive value in young children (132,133)

### **3.11.3 Topical therapy**

*Corticosteroids.* The use of topical corticosteroids is absolutely recommended in AD. Their use requires experience as a choice must be made, depending on the clinical manifestations, the location of the lesions and the type of skin, between the more and the less potent ones, considering both the potential benefits and the side effects, so as to achieve a balance between them. In selecting the most adequate corticosteroid it must be remembered that their use will be required over long periods of time; those

with less side effects should therefore be preferred. The new-generation steroids provide good results, acceptable potency and a low transcutaneous absorption<sup>(134)</sup>.

There is limited clinical trial data to assist in choosing a corticosteroid. Ointment preparations are generally preferred over creams as they provide more uniform coverage and penetration. Also, the least potent preparation required to control AD (particularly in sensitive areas such as the face, neck, groin and underarms) should be utilized and, when possible, therapy should be stopped for short periods to reduce the risk of local and systemic adverse events. Often, a low-potency preparation, such as hydrocortisone acetate 1% or equivalent, is used for the face. Common local side effects of long-term topical corticosteroid use include striae (stretch marks), petechiae (small red/purple spots), telangiectasia (small, dilated blood vessels on the surface of the skin), skin thinning, atrophy and acne; however, these effects are uncommon with low or moderate potency preparations.

Systemic side effects with topical corticosteroid use are rare, but may include growth retardation in children, reduced bone density and hypothalamic-pituitary-adrenal axis suppression<sup>(119,120,122)</sup>.

*Monitoring corticosteroid use.* Topical steroids can cause side effects if abused. Complications related to systemic absorption are rare. It is advisable to educate patients about the quantities to apply—for example, the fingertip unit<sup>(134)</sup>—and to ask them to estimate the quantity used per month. Height and weight should be monitored in young children if they have severe dermatitis requiring moderately potent or potent steroids. Local side effects, such as permanent telangiectasis on the cheeks in babies and striae of the breasts, abdomen and thighs in adolescents, may be minimized if

appropriate steroid strengths are used. Particular care is required around the eyes, as glaucoma may be induced by topical steroids (136).

*Classical antipruritic therapies (camphor and menthol lotions).* These may help in palliating and controlling the itching sensation and can be beneficial for the evolution of the disease. As already stated, topical antihistamines should not be used because of their sensitising ability. However, a number of studies (137,138).

#### *Emollients*

There is no evidence that emollients improve atopic dermatitis directly. However, emollients are widely used because they improve the appearance and symptoms of the dry skin (xerosis) associated with this condition. (139,140,119).

#### *Topical Doxepin*

Topical doxepin produces some relief from itching within 48 hours. However, a clinically useful beneficial effect on disease severity has yet to be shown, and drowsiness may be a problem. (139).

#### Treatment of skin infections

As mentioned earlier, the skin of patients with AD is often heavily colonized with *S. aureus*, even at uninvolved sites. To avoid the development of bacterial resistance, short-term topical and/or oral antibiotic therapy is therefore recommended when an overt secondary bacterial infection is present. Appropriate systemic antibiotics are indicated for widespread secondary infection, and first- or second-generation cephalosporins or penicillins for 7 to 10 days are usually effective in managing the infection. Because erythromycin-resistant organisms are common in patients with

AD, macrolides are less useful alternatives (142,143). Patients with AD are also prone to recurrent viral infections. Eczema herpeticum (a severe disseminated herpes infection that generally occurs at sites of skin damage; also known as Kaposi's varicelliform eruption) is a serious risk in patients with widespread AD and may be easily misdiagnosed as a bacterial superinfection. Patients with the condition will require systemic antiviral treatment with acyclovir or other antiviral agents (141).

### **3.11.4 Intensive topical treatment**

#### **Wet-wrap technique**

This can be a useful technique for the control of severe atopic dermatitis in younger children. Two layers of absorbent tubular bandage are applied to the skin. The inner layer is presoaked in warm water and the outer layer is dry. A generous quantity of a low-potency topical corticosteroid is applied to the skin before the dressings. The dressings can be used overnight or changed every 12 h. This regimen can be used in hospital or for short-term outpatient treatment. Close supervision should be maintained, because suppression of the hypothalamic-pituitary axis can occur when topical steroids are employed (142).

#### Topical calcineurin inhibitors (TCIs)

TCIs are immunosuppressant agents that have also been shown to be effective for the treatment of AD. Two TCIs — pimecrolimus (Elidel) and tacrolimus (Protopic) — are currently approved in Canada for the second-line, intermittent treatment of immunocompetent patients 2 years of age and older with moderate-to-severe AD. Given the high costs of these agents and the fact that their long-term safety is not fully known, they are generally reserved for patients with persistent disease and/or

frequent flares that would require continuous topical corticosteroid treatment, or in patients severely affected in sensitive skin areas (e.g., around the eyes, face, neck and genitals) where systemic absorption and the risk of skin atrophy with topical corticosteroids are of particular concern (119,120,122).

The most common local adverse effects of TCIs are skin burning and irritation. Although a causal link has not been established, rare cases of skin malignancy and lymphoma have also been reported in patients using these agents. Therefore, both Health Canada and the Food and Drug Administration (FDA) recommend caution when prescribing TCIs. Long-term use should be avoided and patients using these agents should be counseled on appropriate sun protection (119,120,122,142).

### **3.11.5 Systemic therapy**

*Antihistamines.* These are by themselves insufficient for the management of AD, but they can be useful as a complementary therapy. Their efficacy is derived from both their antihistamine and their sedative effects; for these reasons, the most useful ones in this context are still hydroxyzine hydrochloride and ciproheptadine hydrochloride. Among the new-generation antihistamines, loratadine, terfenadine, acrivastine and cetirizine (143).

#### **Systemic corticosteroids**

Systemic corticosteroids are generally reserved for the acute treatment of severe AD flare-ups. However, prolonged use of oral steroids are associated with well known and potentially serious adverse effects and, therefore, their long-term use should be avoided. Furthermore, it is important to note that relapses are common following discontinuation of oral corticosteroid therapy (120).

*Interferon.* The altered balance between the Th-1 and Th-2 lymphocyte subpopulations, with predominance of the latter, causes the AD patients to evidence a deficient  $\gamma$ -interferon production; it thus appears to be reasonable to consider that the systemic administration of this substance may be beneficial in this disease. Several studies have demonstrated that therapy with  $\gamma$ -interferon at a dose of 5  $\mu$ g/m<sup>2</sup> per day for three months achieves good results with scarce side effects<sup>(144)</sup>.

*Leukotriene antagonists.* Leukotrienes are inflammatory mediators generated by the arachidonic acid cascade through the lipo-oxygenase pathway. Thus, inhibitors of this enzyme may be useful, fundamentally in bronchial asthma but also in AD. Carucci *et al.*<sup>(144)</sup>.

*Cyclosporin.* This is certainly an interesting option in the management of severe forms of AD<sup>(145)</sup>.

### **3.11.6 Phototherapy**

Numerous types of phototherapy have undergone trials for the treatment of severe atopic dermatitis, and seem to be effective. These include UVB, narrow-band UVB, medium and high-dose UVA1 and PUVA <sup>(146–150)</sup>. Some studies suggest that air-conditioned treatment cabinets improve patients' tolerance of phototherapy <sup>(151)</sup>.

### **3.11.7 Desensitization and immunotherapy**

Desensitization plays a very limited part in the management of patients with atopic dermatitis, even when an allergic factor has been firmly established clinically <sup>(152)</sup>.



## **AIM OF STUDY**

#### **4-AIM OF STUDY**

- 1) Measurement of the impact of atopic dermatitis on quality of life for infants and children up to 11 year attending eczema clinic in Jumhoria Hospital - Benghazi  
.Period extending from November 2010 to 30 July 2013
  
- 2) Assessment of relationship between severity of disease and life quality indices in the specified population

## **MATERIAL AND METHODS**

## 5-Method and material

The design of the study chosen is case series study, a convenience sample of 87 infants and children up to 11y with diagnosis of atopic dermatitis will be selected at their presentation with parents at visit to department of dermatology in Jumhoria hospital. The children as well as infant will be interviewed for the purpose of completing a previously designed form for data collection that contain data relevant to the children and infant (Appendx- I).

The questionnaire of child quality of life index contains 10 questions each question have (0-3) scores and gives a maximum score per questionnaire of 30. The higher the score the more quality of life is impacted. The questions are distributed on different areas; intensity of symptoms, social relationships, sleep, hobbies and also assess the involvement of treatment as a negative factor in the life of the child. (Appendx-II)

The IDQOL covers the following items: sleep and mood disturbances, difficulties in taking part in recreational activities or family life and discomfort when bathing, dressing and eating. It comprises ten questions, answered by the patient's parents on the basis of the previous week.<sup>153,154</sup> (Appendx-III).

Disease severity was assessed using the Eczema Area and Severity Index (EASI), which was developed by Hanifinet al.<sup>155</sup> (Appendx-IV)

EASI ( Eczema Area and Severity Index) score is a clinical index for severity assessment of eczema based on careful clinical examination of the involved areas in the body. It is a compound index that is calculated by summing structured indices for the major areas of the body; head and neck, upper limbs, trunk and lower limbs. Each area is examined for redness, thickening, scratching and lichenification so severity of each sign is scored from 0 to 3, the scores are summed on then being multiplied by fixed factor then by an estimated involved area score, and finally summation of all areas yield the total score. The total score ranges between 0 and 72 with more severity with higher score.

The collected data are then entered to computer program for calculation of scores then statistical analysis using the appropriate tests.

Notes:

The eczema area and severity index

Hanifin used 6 areas so score from (0 – 72)

Mild <6

Moderate 6 – 17

Severe  $\geq 18$

In this study used 4 areas so score from( 0- 48 ) so

Mild < 4

Moderate 4 – 11

Severe  $\geq 12$

#### **Statistical analyses:**

All statistical analyzes were conducted using SPSS Statistical package for social Sciences- version 18. Descriptive statistics ,as mean , standard deviation, median and mode were used. The relationship between the severity of CDLQI Score and eczema area and severity score was assessed using Pearson's correlation. Data were presented in form of tables and figures , were the figures done by Microsoft Excel 2003.

## **RESULTS**

## 6-Results

Among 87 patients of atopic dermatitis (A.D) enrolled in case series study ,51 patients (58.6 %) were female and 36 patient (41.4% ) male (figure -1) and female to male ratio was 1.4 : 1 (table 1)

The distribution of patient according to age under 3 year 50 patient (57.5%) and over 3 year 37 patient (42.5%) (figure -2)and mean = 4.1 With age between1- month to -12 year (table -2)

The distribution of patient according to going school and player schoolUp to 53 patient ( 60.9 % ) no going school and 34 patient ( 39.1 % ) go school and player school (figure -3) significant difference in psychosocial characteristic were observed between male and female significant level of anxiety and higher stress in female going to school due to exposure to life events (figure-3).

The duration of disease was ranging from months to five year (mean = 2.7 year) , (16.1 % of the patient unclear history duration (table -3 )(figure -4)

The patient according to mother occupation was (62.1 % ) of patient mother house wife (figure -5)

Type of eczema (64%) of patient childhood type and (54%) infantile type (figure -6)

Patient have history of allergic disease like bronchial asthma (34.6%) allergic rhinitis (5.7%) and on history of allergic (54%) (figure -7)

Forty six percent of patients have history of other allergic disease, have bronchial asthma and allergic rhinitis together and 8% have cow milk allergy. Family history of allergic diseases were recorded in 79.3% , 25.3% had history of bronchial asthma and 26.4% had bronchial asthma and allergic rhinitis together. Social relation between

Atopic and allergic symptom more severe symptom more severe diseases and more affect( QOL), and atopic without allergic symptom mild. these findings may indicate that mild atopic (asymptomatic) less or not affects quality of life(figure -8)

Quality of life index in (CDLQI)children dermatology quality of life indexExtremely large effect (41.4%) mean = 17.8 (table -4) (figure -9)

Quality of life index in ((IDLQI) infant dermatology quality of life index large and Extremely larger effect (84%) (table -5) (figure -10).

