PREVALENCE OF ATOPIC DERMATITIS AMONG UNDER FIVES ATTENDING PUBLIC HOSPITALS IN DAR ES SALAAM, TANZANIA, 2008/09

By

Delila H Moshi (MD)

A Dissertation Submitted in Partial Fulfillment of the Requirements for the Degree of

Master of Medicine in Paediatrics and Child Health of the

Muhimbili University of Health and Allied Sciences

November, 2009

CERTIFICATION

The undersigned certify that he has read and hereby recommends for the examination of a dissertation entitled *"The prevalence of atopic dermatitis among children under five years in Dar es Salaam public hospitals, Tanzania 2008"* in partial fulfillment of the requirements for the degree of Master of Medicine (Paediatrics and Child Health) of the Muhimbili University of Health and Allied Sciences.

Dr Yassin Mgonda

(Supervisor)

Date.....30/11/2009.....

DECLARATION AND COPYRIGHT

I, **Delila H. Moshi,** declare that this dissertation is my own original work and that it has not been presented and will not be presented to any other University for a similar or any other degree award.

Signature: _____

This dissertation is copyright material protected under the Berne Convention, the Copyright Act 1999 and other international and national enactments, in that behalf, on intellectual property. It may not be reproduced by any means, in full or part except for short extracts in fair dealings, for research or private study, critical scholarly review or discourse with an acknowledgement, without permission of the Directorate of Postgraduate Studies, on behalf of both the author and the Muhimbili University of Health and Allied Sciences.

ACKNOWLEDGEMENT

I wish to express my sincere gratitude to all those who in one way or another made the production of this dissertation possible.

First and foremost I would like to thank my Principal supervisor Dr. Yassin Mgonda for his guidance, patience and for taking much of his valuable time so as to supervise towards this dissertation.

Dr. Maulid Fataki the Head, Department of Paediatrics and Child Health (MUHAS) for encouraging me to go ahead with this dissertation.

A special mention goes to all the staff members of the Health facilities who devoted their time during the data collection period.

My sincere thanks also go to Mrs. Mpembeni of the Department of Biostatistics for her statistical assistance.

My gratitude should also go to all caregivers of the children that made this study possible

With most love, I thank My Mother and Brother Dalton for all the support they have given me.

Special thanks go to The Almighty God who has given me the strength and guided me through out this work, Amen.

DEDICATION

I dedicate this work to my family for all their prayers and support.

To My dearest mother,

To my Brother Dalton,

My niece Danielle

ABSTRACT

Background: Atopic dermatitis (AD), also known as atopic *eczema* is a very common skin disease. Its prevalence is thought to range from 5% to 20% worldwide. The exact cause is not known, but AD results from a combination of family heredity and a variety of conditions in everyday lives that trigger the itchy rash. It can interfere with social function and sleep. Its persistence and the accompanying pruritus may be stressful and frustrating for patients.

Objective: To determine the prevalence of atopic dermatitis and associated co-morbidities among children under the age of 5years attending public hospitals in Dar-es-Salaam.

Methodology: A cross-sectional descriptive study was carried out between June 2008 and January 2009 in children less than five years attending RCH clinics in Dar es Salaam municipal hospitals. Simple random technique was used to recruit participants in the study. Data was collected using a standardized questionnaire and analysis done using EPI Info version 6 and SPSS version 13.

Results: Of the 301 children enrolled in the study, 39 (13%) had atopic dermatitis. Of the 39 children, the majority 27 (69.2%) were below 1 year and most of them 15 (38.5%) were below 6 months of age. Children with associated co-morbidities were 19 (48.7%) and most of them were males 10/19 (52.6%). Allergic rhinitis was found in 10 (52.6%) children being the most frequent co-morbidity disorder. Out of the 39 children with atopic dermatitis, 9 (53%) had a positive family history of atopic dermatitis.

Conclusion and recommendation: The prevalence of AD was 13%. The same applied to the age of onset, clinical manifestation and associated co-morbidities. This study was conducted in public hospitals only, other children attending dispensaries and private hospitals were missed. A community based study is therefore recommended so as to determine the true prevalence of atopic dermatitis in Tanzania.