For CDLQI the majority of patient had score between very large and extremely large effect about (91.9%) (table-6) (figure-11)

Clinical assessment of the severity of atopic dermatitis according to EASIEczema area severity index demonstrated that the majority of patient had moderate (74.7%) and severe (6.9%) of patient with mean score  $6.4 \pm 3.53$  and clinically patient with severe eczema have poor QOL (table -7) (figure -12)

IDQOL high score was for itching, scratching and mood disturbance (question 1 and 2) which the mean score was 2.45 and 2.28 respectively.(table-8)

The high score of CDLQI was for itching,scratching, mood and other peoplebehavior (question 1,2 and 8)in which mean score was 2.4, 2.3 and 2.2 respectively.(table-9)

The EASI score revealed that majority of patients (74.7%) have moderate severity18.4% have mild severity and 6% have severe severity

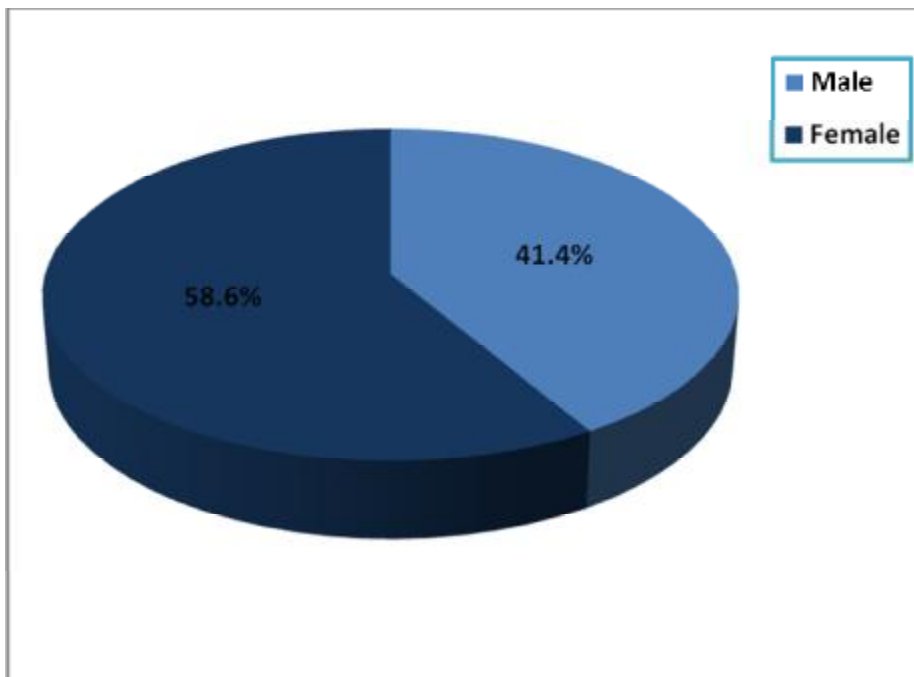
No significant association were found by statistical analysis regarding CDQOL and EASI ( $r = 0.14$ ) (table -10 ) (figure -13)



**Table 1: Distribution of patients according to sex .**

Sex	No.	%
Male	36	41.4
Female	51	58.6
Total	87	100

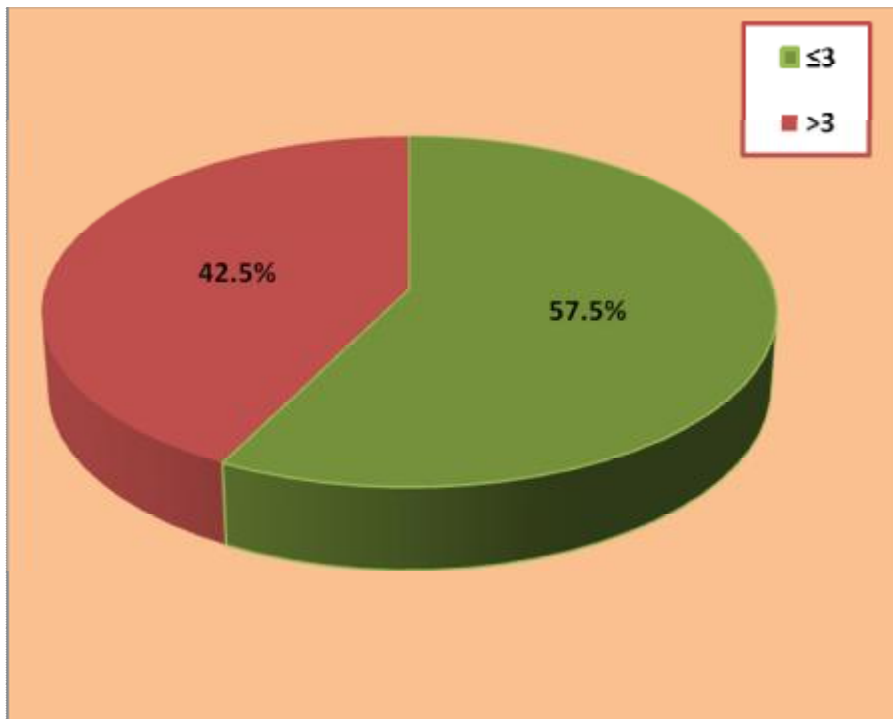
Female to male ratio was.1.4:1

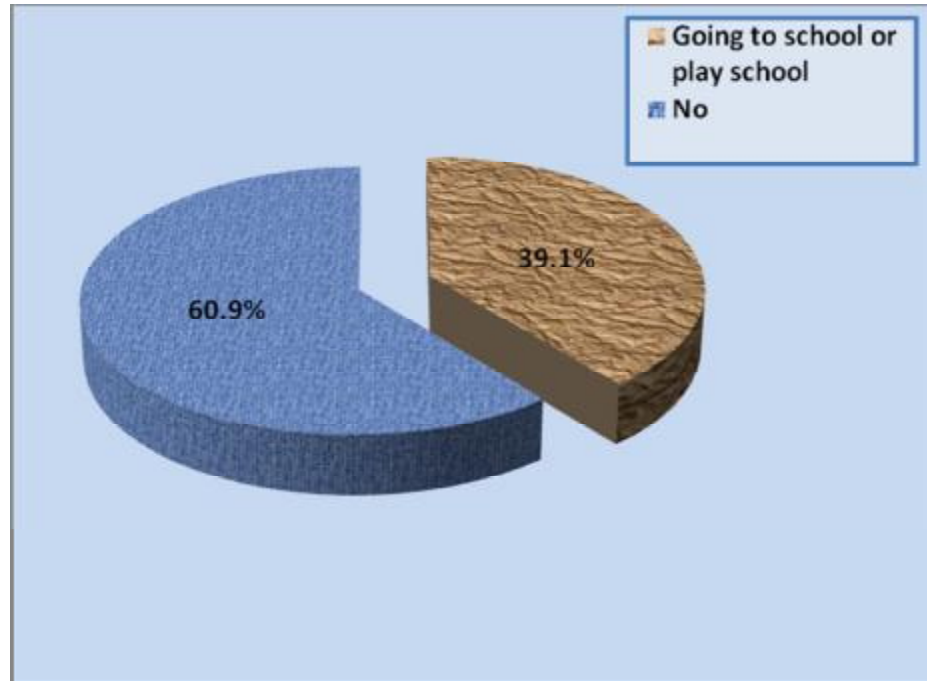
**Fig. 1: Distribution of patients according to sex .**

**Table 2: Distribution of patients according to age .**

Age /year	No.	%
$\leq 3$	50	57.5
$>3$	37	42.5
<b>Total</b>	<b>87</b>	<b>100</b>

Mean= 4.1 years. Std.Deviation =3.4years.Minimum age =One month. Maximum =12 years.

**Fig. 2: Distribution of patients according to age .**

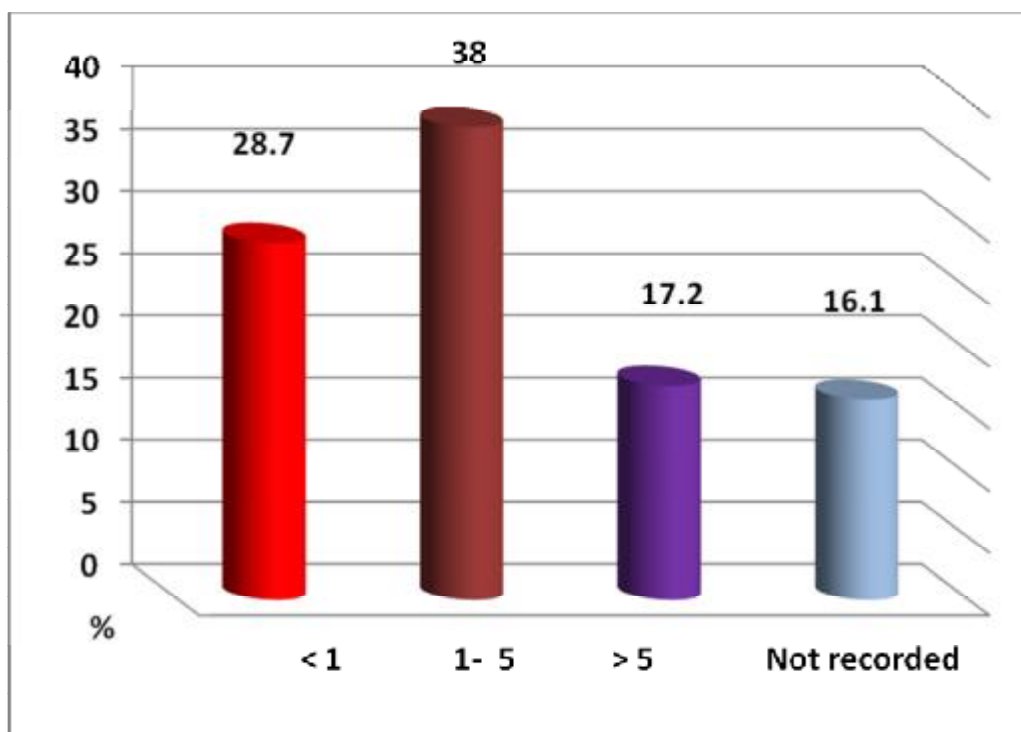


**Fig.3: Distribution of patients according to going to school.**

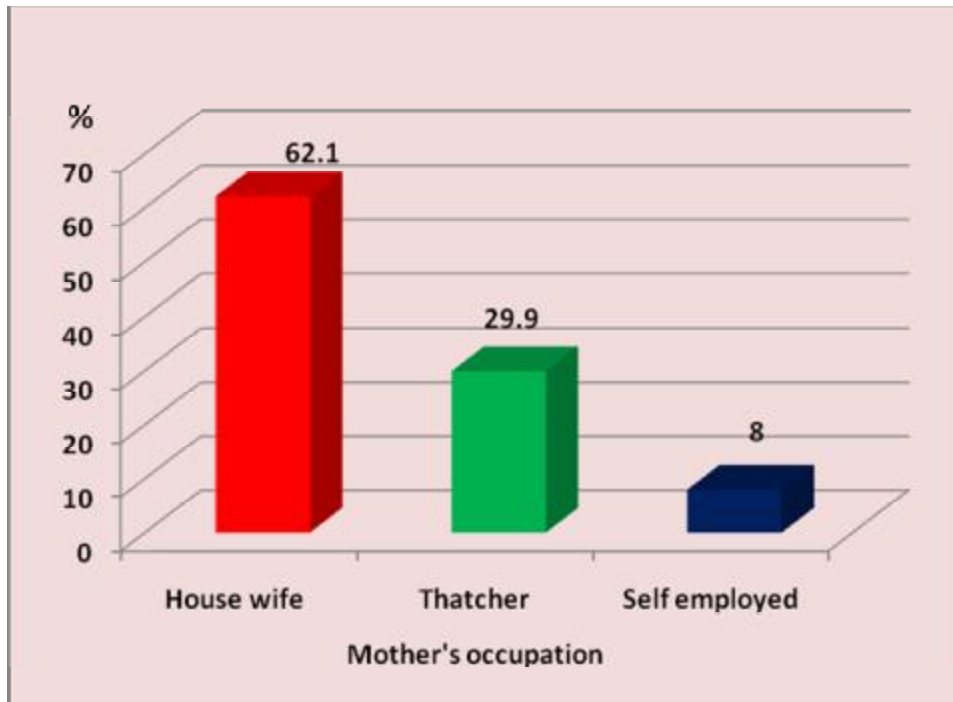
**. Table3: Distribution of patients according to duration of disease**

<b>Duration of disease/year</b>	<b>No.</b>	<b>%</b>
<b>&lt; 1</b>	<b>25</b>	<b>28.7</b>
<b>1 – 5</b>	<b>33</b>	<b>38</b>
<b>&gt;5</b>	<b>15</b>	<b>17.2</b>
<b>Not clear history</b>	<b>14</b>	<b>16.1</b>
<b>Total</b>	<b>87</b>	<b>100</b>

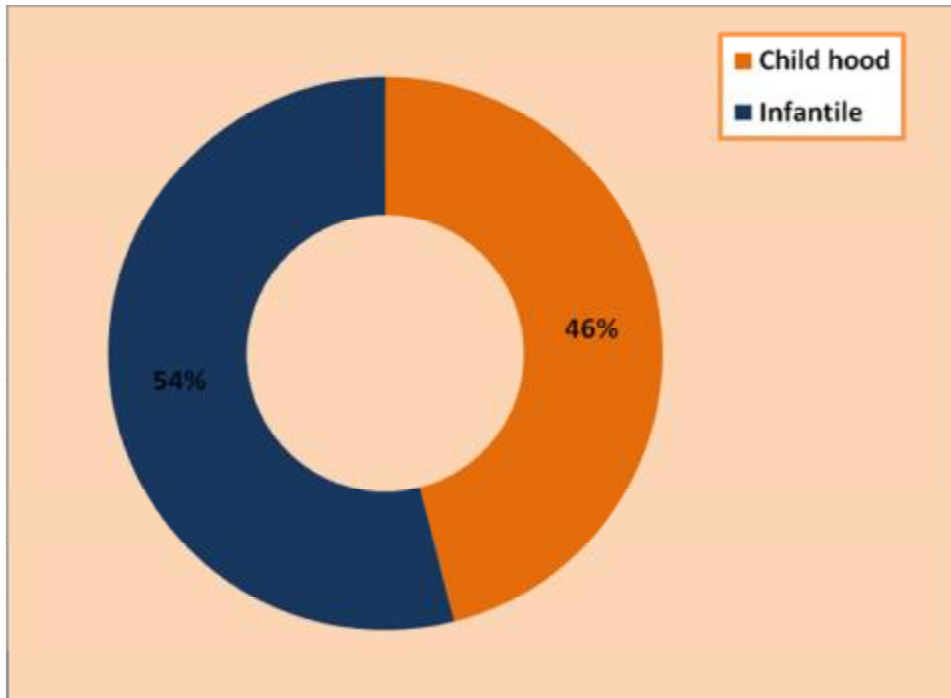
**Mean= 2.7years. Std.Deviation = 2.6years.Minimum = 2months Maximum = 8 years and half.**



**Fig.4: Distribution of patients according to duration of disease .**



**Fig.5: Distribution of patients according to mother's occupation .**



**Fig.6: Distribution of patients according to type of eczema.**

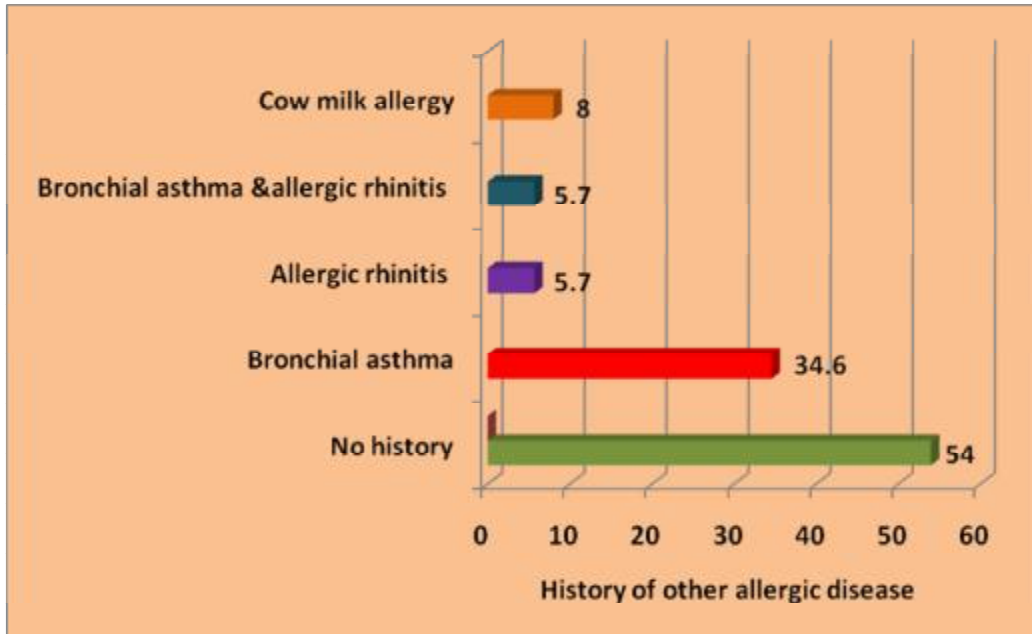


Fig .7:Distribution of patients according to history of other allergic disease .

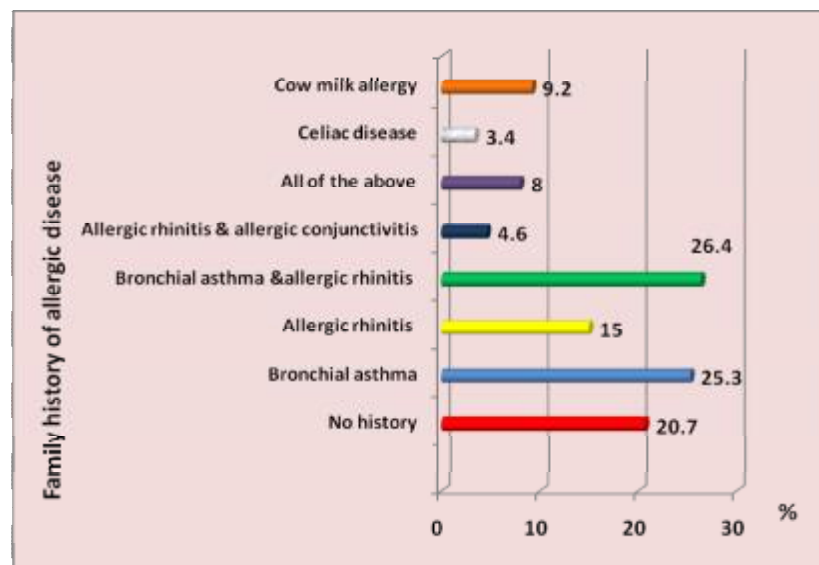
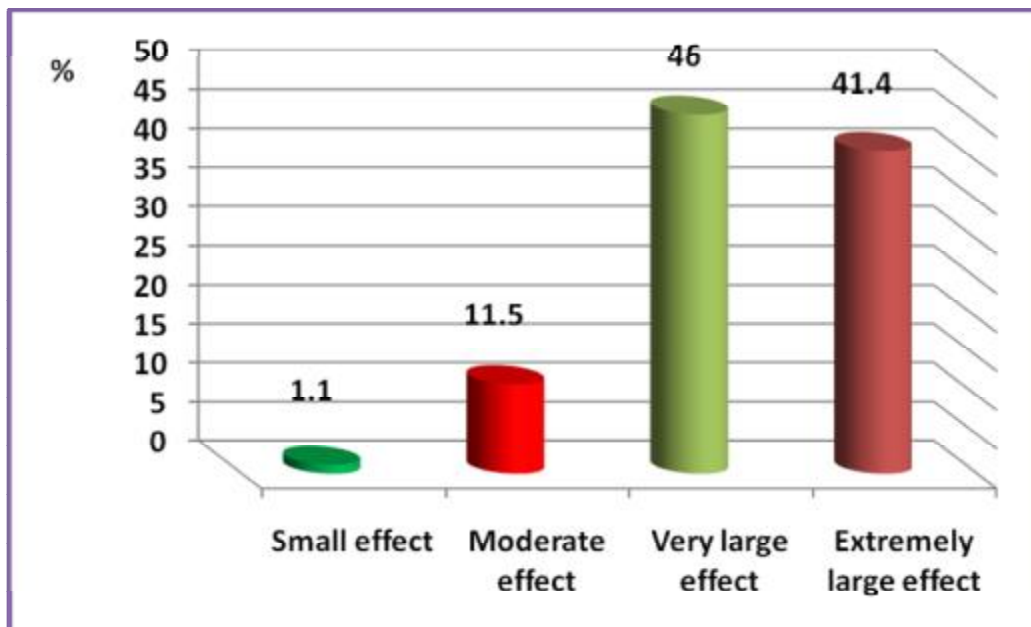


Fig.8: Distribution of patients according to family history of allergic disease .

**Table4: Distribution of patients according to severity of CDLQI scores (All children).**

Severity of CDLQI scores	No.	%
Small effect	1	1.1
Moderate effect	10	11.5
Very large effect	40	46
Extremely large effect	36	41.4
Total	87	100

Mean=17.8. Std.Deviation =4.76 . Minimum=6. Maximum= 28.Range =22.



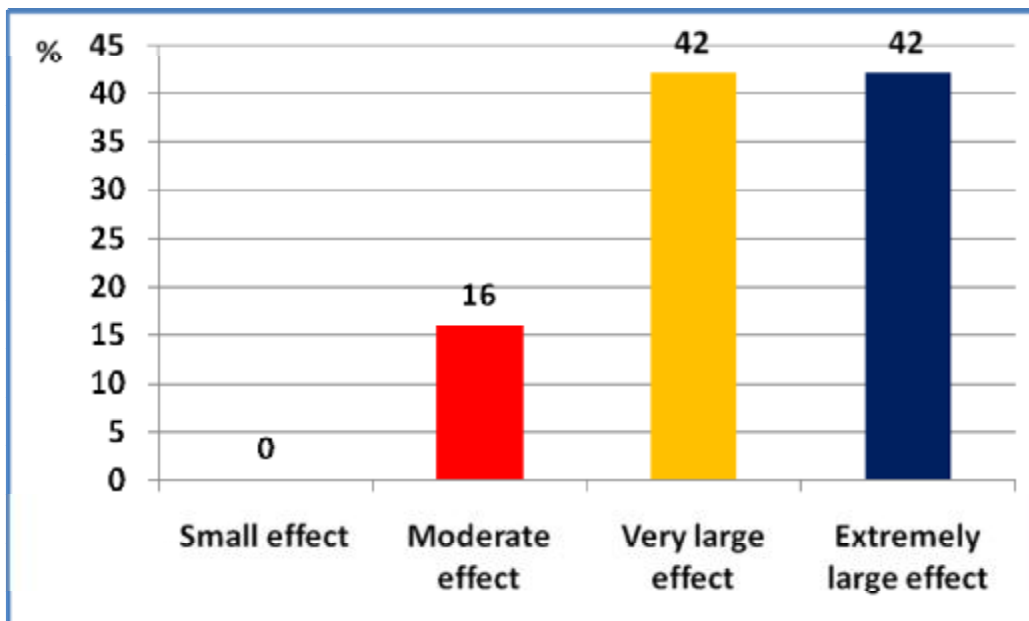
**Fig9: Distribution of patients according to severity of CDLQI scores ( All children).**



**Table5: Distribution of patients according to severity of IDQLI scores.( Age ≤ 3years)**

Severity of CDLQI scores	No.	%
Small effect	0	0
Moderate effect	8	16
Very large effect	21	42
Extremely large effect	21	42
Total	50	100

Mean= 17.86. Std.Deviation = 4.79. Minimum= 8. Maximum= 26.Range = 18.