LIST OF TABLES

Table 1: Characteristics of study population..... 16

Table 2: Age at first onset of AD..... 17

Table 3: Distribution of co-morbidities of AD according to age..... 17

Table 4: Distribution of co-morbidities according to sex..... 18

Table 5: Distribution of positive family history of atopy in children with AD according to age..... 18

Table 6: Distribution of children with AD and a positive history of AD and the associated co-morbidities..... 19

Figure 1-6..... 38

Figure 7-8..... 39

ABBREVIATION

cAMP- cyclic adenosine monophosphate phosphodiesterase

TH_2- T 2 helper cells

AD- atopic dermatitis

IgE- immunoglobulin E

ISAAC- International study of asthma and allergies in childhood

IL- interleukine

GM-CSF- granulocyte macrophage colony stimulating factor

PGE- proglandin E

RAST- radioallergosorbent test

RCH – reproductive and child health

NFAT- nuclear factor of activated T cells

TABLE OF CONTENTS

ITEM	PAGE
Title	
Certification.....	i
Declaration and copyright.....	ii
Acknowledgement.....	iii
Dedication.....	iv
Abstract.....	v
Table of tables.....	vi
Abbreviation	vii
1. Introduction and literature review.....	1
1.1 Pathogenesis and etiology.....	2
1.2 Trigger factors.....	3
1.3 Clinical presentation.....	3
1.3.1 Age of onset.....	3
1.3.2 Family history.....	4
1.3.3 Clinical features.....	4
1.3.4 Differential diagnosis.....	6
1.4 Treatment.....	6
1.4.1 Bathing and moisturizers.....	7
1.4.2 Silk clothes.....	7
1.4.3 Antihistamine.....	7
1.4.4 Antibiotics.....	7
1.4.5 Topical corticosteroids.....	7
1.4.6 Phototherapy.....	8
1.4.7 Immunomodulators and Antineoplastics.....	8
1.5 Atopic dermatitis and other atopic disorders.....	9
2.0 Hypothesis.....	10
3.0 Problem statement.....	10

4.0 Rationale of the study 10
5.0 Objectives..... 10
5.1 Broad objectives..... 10
5.2 Specific objectives..... 11
6.0 Methodology..... 11
6.1 Study design..... 11
6.2 Study area..... 11
6.3 Study population 12
6.4 Inclusion and exclusion criteria..... 12
6.4.1 Inclusion criteria..... 12
6.4.2 Exclusion criteria..... 12
6.5 Sample size estimation.....12
6.6 Sampling technique/method..... 12
6.7 Data collection..... 13
7.0 Ethical clearance and issues..... 14
8.0 Data analysis and management 14
9.0 Results..... 15
10.0 Discussion 19
10.1 Limitation of the study..... 22
11. Conclusion and recommendation..... 23
12. References..... 24
13. Appendix I
Questionnaire: English version..... 29
Swahili version..... 30
14. Appendix II
Consent form: English version..... 31
Consent form: Swahili version..... 33
Figures 1-6..... 35
Figures7-8..... 36

1.0 INTRODUCTION AND LITERATURE REVIEW

Atopic dermatitis (AD), also known as atopic *eczema* is part of a syndrome called atopy which means "not in the right place". Atopic dermatitis is a chronically relapsing skin disease that occurs most commonly during early infancy and childhood. The exact cause is not known, but it results from a combination of heredity and a variety of conditions in everyday lives that trigger the red, itchy rash which is an interaction between genetic predisposition and the environment. It can interfere with social function and sleep. Its persistence and the accompanying pruritus may be stressful and frustrating to some patients. An attempt to relieve the itch by scratching simply worsens the rash, creating a vicious circle. In infants it affects the extensor surfaces of extremities, face, trunk and neck. Later it presents as symmetric lesions on the ankles, feet, flexor areas of the extremities, cheeks, forehead and the scalp. It is a worldwide distributed disease and is associated with environmental factors (allergens)

Atopic dermatitis is one of the diseases in which it is difficult to establish its prevalence due to several factors including small sample size, lack of control groups in most studies, lack of clearly defined inclusion criteria. In addition, difficulties arise in interpreting, combining and directly comparing findings across studies because of the diverse ways in which the disease has been defined, differences in the age ranges of the children studied and variations in the study methods used.[1] Atopic dermatitis usually affects babies and young children, and may last until they are fully grown up. There is a controversy in the prevalence of atopic dermatitis done by different researchers either in the same country or worldwide.