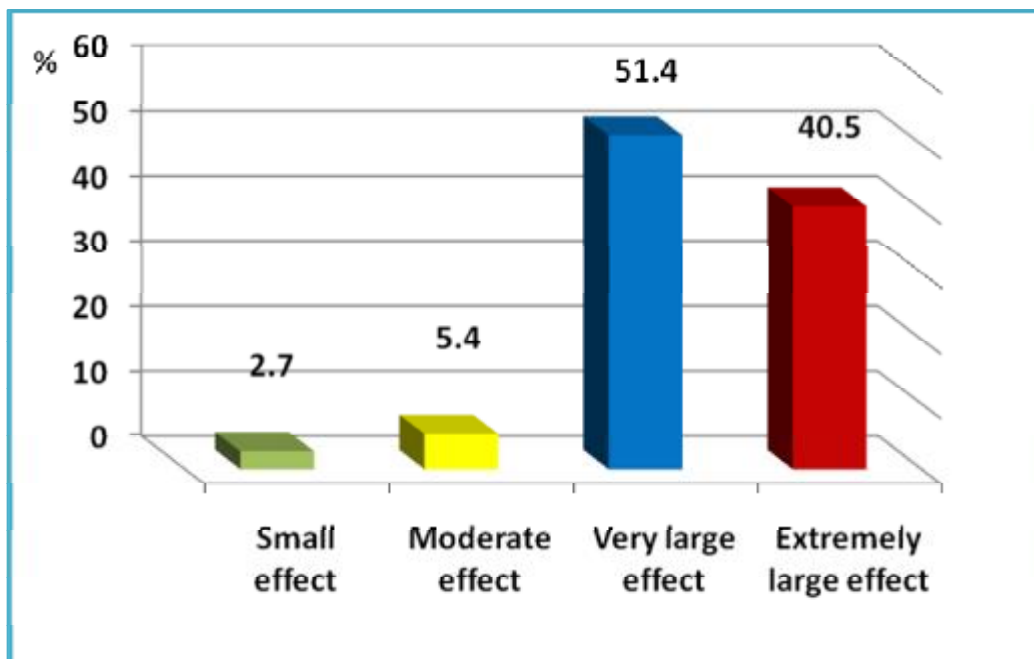


**Fig. 10: Distribution of patients according to severity of IDQLI scores.( Age ≤ 3years)**

**Table6: Distribution of patients according to severity of CDLQI scores( Age >3years).**

Severity of CDLQI scores	No.	%
Small effect	1	2.7
Moderate effect	2	5.4
Very large effect	19	51.4
Extremely large effect	15	40.5
Total	37	100

Mean= 17.6. Std.Deviation =4.79. Minimum= 6. Maximum=28.Range =22.

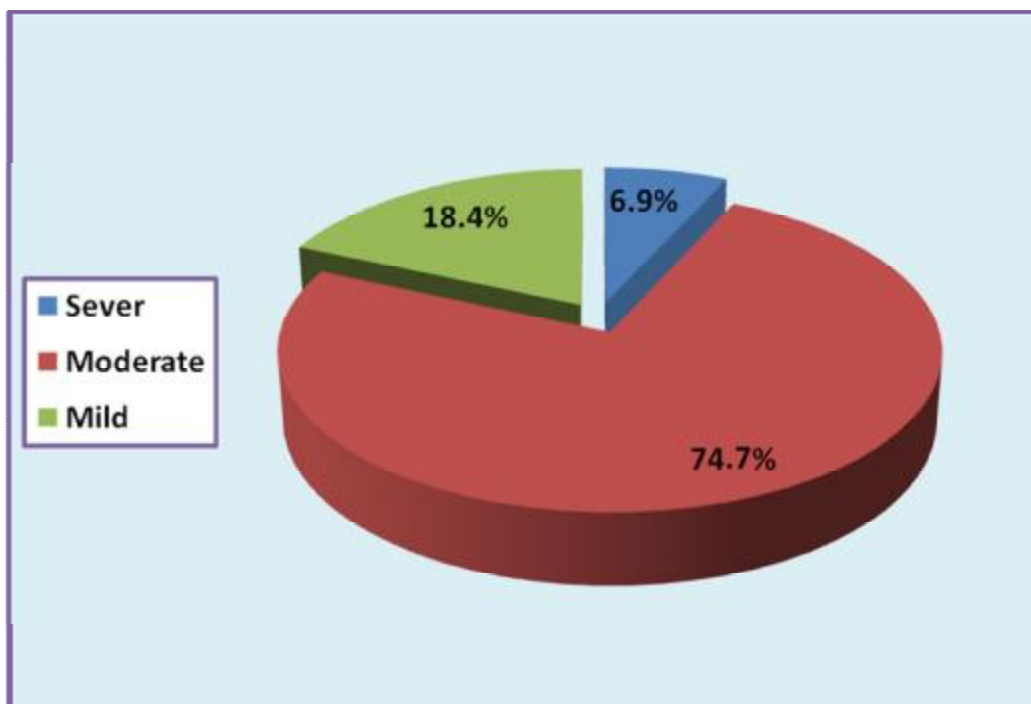


**Fig.11: Distribution of patients according to severity of CDLQI scores( Age >3years).**

**Table7: Distribution of patients according to eczema area and severity.**

Severity	No.	%
Mild	16	18.4
Moderate	65	74.7
Sever	6	6.9
Total	87	100

Mean=6.43 Std.Deviation =3.53 Median = 6.3 Minimum=0 . Maximum = 16.4 .

**Fig.12: Distribution of patients according to eczema area and severity score.**

**Table8:Mean and Standard deviation for the Question about the severity of atopic dermatitis IDQLI.**

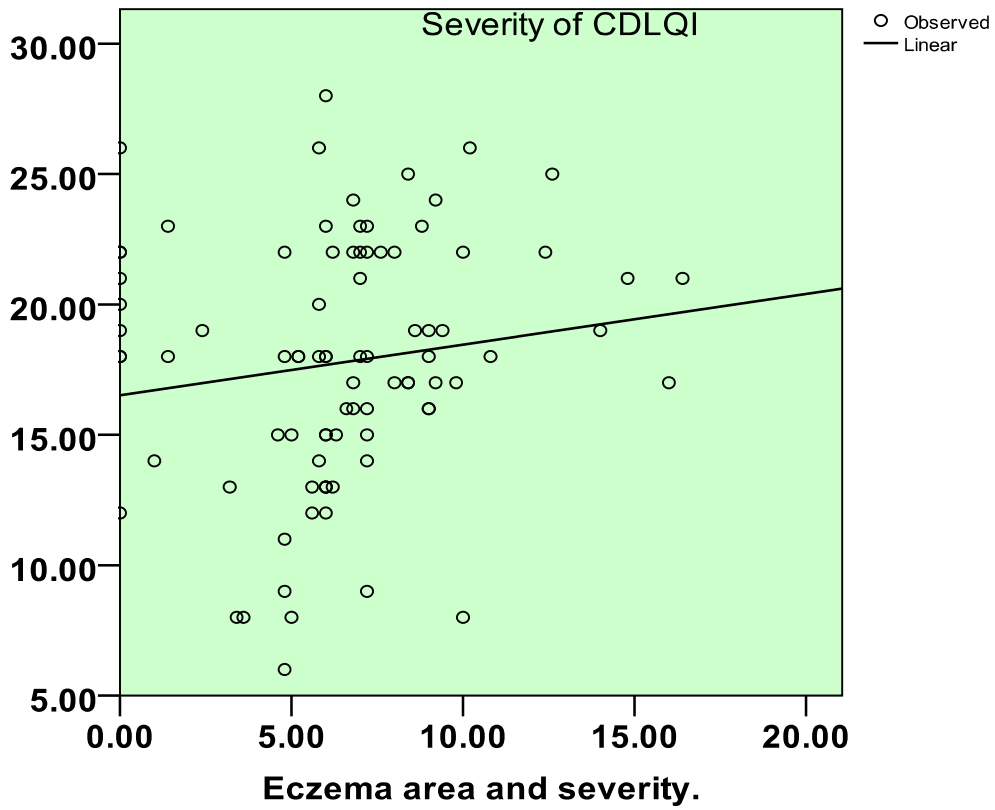
<b>Questions</b>	<b>Mean (SD)</b>
<b>1. Itching and scratching</b>	<b>2.45(0.62)</b>
<b>2. Mood</b>	<b>2.28(0.85)</b>
<b>3. Time to get to sleep</b>	<b>1.7(0.73)</b>
<b>4. Sleep disturbances</b>	<b>1.8(0.59)</b>
<b>5. Disturbed playing or swimming</b>	<b>1.5(0.59)</b>
<b>6. Disturbed family activities</b>	<b>1.5(0.58)</b>
<b>7. Problems during mealtimes</b>	<b>1.59(0.82)</b>
<b>8. Problems from treatment</b>	<b>1.4(0.88)</b>
<b>9. Dressing problems</b>	<b>1.7(0.79)</b>
<b>10. Problems at bath time</b>	<b>1.7(0.84)</b>
<b>Total score</b>	<b>17.9(4.8)</b>

**Table9:Mean and Standard deviation for the Question about the severity of atopic dermatitis CDLQI.**

<b>Questions</b>	<b>Mean (SD)</b>
<b>1. Itching and scratching</b>	<b>2.4(0.59)</b>
<b>2. Mood</b>	<b>2.3(0.48)</b>
<b>3.Effect relation with friends</b>	<b>1.8(0.68)</b>
<b>4. Problem with clothing</b>	<b>1.6(0.76)</b>
<b>5. Disturbed playing or going out</b>	<b>1.6(0.76)</b>
<b>6.Avoide sporting</b>	<b>1.5(0.69)</b>
<b>7. Effect their study</b>	<b>1.5(0.76)</b>
<b>8. Other people behavior</b>	<b>2.2(0.73)</b>
<b>9. Sleep problem</b>	<b>1.5(0.83)</b>
<b>10. Problems with treatment</b>	<b>0.84(0.79)</b>
<b>Total score</b>	<b>17.5(4.82)</b>

**Table10: Distribution of patients according severity of CDLQI scores and EASIA.**

Severity of CDLQI scores (All)	EASIA							
	Mild		Moderate		Sever		Total	
	No.	%	No.	%	No.	%	No.	%
Small	0	0	1	100	0	0	1	100
Moderate	3	30	7	70	0	0	10	100
Very large	5	12.5	34	85	1	2.5	40	100
Extremely large	8	22.2	23	63.9	5	13.9	36	100
Total	16	18.4	65	74.7	6	6.9	87	100



**Fig.13: Correlation between severity of CDLQI Score and eczema area and severity score.**

**Pearson Correlation between severity of CDLQI severity score.  $r = 0.144$   
 $p=0.183$ (Not significant).scores and eczema area and**



Picture 1: 6 month female with sever AD



Picture 2: same patient after one week receive treatment

## **DISCUSSION**



## Discussions:

Total of 87 patients was enrolled in this study , 41.4% males and 58.6% females, female to male ratio was 1.4:1, compared to 45.5% males and 54.5% were females in one study<sup>29</sup> , and 55% males and 45% females, in another study.<sup>30</sup>

We observed differences in psychosocial characteristics between males and females. The level of anxiety and stress were higher in females of school age, probably due to exposure to life events.

Age  $\leq$  3 years constitutes 57.5% of patients, and 42.5% were older than 3 years. Mean age was equal to  $4.1 \pm 3.4$  years , with minimum age of one month and maximum age of 12 years. 39.1% of them were school pupils, or enrolled in playschool .

More than half (62.1%) of mothers are house wives; 29.9% of them were teachers and 8% were self-employed. However, in other studies, the majority of mothers complained of spending most of their caring for their affected children disease, and many mothers chose to stay home and not to return to work after maternity leave is over, because of their children's disease. Parents were reluctant to leave their children with relatives, child care, or babysitters. Moreover, they had difficulty finding child care service that would comply with the medication regimen.<sup>77</sup> Mean duration of disease was  $2.7 \pm 2.6$  years, with a minimum of 2 months and the longest duration was 8 years. Childhood eczema constitutes 46% , and infantile eczema 54%.

Forty six percent of patients have history of other allergic disease, or have bronchial asthma and allergic rhinitis together, and 8% have cow milk allergy. Family history of allergic diseases were recorded in 79.3%.

25.3% had history of bronchial asthma and 26.4% had bronchial asthma and allergic rhinitis together. Social relationships and QOL were severely affected in children with

atopic and allergic symptoms, while those without symptoms of allergy and atopy had mild impact on QOL..

These findings may indicate that mild atopic (asymptomatic) **lees** or does not affect quality of life. Another study found that genetics has clearly been shown to play a large role in the development of AD. Approximately 70% of AD patients have a family history of atopy. Children with one allergic parent becomes allergic 50% of the time, increasing to 75% if both parents are allergic.<sup>77</sup> For age  $\leq$  3years (41.4% of patients) they had extremely large effect of the disease on QOL, and 46% had very large effect. Mean CDLQI score for children  $\geq$  3years was  $17.86 \pm 4.79$ , and for patients  $<$ 3years IDLQI was of very large effect in 51.4%, and in 40.5% was of extreme large effect, with mean score equal to  $17.6 \pm 4.79$ . In other studies, the mean score of CDLQI was  $12.83 \pm 6.52$ .<sup>97</sup>

Clinical assessment of the severity of AD, according to EASI, demonstrated that the majority of patients had moderate (74.7%) and severe (6.9%), with mean score  $6.4 \pm 3.53$ . One study demonstrated that the majority of patients had mild forms of dermatosis, with mean of 9.2.<sup>29</sup> Another study found that the mean was 6.0 (0.3-60), which is less than that of this study.<sup>9</sup> A third study found that 27% of the children had mild disease, 62% a moderate, and 11% a severe disease.<sup>78</sup>

The range of score for IDQLI was 8 - 26, while in CDLQI it was 6 - 28. In another study, it was found that the mean score for IDQOL1 was 6.64 with a standard deviation (SD) of 4.32, and a range of 1-20, and for IDQOL2, the mean score was 6.43, SD of 4.33, and a range of 1-22. The mean score for IDQOL3 was 4.52, SD of 3.67, with a range of 0-20.<sup>99</sup>

For infants  $\leq 3$  years, the mean score of the 10 questions was  $(17.9 \pm 4.8)$  with high score for itching and scratching  $(2.45 \pm 0.62)$ , mood  $(2.45 \pm 0.85)$  and sleep Disturbances  $(1.8 \pm 0.59)$ .

For children  $> 3$  years, there was high score for itching and scratching  $(2.4 \pm 0.59)$ , mood  $(2.3 \pm 0.48)$  and other people behavior  $(2.2 \pm 0.73)$ . The total mean score  $(17.5 \pm 4.82)$ .

Another study recorded that the cardinal symptom of itching causes scratching, which is associated with markedly disturbed sleep patterns for the majority of children (over 60%)<sup>8-94</sup>. Sleep loss leads to physical and mental exhaustion for part or all of the family causing mood disturbance, loss of concentration and impaired performance at school or work<sup>10,78</sup>. Parents may lose an average of 2.5 hours sleep per night, particularly during flares of the disease<sup>96</sup>, and others found that 38% of siblings of children with AD also had disturbed sleep<sup>77</sup>.

A result from another study recorded that sleep disruption and itching/scratching were the most prevalent physical symptoms for affected children. Sleep problems for the child were reported in 22 of the 23 parent interviews and in all of the expert interviews, and itching and scratching were reported in all interviews.<sup>77</sup>

The mean score obtained by questions about the effect on mood (psychological disturbance), for infants was  $(2.28 \pm 0.85)$ , and for children was nearly the same  $(2.3 \pm 0.48)$ . A study showed that atopic dermatitis can cause psychological difficulties for the child, parent(s) or other family members and may affect their interpersonal relationships.<sup>100-103</sup> Psychological disturbance may also promote disease flares or affect clinical management. Others found double the rate of psychological disturbance in school-aged children.<sup>104</sup> Another study regarding separate questions, the highest score was found for itching and scratching (question 1: mean 1.28, SD 0.89).

The lowest scores concerned family activities (question 6: mean 0.20, SD 0.47) and problems during mealtimes (question 7: mean 0.14, SD 0.35)<sup>11</sup>, others reported that itching, treatment, and sleep showed the highest scores.<sup>8</sup>

According to severity of CDLQI score and EASIA, a mild score was 18.4%, a moderate 74.7%, and a severe was 6.9%. 85% of very large severity of CDLQI (for all children) was moderate in EASIA correlation. 63.9% of extremely large severe in

CDLQI (all children) also moderate in EASIA correlation.

Correlation between severity of CDLQI score and eczema area and severity score was estimated using Pearson's correlation coefficient, which was 0.144, indicating an insignificant association. The same result was recorded in another study where Pearson's correlation coefficient was 0.219.<sup>29</sup> While another reported a mean EASIA score of  $8.51 \pm 8.64$ , and both CDLQI and EASIA scores showed significant correlation ( $p < 0.001$ ).<sup>97</sup>

Atopic dermatitis may have impact on the quality of life in various domains. In our study, we did not find significant differences in subjective estimate of quality of life in the domains of physical health.

## **CONCLUSION**

## 8-Conclusions

Educational and psychological support for patient and their family need more than physical treatment of symptoms to improve their long-term physical outcome.

Atopic dermatitis most often begins in infancy or early childhood, with about 90% of cases appearing in the first five years of life, which is a critical period for psychosocial development. Both physiological and psychological abnormalities have been documented in young children with atopic dermatitis.

In this study;

Female more affect than male

IQOL higher score for itching scratching and mood disturbance

CQOL high score for itching and scratching and mood and other people behavior

Majority of patient with history of allergy diseases more severe effect QOL

Strong association between sever A.D and decrease QOL

We conclude that AD has a negative impact on the quality of life of pediatric patients and their families.

# **RECOMMENDATION**

## **9-RECOMMENDATION**

Data obtained in studies of quality of life in AD should be used to guide clinical practice in order to identify individual treatment strategies and should lead to the adoption of measures to reduce the impact of the disease on patients and their families.



# **APPENDIX**

NO.[ ] NAME[ ] DATE OF COLLECTION [ - - 2011 ] T.N.....

**1**Age [ ] **2**Gender [M F] **3**Nationality [L E Other] **4**Residency [Ben Other] **5**School class[ ] **6**Duration[ ]

**7**Mother's job [ H.W.\*Teacher\*Other.....] **8**Mother's education:[ No level\*Basic\*Secondary\*High\*]

Disorder / Symptoms		a. No sym.	b. Has symptoms	Diagnosed	
				e. In the Patient	f. In the Family
<b>1</b> Eczema	1.1.Itching	<input type="radio"/>	<input type="radio"/>		
	1.2.Dryness	<input type="radio"/>	<input type="radio"/>		
	1.3.Erythema	<input type="radio"/>	<input type="radio"/>		
	1.4.Scaling	<input type="radio"/>	<input type="radio"/>		
<b>2</b> B.A.	2.1.Wheeze	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
	2.2.Cough	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
	2.3.Dyspnea	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
<b>3</b> A.R.	3.1.Sneezing	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
	3.2.Itching	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
	3.3.Runny	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
<b>4</b> A.C.	4.1.Redness	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
	4.2.Itchy	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
	4.3.Tearing	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>	<input type="radio"/>
<b>5</b> Celiac disease				<input type="radio"/>	<input type="radio"/>
<b>6</b> Cow milk allergy				<input type="radio"/>	<input type="radio"/>

**1**Type of eczema [ ] **2**Treatment: STARTED ANY KIND  NOT

**1**H/N :-1.1.Redness[ ] 1.2. Thickening[ ] 1.3. Scratching[ ] 1.4.Lichenification[ ] • 1.5. Area involved [ ]

**2**UL :-2.1.Redness[ ] 2.2. Thickening[ ] 2.3. Scratching[ ] 2.4.Lichenification[ ] • 2.5. Area involved [ ]

**3**Trunk:-3.1.Redness[ ] 3.2. Thickening[ ] 3.3. Scratching[ ] 3.4.Lichenification[ ] • 3.5. Area involved [ ]

**4**LL :-4.1.Redness[ ] 4.2. Thickening[ ] 4.3. Scratching[ ] 4.4.Lichenification[ ] • 4.5. Area involved [ ]



## REFERENCES

## 10-Reference

- 1) Ring J, Huss-Marp J. Atopic Eczema. *Karger Gazette* 2004;67: 7-9.
- 2) Lilja G, Wickman M. Allergy-atopy-hypersensitivity-a matter of definition. *Allergy* 1998; 53: 1011-2.
- 3) Stoenescu M. “Atopy”, “allergy” and “hypersensitivity” are-explicitly or implicitly-defined in different ways. *Allergy* 1999; 54: 640-2.
- 4) Dubois AE, de Monchy GR, Schouten JP, et al. Basic concepts relating to the field of allergology. *Allergy* 1999; 54: 760-2.
- 5) Kiebert G, Sorensen SV, Revicki D *et al.* Atopic dermatitis is associated with a decrement in health-related quality of life.*Int J Dermatol*2002; **41**: 151–158.
- 6) Lapidus CS. Role of social factors in atopic dermatitis: the US perspective. *J Am Acad Dermatol*2001; **45** (Suppl. 1): S41–S43.
- 7) Simpson EL, Hanifin JM. Atopic dermatitis. *J Am Acad Dermatol*2005; **53**: 115–128.
- 8) Stores G, Burrows A, Crawford C. Physiological sleep disturbance in children with atopic dermatitis: a case control study. *PediatrDermatol.* 1998;15:264–268
- 9) Reuveni H, Chapnick G, Tal A, Tarasiuk A. Sleep fragmentation in children with atopic dermatitis. *Arch PediatrAdolesc Med.* 1999;153: 249–253
- 10) Dahl RE, Bernhisel-Broadbent J, Scanlon-Holdford S, Sampson HA, Lupo M. Sleep disturbances in children with atopic dermatitis. *Arch PediatrAdolesc Med.* 1995;149:856–860
- 11) Beattie PE, Lewis-Jones Ms. A comparative study of Impaired quality of life in children with skin disease and children with other chronic diseases. *British Journal of dermatology* 2006; 155: 145-151.
- 12) Noor A Ziah Ma, Rosnah T, et al. Childhood atopic dermatitis: A measurement of quality of life and family impact. *Medical journal of Malaysia* 2002; 57(3): 329-336.

- 13) Ben-Gashir, M. A. Relationship between quality of life and disease severity in atopic dermatitis/eczema syndrome during childhood. *Current Opinion in Allergy & Clinical Immunology*: October 2003 - Volume 3 - Issue 5 - pp 369-373
- 14) Grange A, Bekker H, Noyes J, et al. Adequacy of health-related quality of life measures in children under 5 years old: systematic review. *J Adv Nurs*. 2007;59:197-220.
- 15) Klatchoian DA, Len CA, Terreri MT, et al. Quality of life of children and adolescents from São Paulo: reliability and validity of the Brazilian version of the Pediatric Quality of Life Inventory™ version 4.0 Generic Core Scales. *JPediatr (Rio J)*. 2008;84:308-15.
- 16) Misery L, Finlay AY, Martin N, et al. Atopic dermatitis: impact on the quality of life of patients and their partners. *Dermatology*. 2007;215:123-9.
- 17) Lewis-Jones MS, Finlay AY. The children's Dermatology Life Quality Index (CDLQI): initial validation and practical use. *Br J Dermatol*. 1995;132:942-9.
- 18) Finlay AY, Khan GK. Dermatology Life Quality Index (DLQI): a simple practical measure for routine clinical use. *Clin Exp Dermatol*. 1994;19:210-6.
- 19) Drake L, Prendergast M, Maher R *et al*. The impact of tacrolimus ointment on health-related quality of life of adult and paediatric patients with atopic dermatitis. *J Am Acad Dermatol* 2001;44 (Suppl. 1): S65–72.
- 20) Williams H. Disease definition and measures of disease frequency. *J Am Acad Dermatol* 2001;45: S33–6.
- 21) Hanifin J, Saurat JH, eds. Understanding atopic dermatitis: pathophysiology and etiology. *J Am Acad Dermatol* 2001;45: S1–68.
- 22) Eedy DJ. What's new in atopic dermatitis? *Br J Dermatol* 2001; 145: 380–4.

- 23) Lilja G, Wickman M. Allergy-atopy-hypersensitivity-a matter of definition. *Allergy* 1998; 53: 1011-2.
- 24) Stoenescu M. "Atopy", "allergy" and "hypersensitivity" are-explicitly or implicitly-defined in different ways. *Allergy* 1999; 54: 640-2.
- 25) Dubois AE, de Monchy GR, Schouten JP, et al. Basic concepts relating to the field of allergology. *Allergy* 1999; 54: 760-2.
- 26). Larsen FS, Hanifin JM: Epidemiology of atopic dermatitis. *Immunol Allergy Clin North Am* 2002, 22:1-25.
- 27) Bieber T: Mechanisms of disease: atopic dermatitis. *N Engl J Med* 2008, 358:1483-1494.
- 28). Kuster W, Petersen M, Christofer E. A family study of atopic dermatitis. Clinical and genetic characteristics of 188 patients and 2.151 family members. *Arch Dermatol Res* 1990; 282: 98-102.
- 29). Sarah L. Chamlin, David Cella, Wilona J. Frieden, zy Mary L. Williams, zy Anthony J. Mancini, Jin-Shei Lai, w and Mary-Margaret Chrenzz Development of the Childhood Atopic Dermatitis Impact Scale: Initial Validation of a Quality-of-Life Measure for Young Children with Atopic Dermatitis and their Families. *J Invest Dermatol* .125:1106 –1111, 2005
- 30)-Stephanie Sturgill and Laurie A. Bernard. Atopic dermatitis update *Curr Opin Pediatr* 2004;16:396–401.
- 31) Rothe MJ, Grant-Kels JM. Atopic dermatitis: an update. *J Am Acad Dermatol* 1996; 35: 1-13.
- 32)-A. H. Liu, F. D. Martinez, and L. M. Taussig, "Natural history of allergic diseases and asthma," in *Pediatric Allergy: Principles and Practice*, D. Y. M. Leung, H. A. Sampson, R. S. Geha, and S. J. Szefler, Eds., pp. 10–22, Elsevier, Philadelphia, Pa, USA, 2003

- 33). Coleman R, Trembath RC, Harper JI. Chromosome 11q13 and atopy underlying atopic eczema. *Lancet* 1993; 341: 1121-1122.
- 34). Cookson WO, Young RP, Sanford AJ, Moffat MF, Shirakawa T, Sharp PA, et al. Maternal inheritance of atopic IgE responsiveness on chromosome 11q. *Lancet* 1992; 340: 381-384
- 35) Cork MJ, et al. Epidermal Barrier Dysfunction in Atopic Dermatitis. *J Inv Dermatol* 2009. 129:1892-1908
- 36)\_ Leung DYM, Bhan AK, Schneeberger EE, et al. Characterization of mononuclear cell infiltrate in atopic dermatitis using monoclonal antibodies. *J Allergy Clin Immunol* 1983;71:47-56
- 37)\_ Boss JD, Hagenara C, Das PK, et al. Predominance of 'memory' T cells (CD4+, CDw29+) over 'naive' T cells (CD4+, CD45R+) in both normal and diseased human skin. *Arch Dermatol Res* 1989;81:24-30
- 38)\_ Picker LJ, Martin RJ, Trumble A, Newman LS, Collins PA, Bergstresser PR, et al. Differential expression of lymphocyte homing receptors by human memory/effector T cells in pulmonary versus cutaneous immune effector sites. *Eur J Immunol* 1994;24: 1269-77
- 39)\_ Wakita H, Sakamoto T, Tokura Y, Takigawa M. E-selectin and vascular cell adhesion molecule-1 as critical adhesion molecules for infiltration of T lymphocytes and eosinophils in atopic dermatitis. *J Cutan Pathol* 1994;21:33-9
- 40)\_ Rossiter H, Van Reijssen F, Mudde G. Skin disease-related T cells bind to endothelial selectins: expression of cutaneous lymphocyte antigen (CLA) predicts E-selectin but not P-selectin binding. *Eur J Immunol* 1994;24:205-10
- 41)\_ Bochner BS, Klunk DA, Sterbinsky SA, et al. IL-13 selectively induces vascular cell adhesion molecule-1 expression in human endothelial cells. *J Immunol* 1995; 154:799-803



- 42)\_ Bruynzeel-Koomen C, van Wichen DF, Toonstra J, et al]. The presence of IgE molecules on epidermal Langerhans cells in patients with atopic dermatitis. *Arch Derm Res* 1986;287:199-205
- 43)\_ Irani AM, Sampson HA, Schwartz LB. Mast cells in atopic dermatitis. *Allergy* 1989;44(suppl 9):31-4
- 44)\_ Singer KH, Tuck DT, Sampson HA, et al]. Epidermal keratinocytes express the adhesion molecule intercellular adhesion molecule-1 in inflammatory dermatoses. *J Invest Dermatol* 1989;92:746-50
- 45). Renz H, Jujo K, Bradley K, Domenico J, Gelfand EW, Leung I, et al. Enhanced IL-4 production and IL-4 receptor expression in atopic dermatitis and their modulation by interferon-gamma. *J Invest Dermatol* 1992; 99: 403-408.
- 46). Bock SA. Prospective appraisal of complaints of adverse reactions to foods in children during the first 3 years of life. *Pediatrics* 1987; 79: 683-8.
- 47). Sampson HA. Food allergy. Part 1: immunopathogenesis and clinical disorders. *J Allergy Clin Immunol* 1999; 103: 717-28.
- 48). Arshad SH, Stevens M, Hide DW. The effect of genetic and environmental factors on the prevalence of allergic disorders at the age of two years. *Clin Exp Allergy* 1993; 23: 504-11.
- 49). Colloff MJ. Exposure to house dust mites in homes of people with atopic dermatitis. *Br J Dermatol* 1992; 127: 322-327.
- 50). Riley G, Sebers R, Rains N, Crane J, Fitzharris P. House dust mite allergen on skin and sheets. *Lancet* 1998; 351: 649-650.
- 51). Fitzharris P, Riley G. House dust mites in atopic dermatitis. *Int J Dermatol* 1999; 38: 173-175

- 52). Leung DYM Atopic dermatitis: new insight and opportunities for therapeutic intervention. *J Allergy Clin Immunol* 2000;105:860-76
- 53). Leyden JJ, Marples RR, Kligman AM. Staphylococcus aureus in lesions of atopic dermatitis. *BrJ Dermatol* 1974;90:525-30.
- 54). Hanifin JM, Rogge JL. Staphylococcal infections in patients with atopic dermatitis. *Arch Dermatol* 1977;113:1383-6.
- 55). Tanaka M, Aiba S, Matusumura N, et al. IgE-mediated hypersensitivity and contact sensitivity to multiple environmental allergens in atopic dermatitis. *Arch Dermatol* 1994;130:1393-1401.
- 56). Braunstein B, deuell B, Platts-Mills TAE. Atopic dermatitis associated with dermatophytes infection and Trichophyton hypersensitivity. *Cutis* 1993; 51:191-2.
- 57). Hauser C, Prins C, Lacour M. The Role of Infectious Agents in Atopic Dermatitis. In: *Atopic Dermatitis: from Pathogenesis to treatment*. Ed. DYM Leung. R.G.Landescompany,Austen.1996;pp89-96.
- 58.) Rudikoff D, Lebowohl M. Atopic dermatitis. *Lancet* 1998; 351:1715–1
- 59). Hanifin JM. Atopic dermatitis in infants and children. *PediatrClin North Am* 1991;38:763–89
- 60). Abeck D, Cremer H (eds) *Häufige Hautkrankheiten im Kindesalter: Klinik – Diagnose – Therapie*. Steinkopff, Darmstadt, 2001
- 61). Kunz B, Ring J. Clinical features and diagnostic criteria of atopic dermatitis. In: Harper J, Oranje A, Prose N (eds) *Textbook of pediatric dermatology*. Blackwell Science, Oxford, 2000: 199–215
- 62). Dotterud LK, Kvammen B, Lund E, Falk ES. Prevalence and some clinical aspects of atopic dermatitis in the community of Sor-Varanger. *Acta Derm Venereol* 1995; 75:50–54

- 63). Schudel P, Wüthrich B. Klinische Verlaufsbeobachtungen bei Neurodermitis atopica nach dem Kleinkindesalter. *ZHautkrankh* 1985; 60:479–486
- 64) Finlay AY, Nicholls S, King CS, Marks R. The dry non-eczematous skin associated with atopic eczema. *Br J Dermatol* 1980; **102**: 249–56.
- 65) Uehara M. Clinical and histological features of dry skin in atopic dermatitis. *ActaDermVenereolSuppl (Stockh)* 1985; **114**: 82–6.
- 66). Rudzki E, Samochocki Z, Rebandel P, Saciuk E, Galecki W, Raezk A, et al. Frequency and significance of the major and minor features of Hanifin and Rajka among patients with atopic dermatitis. *Dermatology* 1994; 189: 41-46.
- 67). Mevorah B, Frenk E, Wietlisbach V, Carrel CF. Minor clinical features of atopic dermatitis. *Dermatologica* 1988; 177: 360–364
- 68). Diepgen TL, Fartasch M. Recent epidemiological and genetic studies in atopic dermatitis. *ActaDermVenereol* 1992; 176 (suppl):13–18
- 69). Przybilla B. Stigmata of the atopic constitution. In: Ruzicka T, Ring J, Przybilla B (eds) *Handbook of atopic eczema*. Springer, Berlin Heidelberg New York, 1991: 31–42
- 70). Whitfield A. On the white reaction in dermatology. *Br J Dermatol* 1938; 50:71–76
- 71). Ebling FJG, Marks R, Rook A. Disorders of keratinization. In: Rook A, Wilkinson DS, Ebling FJG, Champion RH, Burton JL (eds.). *Textbook of dermatology*, 4th edn. Blackwell Scientific Publications, Oxford, 1986: 1435–1436
- 72). Breit R. Neurodermatitis (Dermatitis atopica) in Kindesalter. *Z Hautkr* 1977;1:72-82.
- 73). Svensson A. *Diagnosis of Atopic Skin Disease Based on Clinical Criteria*. Lund University Press, Kristianstad. 1989; p.77.
- 74). Rudzki E, Samochocki Z, Rebandel P, et al. Frequency and significance of the

major and minor features of Hanifin and Rajka among patients with atopic dermatitis. *Dermatology* 1994;189:41-6.

75). Hunecke P, Bosse K, Finckh H. Krankheitsverlauf und psychosozialeereignisse wahrend der stationaren behandlung atopischer ekzematiker. *ZHautkr* 1990; 65: 428-434.

76). Ginsburg IH, Prytowsky JH, Kornfield DS, Wolland H. Role ofemotional factor in adults with atopic dermatitis. *Int J Dermatol*1993; 32: 656-660.

77).Soo Ick Cho, Jin Hye Kim, Bo Young Chung,In Su Ahn,Hye One Kim, Chun Wook Park, CheolHeonLee,Quality of Life in Children and Adolescents with Atopic .Dermatitis (Korean J Dermatol 2011;49(5):415-421

78).A. Wolkerstorfer, F. B. DE Waard Van der Spek, E. J. Glazenburg, P. G. H. Mulder and A. P. Oranje Scoring the Severity of Atopic Dermatitis: Three Item Severity Score as a Rough System for Daily Practice and as a Pre-screening Tool for .Studies. *ActaDermVenereol* 1999; 79: 356- 359

79). Stores G, Burrows A, Crawford C. Psychological sleep disturbance inchildren with atopic dermatitis: a case control study. *Pediatr Dermatol*1988; 15: 324-325

80). Prochazka P, Von Uslar A. Power relations in mother-child interactions in neurodermatitis constitucionalis atopica (atopic dermatitis). *Z Hutkr* 1989; 64: 863-866.

81).Hanifin JM, Rajka G. Diagnosis features of atopic dermatitis. *Acta DermVenereol (Stockh) (Suppl)* 1980; 92: 44-47.

82). Juhlin L, Johansson SGO, Bennick H *et al.* Immunoglobulin E in dermatoses. *Arch Dermatol* 1969; **100**: 12-6.

83) David TJ. Conventional allergy tests. *Arch Dis Child* 1991; **66**: 281-2.

- 84 )Pryzbilla B, Ring J. Food allergy and atopic eczema. *SeminDermatol*1990; **9**: 220–5.
- 85 )Darsow U, Ring J. Airborne and dietary allergens in atopic eczema: a comprehensive review of diagnostic tests. *ClinExpDermatol*2000; **25**: 544–51.
- 86) Patrizi A, Guerrini V, Ricci G *et al.* The natural history of sensitizations to food and aeroallergens in atopic dermatitis: a 4-year follow-up. *PediatrDermatol*2000; **17**: 261–5.
- 87) Cronin E, McFadden JP. Patients with atopic eczema do become sensitized to contact allergens. *Contact Dermatitis* 1993; **28**: 225–8.
- 88) Lever R, Forsyth A. Allergic contact dermatitis in atopic dermatitis. *ActaDermVenereolSuppl (Stockh)* 1992; **76**: 95–8.
- 89) Niggemann B, Reibel S, Wahn U. The atopy patch test (APT): a useful tool for the diagnosis of food allergy in children with atopic dermatitis. *Allergy* 2000; **55**: 281–5
- 90) Mihm MC, Soter NA, Dvorak HF *et al.* The structure of normal skin and the morphology of atopic eczema. *Dermatology* 1976; **67**: 305–12.
- 91) Prose PH. Pathological changes in eczema. *J Pediatr*1965; **66**: 178–99.
- 92) Finlay AY. Quality of life in atopic dermatitis. *J Am AcadDermatol*2001;**45** (Suppl. 1): S67–8.
- 93) Lundberg L, Johannesson M, Silverdahl M *et al.* Health related quality of life in patients with psoriasis and atopic dermatitis measured with SF-36, DLQI and a subjective measure of disease activity. *ActaDermVenereol(Stockh)* 2000; **80**: 430–4.
- 94) Lewis-Jones MS, Finlay AY. The children's dermatology life quality index (CDLQI): initial validation and practical use. *Br J Dermatol*1995; **132**: 942–9.

- 95) Lawson V, Lewis-Jones MS, Finlay AY *et al.* The family impact of childhood atopic dermatitis: the dermatitis family impact questionnaire. *Br J Dermatol* 1998; **138**: 107–13.
- 96) Reid P, Lewis-Jones MS. Sleep difficulties and their management in preschoolers with atopic eczema. *ClinExpDermatol*1995; **20**: 38–41.
- 97) Lawson V, Lewis-Jones MS, Reid P *et al.* Family impact of childhood eczema. *Br J Dermatol* 1995; **133** (Suppl. 45): 19.
- 98).V.E.V. Rullo, A. Segato, A. Kirsh and D. Sole. Severity scoring of atopic dermatitis: a comparison of two scoring systems. *Allergol et Immunopathol* .2008;36(4):205-11
- 99).Rosalinda W.C. van Val burg, Marjolein G. Will emsen1, Pauline C. Dirven - Meijer, Arnold P. Oranje, Johannes C. van der Wouden and Heleen Moed. Quality of Life Measurement and its Relationship to Disease Severity in Children with Atopic Dermatitis in General Practice. *ActaDermato-Venereologica* 2011 Epub ahead of .print.1-5
- 100). Warschburger P, Buchholz HT, Petermann F. Psychological adjustment in parents of young children with atopic dermatitis: which factors predict parental .quality of life? *Br J Dermatol* 2004; 150: 304–11
- 101). Torsten Z, Seth J, Orlow MD *et al.* Patient perspectives on the management of .(atopic dermatitis. *J Allergy ClinImmunol* (in press
- 102). Koblenzer CS, Koblenzer PJ. Chronic intractable atopic eczema. Its occurrence as a physical sign of impaired parent– child relationships and psychologic developmental arrest: improvement through parent insight and education. *Arch .Dermatol* 1988; 124: 1673–7
- 103). Absolon CM, Cottrell D, Eldridge SM, Glover MT. Psychological disturbance in atopic eczema: the extent of the problem in school-aged children. *Br J Dermatol* 1997; .137: 241–5

- 104) Titman PS, Barker C, Smith CH. The psychological impact of chronic eczema on children and their families. *Br J Dermatol* 2001; 145(S59): 128–9
- 105) S. LEWIS-JONES. Quality of life and childhood atopic dermatitis: the misery of living with childhood eczema *Int J Clin Pract*, August 2006, 60, 8, 984–992
- 106) Agostoni C, Grandi F, Scaglioni S *et al.* Growth pattern of breastfed and nonbreastfed infants with atopic dermatitis in the first year of life. *Pediatrics* 2000; **106**: E73.
- 107) Bode HH. Dwarfism following long-term corticosteroid therapy. *JAMA* 1980; **244**: 813–4.
- 108) Williams JV, Vowels B, Honig P, Leyden JJ. *Staphylococcus aureus* isolation from the lesions, the hands, and anterior nares of patients with atopic dermatitis. *J Emerg Med* 1999; **17**: 207–11.
- 109) Rystedt I, Stranegard IL, Stranegard O. Recurrent viral infections in patients with past or present atopic dermatitis. *Br J Dermatol* 1986; **114**: 575–82.
- 110) Sehgal VN, Jain S. Atopic dermatitis: ocular changes. *Int J Dermatol* 1994; **33**: 11–4.
- 111) Uehara MAD. Infra-orbital fold in atopic dermatitis. *Arch Dermatol* 1981; **117**: 627.
- 112) Williams HC, Pembroke AC. Infraorbital crease, ethnic group, and atopic dermatitis. *Arch Dermatol* 1996; **132**: 51–4.
- 113) Karel I, Myska V, Koicalova E. Ophthalmological changes in atopic dermatitis. *Acta Derm Venereol (Stockh)* 1965; **45**: 381–6.
- 114) Brunsting LA, Reed WB, Bair HL. Occurrence of cataracts and keratoconus with atopic dermatitis. *Arch Dermatol* 1955; **72**: 237–41.
- 115) Copeman PWM. Eczema and keratoconus. *BMJ* 1966; **ii**: 977–9

- 116) Yates VM, Kerr REI, MacKie RM. Early diagnosis of infantile seborrhoeic dermatitis and atopic dermatitis, 1: clinical features; 'total' and specific IgE levels. *Br J Dermatol* 1983; **108**: 633–9.
- 117) Bieber T: Mechanisms of disease: atopic dermatitis. *N Engl J Med* 2008, 358:1483-1494
- 118). Krakowski AC, Eichenfield LF, Dohil MA: Management of atopic dermatitis in the pediatric population. *Pediatrics* 2008, 122:812-824.
- 119). Akdis CA, Akdis M, Bieber T, et al, Diagnosis and treatment of atopic dermatitis in children and adults: *J Allergy Clin Immunol* 2006, 118:152-169.
- 120). Leung DY, Boguniewicz M, Howell MD, Nomura I, Hamid QA: New insights into atopic dermatitis. *J Clin Invest* 2004, 113:651-657.
- 121). Krakowski AC, Dohil MA: Topical therapy in pediatric atopic dermatitis. *Semin Cutan Med Surg* 2008, 27:161-167
- 122). Hanifin JM. Breaking the cycle. How I manage difficult cases of atopic dermatitis. *Fitzpatrick's J Clin Dermatol* 1994; 1: 13-26.
- 123).. Giménez Camarasa JM. Tratamiento de la dermatitis atópica. *Monogr Dermatol* 1992; 5: 448-454
- 124) Holden C, English J, Hoare C *et al*. Advised best practice for the use of emollients in eczema and other dry skin conditions. *J Dermatol Treat* 2002; **13**: 103–6.
- 125). Buske-Kirschbaum A, Geiben A, Hellhammer D. Psychobiological aspects of atopic dermatitis: an overview. *Psychother Psychosom* 2001; **70**: 6–16.
- 126) Linnet J, Jemec GB. The assessment of anxiety and dermatological life quality in patients with atopic dermatitis. *Br J Dermatol* 1999; **140**: 268–72.



- 127) Staughton R. Psychologic approach to atopic skin disease. *J Am Acad Dermatol* 2001;**45** (Suppl. 1): S53–4.
- 128) Lever R. The role of food in atopic eczema. *J Am Acad Dermatol* 2001;**45** (Suppl. 1): S57–60.
- 129) Sampson HA, McCaskill CC. Food hypersensitivity and atopic dermatitis: evaluation of 113 patients. *J Pediatr* 1985; **107**: 669–75.
- 130) Atherton DJ, Sewell M, Soothill JF *et al.* A double-blind controlled crossover trial of an antigenic avoidance diet in atopic eczema. *Lancet* 1978; **i**: 401–3.
- 131) Lever R, MacDonald C, Waugh P, Aitchison T. Randomised controlled trial of advice on an egg exclusion diet in young children with atopic eczema and sensitivity to eggs. *Pediatr Allergy Immunol* 1998; **9**: 13–9.
- 132) Sampson H, Ho DG. Relationship between food-specific IgE concentrations and the risk of positive food challenges in children and adolescents. *J Allergy Clin Immunol* 1997; **100**: 444–51.
- 133). Lane A. Efficacy and safety of topical steroids in paediatric atopic dermatitis. *J Eur Acad Dermatol Venereol* 1997; **88** (suppl. 1): S24-S27.
- 134) Long CC, Finlay AY. The finger tip unit: a new practical measure. *Clin Exp Dermatol* 1991; **16**: 444–7.
- 135) Cubey RB. Glaucoma following the application of corticosteroids to the skin of the eyelids. *Br J Dermatol* 1976; **95**: 207–8.
- 136). Drake LA, Fallon JD, Sober A. Relief of pruritus in patients with atopic dermatitis after treatment with topical doxepin cream. The Doxepin Study Group. *J Am Acad Dermatol* 1994; **31**: 613-616.

- 137). Drake LA, Millikan LE. The antipruritic effect of 5% doxepin cream in patients with eczematous dermatitis The Doxepin Study Group. *Arch Dermatol* 1995; 131: 1403-1408.
- 138). Hoare C, Li Wan Po A, Williams H. Systematic review of treatments for atopic eczema. *Health Technol Assess* 2000;4:1-191.
- 139). McHenry PM, Williams HC, Bingham EA. Management of atopic eczema: Joint Workshop of the British Association of Dermatologists and the Research Unit of the Royal College of Physicians of London. *BMJ* 1995;310:843-7.
- 140). Hanifin JM, Cooper KD, Ho VC, et al. Guidelines of care for atopic dermatitis, developed in accordance with the American Academy of Dermatology (AAD)/American Academy of Dermatology Association "Administrative Regulations for Evidence- Based Clinical Practice Guidelines." *J Am Acad Dermatol* 2004;50:391-404.
- 141). Wasserbauer N, Ballow M: Atopic dermatitis. *Am J Med* 2009, 122:121-125
- 142). Doherty V, Sylvester D, Kennedy C, et al. Treatment of itching in atopic eczema with antihistamines with low sedation profile. *BMJ* 1989; 298: 96.
- 143). Stevens SR, Hanifin JM, Hamilton T, et al. Long-term effectiveness and safety of recombinant human interferon gamma therapy for atopic dermatitis despite unchanged serum IgE levels. *Arch Dermatol* 1998; 134: 613-616.
- 144). Carucci JA, Washenik K, Weinstein A, Shupack J, Cohen DE. The leukotriene antagonist zafirlukast as a therapeutic agent for atopic dermatitis. *Arch Dermatol* 1998; 134: 785-786.
- 145) Berth-Jones J, Finlay AY, Zaki I, Tan B, Goodyear H, Lewis-Jones S, et al. Cyclosporine in severe childhood atopic dermatitis: a multicenter study. *J Am Acad Dermatol* 1996; 34: 1016-1021.

146) Krutmann J. Phototherapy for atopic dermatitis. *ClinExpDermatol*2000; **25**: 552–8.

147) Reynolds NJ, Franklin V, Gray JC *et al.* Narrow-band ultraviolet B and broad-band ultraviolet A phototherapy in adult atopic eczema: a randomized controlled trial. *Lancet* 2001; **357**: 2012–6

148)Tzaneva S, Seeber A, Schwaiger M *et al.* High-dose versus medium-dose UVA1 phototherapy for patients with severe generalized atopic dermatitis. *J Am AcadDermatol*2001;**45**: 503–7.

149) Der-Petrossian M, Seeber A, Honigsmann H, Tanew A. Half-side comparison study on the efficacy of 8-methoxypsoralen bath-PUVA versus narrowband ultraviolet B phototherapy in patients with severe chronic atopic dermatitis. *Br J Dermatol*2000; **142**: 39–43.

150) George S, Bilsand D, Johnson BE *et al.* Narrow-band UVB (TL-01) air-conditioned therapy for chronic severe adult atopic eczema. *ActaDermVenereolSuppl (Stockh)* 1992; **176**: 137–8.

151) Kay AB. Allergen injection immunotherapy (hyposensitisation) on trial. *ClinExp Allergy* 1989; **119**: 591–6.

152)-. Alvarenga T.M.M, Caldeira.A.P, Quality of life in pediatric patients with atopic dermatitis.*JPediatr (Rio J)*. 2009;**85**(5):415-420.

153).Lewis-Jones MS, Finlay AY. Wales College of Medicine, Quality of .liferesearch. <http://www.dermatology.org.uk>. Access:10/04/2006

154) Lewis-Jones MS, Finlay AY, Dykes PJ. The infant's DermatitisQuality of Life Index. *Br J Dermatol*. 2001;**144**:104-10.

155) Hanifin JM, Thurston M, Omoto M, Cherill R, Tofte SJ, GraeberM.The eczema area and severity index (EASI) assessment of reliabilityin atopic dermatitis. *ExpDermatol*. 2001;**10**:11-8.



## APPENDEX-IV EASI score

An EASI score is a tool used to measure the severity and extent of atopic eczema (**E**czema **A**rea and **S**everity **I**ndex). It takes a few minutes and experience to calculate it accurately.

### Body regions

There are four body regions:

1. Head and neck
2. Upper limbs
3. Trunk
4. Lower limbs

The intensity of a representative area of eczema and the approximate percentage affected by eczema are calculated for each region.

### Intensity

A representative area of eczema is selected for each body region. The intensity of redness (erythema), thickness (induration, papulation, oedema), scratching (excoriation) and lichenification (lined skin) of the eczema is assessed as none (0), mild (1), moderate (2) and severe (3). Half scores are allowed.

#### Eczema: severity scoring

Intensity	Absent	Mild	Moderate	Severe
Redness				
	Score 0	Score 1	Score 2	Score 3
Thickness				
	Score 0	Score 1	Score 2	Score 3



### **Calculation for intensity**

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The four intensity scores are added up for each of the four body regions to give subtotals A1, A2, A3, A4.

Each subtotal is multiplied by the body surface area represented by that region.

- $A1 \times 0.1$  gives B1 (in children 0-7 years,  $A1 \times 0.2$  gives B1)
- $A2 \times 0.2$  gives B2
- $A3 \times 0.3$  gives B3
- $A4 \times 0.4$  gives B4 (in children 0-7 years,  $A1 \times 0.3$  gives B1)

### **Area**

---

The percentage area affected by eczema is evaluated in the four regions of the body. In each region, the area is expressed as nil (0), 1-9% (1), 10-29% (2), 30-49% (3), 50-69% (4), 70-89% (5) or 90-100% (6).

- Head and neck
- Upper limbs
- Trunk
- Lower limbs

### **Calculations for area**

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Each of the body area scores is multiplied by the area affected.

- $B1 \times (0 \text{ to } 6) = C1$

- $B2 \times (0 \text{ to } 6) = C2$
- $B3 \times (0 \text{ to } 6) = C3$
- $B4 \times (0 \text{ to } 6) = C4$

## Total score

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The EASI score is  $C1 + C2 + C3 + C4$ .

## Example

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A 3-year-old child has an acute flare-up of atopic eczema. The flare-up affects limb flexures, and the child's trunk is also rather pink and dry.

- The head and neck intensity score is nil, as it is unaffected:  $A1$  is 0.
- The eczema in the elbow flexure is moderately red, mildly thickened, moderately scratched but not lichenified:  $A2$  is  $2 + 1 + 2 = 5$
- On the trunk it is mildly red, mildly thickened and not scratched or lichenified:  $A3$  is  $1 + 1 + 0 + 0 = 2$ .
- The eczema behind the knees is severely red, severely thickened, severely scratched and mildly lichenified:  $A4$  is  $3 + 3 + 3 + 1 = 10$

The intensity scores are then adjusted as follows:

- $B1 = A1 \times 0.2 = 0$
- $B2 = A2 \times 0.2 = 1.0$
- $B3 = A3 \times 0.3 = 0.6$
- $B4 = A4 \times 0.4 = 4.0$

Less than 10% of the arms is affected, around 60% of the trunk and somewhere between 10 and 29% of the lower limbs.

- $C1 = B1 \times 0 = 0.0$
- $C2 = B2 \times 1 = 1.0$
- $C3 = B3 \times 4 = 2.4$
- $C4 = B4 \times 2 = 8.0$

$EASI = C1 + C2 + C3 + C4 = 11.4$

## Related information

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### References:

- Hanifin JM, Thurston M, Omoto M, Cherill R, Tofte SJ, Graeber M. The eczema area and severity index (EASI): assessment of reliability in atopic dermatitis. EASI Evaluator Group. *Exp Dermatol*. 2001 Feb;10(1):11-8.

### On DermNet NZ:

- [SCORAD](#)
- [Atopic eczema](#)

**Other websites:**

**Books about skin diseases:**



Name:  
Address:

Date:

IDQOL  
SCORE

The aim of this chart is to record how your child's dermatitis has been. Each question concerns THE LAST WEEK ONLY. Please could you answer every question.

### Dermatitis Severity

Over the last week, **how severe** do you think your child's dermatitis has been?; i.e. how red, scaly, inflamed or widespread.

Extremely severe  
Severe  
Average  
Fairly good  
None

### Life Quality Index

1. Over the last week, how much has your child been **itching and scratching**?  
All the time  
A lot  
A little  
None
2. Over the last week, what has your child's **mood** been?  
Always crying, extremely difficult  
Very fretful  
Slightly fretful  
Happy
3. Over the last week approximately how much **time** on average has it taken to **get your child off to sleep** each night?  
More than 2 hrs  
1 - 2 hrs  
15mins - 1 hr  
0-15mins
4. Over the last week, what was the **total time** that your child's **sleep was disturbed** on average each night?  
5 hrs or more  
3 - 4 hrs  
1 - 2 hrs  
Less than 1 hour
5. Over the last week, has your child's eczema interfered with **playing or swimming**?  
Very much  
A lot  
A little  
Not at all
6. Over the last week, has your child's eczema interfered with your child **taking part in or enjoying other family activities**?  
Very much  
A lot  
A little  
Not at all
7. Over the last week, have there been problems with your child at **mealtimes** because of the eczema?  
Very much  
A lot  
A little  
None
8. Over the last week, have there been problems with your child caused by the **treatment**?  
Very much  
A lot  
A little  
None
9. Over the last week, has your child's eczema meant that **dressing and undressing** the child has been **uncomfortable**?  
Very much  
A lot  
A little  
None
10. Over the last week how much has your child having eczema been a problem at **bathtime**?  
Very much  
A lot  
A little  
None

Please can you check that you have answered every question.

## نوعية الحياة في الأطفال و الرضع المصابين بالاكزيما التآيبيية في بنغازي

التهاب الجلد التآيبي (م) - المعروف أيضا باسم الأكزيما الاستشرائية هو الانتكاس مزمنة، حاكة للغاية، والتهاب في الجلد الجاف مرض يصيب 2-5% من عامة الناس. م لديها أكبر تأثير على الأطفال والرضع ، مما يؤثر على ما يقدر ب 10-20% أو أكثر ، ولكن يعتقد تأثيرها في 3% من البالغين

يتميز الطفح حكة حطاطات (حويصلات أحيانا في الأطفال الرضع)، والتي أصبحت متسحجة ومتحززة، وعادة ما يكون توزيع العاطفة. وكثيرا ما يرتبط مع ظروف اندلاع التآيبي الأخرى في فرد أو أفراد الأسرة الآخرين

غالبا ما يبدأ في مرحلة الطفولة التهاب الجلد التآيبي أو في مرحلة الطفولة المبكرة، وهي الفترة حرجللتسمية النفسية والاجتماعية.

وقد تم توثيق كل من التأثير الفسيولوجية والنفسية في الأطفال الصغار مع أسرهم

التهاب الجلد التآيبي غالبا ما يبدأ في سن قبل 5 و قد تستمر في بالنسبة للبعض، فهي لا تتطور بشكل دوري ومن ثم تهدأ لبعض الوقت، حتى تصل إلى عدة سنوات حتى الآن، ويقدر أن 75% من حالات التهاب الجلد التآيبي تحسین من قبل الأطفال مرة وصول، في حين يستمر 25% لديهم صعوبات مع حالة خلال مرحلة البلوغ وكانت هذه الدراسة لتحديد نوعية الحياة في التهاب الجلد التآيبي في الرضع والأطفال وعلاقة مع شدة المرض.

سبعة وثمانون المريض حضور عيادة الاكزيما في مستشفى الجمهوريه بنغازي ليبيا الفترة الممتدة من نوفمبر 2010 إلى st30 يوليو 2013 مع تشخيص المرض، كل مريض سيخضع لتاريخ مفصل، الفحص السريري، الأم والطفل طلب QOL السائل ((IDQOL - CDQOL)، 10 السائل يكون كل سؤال (0-3) درجات والدرجة القصوى في مسألة (30)، EASI النتيجة هي مؤشر السريرية لشدة من الأكزيما يعتمد على الفحص السريري

النتائج: التحق ما مجموعه 87 المرضى في هذه الدراسة 41.4% من الذكور و 58.6% الانثوي، كانت النتيجة من (26-8) IDQOL درجة عالية لالحكة والسلوك ، بينما في (28-6) CDQOL درجة عالية للحكة وسلوك الآخرين، ستة في المئة من المرضى الذين لديهم تاريخ من أمراض الحساسية الأخرى، يعانون من مرض الربو القصيبي والتهاب الأنف التحسسي معا و 8% لديهم حساسية حليب البقر. تاريخ عائلي من أمراض الحساسية وسجلت في 79.3%، 25.3% كان التاريخ من الربو القصيبي، وكان 26.4% الربو القصيبي والتهاب الأنف التحسسي معا.

بالنسبة للعمر  $\geq 3$  سنوات كان 41.4% من المرضى تأثير كبير للغاية، وكان 46% تأثير كبير جدا. كان يعني CDLQI النتيجة  $\leq 4.793 \pm 17.86$  ، وبالنسبة للمرضى  $< IDLQI3$  كان تأثير كبير جدا في 51.4% و 40.5% تأثير كبير المدقع، مع متوسط درجة تساوي  $4.79 \pm 17.6$

النتيجة من مجموعة 26-8 IDQOL، بينما في CDLQI نطاق 6-28

لل  $\geq 3$  years الرضع، ومتوسط من 10 أسئلة حول شدة تراوحت بين  $(0.86 \pm 1.4)$  لهذه المشكلة من العلاج إلى  $(0.62 \pm 2.45)$  للالحكة والهرش. كان المتوسط الكلي للأسئلة 10  $(4.8 \pm 17.9)$ . بالنسبة

للمرضى <YEARS3، يعني تراوحت النتيجة بين (0.79 ± 0.84) لهذه المشكلة مع العلاج ل(2.4 ± 0.59) للالحكة والهرش، فيما يبلغ متوسط درجة (4.82 ± 17.5).

أظهرت الدراسات السريرية لشدة EASI أن غالبية المرضى كان 74.7% المعتدلة وحادة 6.9%.

الاستنتاجات:

من هذه الدراسة الإناث أكثر تأثرا من الذكور، والغالبية العظمى من المرضى كان لديه تاريخ عائلي من أمراض الحساسية و87.4% تأثير كبير وقطع تأثير كبير، من هذه النتيجة، فإننا نستنتج أن مرض الاكزيما التائبية عند الرضع والأطفال له تأثير سلبي على نوعية الحياة للأطفال المرضى وأسراهم