Studies done in developing countries show that there has been an increase in the prevalence of atopic dermatitis over the last five decades. Studies done in developed countries show that 10% of all children get atopic dermatitis sometime in their life.[2] Atopic dermatitis affects approximately five to 20% of children worldwide.[3]The prevalence of atopic dermatitis in the United States is around 17%. [3]

Several studies done in African countries showed the prevalence of atopic dermatitis ranging from 3% to 20%, with severe atopic dermatitis being below 3%. [4] Approximately 60% of all cases of atopic dermatitis are diagnosed within the first year of life and 90% of

them are diagnosed by the age of five. About 10% of all children with AD are diagnosed over the age of five and it is rare for the condition to continue unidentified before a patient reaches his or her teens.[5]

Atopic dermatitis occurs anywhere in the world and in any race [6], but other studies report that AD is most common in industrialized countries and among Caucasians and Asians[7].

1.1. Pathogenesis and Etiology

Although the pathogenesis of atopic dermatitis is unknown, the disease seems to be a result of genetic susceptibility, immune dysfunction and epidermal barrier dysfunction.[8] Current theories suggest that there is an unidentified genetic abnormality that causes increased levels of cyclic adenosine monophosphate phosphodiesterase (cAMP), which in turn leads to decreased levels of intracellular cAMP; a secondary messenger controlling cell activity. This change in the level of intracellular cAMP causes basophils and mast cells to be hyperactive, with the end result being increased histamine and leukotriene production and release. [9, 10, 11]

Researchers believe that IgE- mediated reaction and cellular responses may contribute to the chronic inflammatory nature of this disorder.[12] IgE has a multifunctional role in the pathogenesis of allergic inflammation. Apart from its involvement in IgE-mediated degranulation of mast cells and basophils, it is also involved in the activation of macrophage/monocytes and the stimulation of TH_2 cells. Recent data also suggest that the pattern of cytokine expression in AD depends on the time of onset or duration of the skin lesion. The acute onset of skin inflammation in AD is associated with a predominance of T lymphocytes and IL-4 gene expression. In chronic AD, macrophage and eosinophil activation dominate. These effector cells over expresses IL-5, IL-10, GM-CSF, and PGE2, all of which may contribute to the persistence of this disease. Both acute and chronic involve local infiltration of IL-4--and IL-5--secreting TH2-like cells, and show pathologic evidence of epithelial damage, which likely serves to amplify tissue inflammation. [13]

Besides the humoral immunity, cell mediated immune dysfunction also participate in atopic dermatitis. There is significant reduction in erythrocyte rosette formation, reduced

response to T lymphocytes to mitogens, reduced in vitro stimulation of lymphocytes to bacterial and mycotic antigens and reduced tendency to contact sensitization.[12]

Atopic dermatitis could also be a result of environmental factors leading to the immediate type of hypersensitivity demonstrated by urticarial reaction to allergens.

1.2 Trigger factors

Food which may cause irritation to the skin includes cow's milk, eggs, peanuts, wheat, soya and sea foods. The exact roles are controversial because of the limitation of in vitro RAST and prick test. [19]

Clothing which can cause irritation to the child's skin include wool, rough or tight clothes.

Animal dandruff: dogs, cats, mice shed skin cells from their skin, fur and feathers which are irritants to sensitive children. Dust mites found in homes also trigger AD. Dry skin, extreme temperature and infections may also trigger AD.[13] However; dry skin may also be a result of AD.

Infections e.g. viral infection (especially the upper respiratory tract), *Staphylococcus aureus, Candida, dermatophyte* do trigger atopic dermatitis in susceptible children.[15]

Pollen and molds during the afternoon and in temperate countries are usually highly irritating to the impaired immune system of these children. Aeroallergens are trigger factors which may play a role in the pathogenesis and exacerbation of atopic dermatitis. However, the exact roles of aeroallergens and food allergy are controversial because of limitations of the in vitro radioallergosorbent test (RAST) and prick skin test.[16]

1.2 CLINICAL PRESENTATION

The diagnosis of AD in children is based on age of onset, location of lesions, and positive family history of AD and use of major and minor clinical features.

1.2.1 Age of onset

The age of onset varies in different studies in different countries. It can start as early as six weeks to 12 weeks of age.[17]Forty five per cent of all patients with atopic dermatitis, symptoms begin within the first 6 months of life, 60% start within the first year, and 85% begin before five years of age. More than 50% of children who are affected in the first two

years of life do not have any sign of IgE sensitization. However sensitization occurs during the course of atopic dermatitis. Up to 70% of these children have a spontaneous remission before adolescence. [18]

1.2.2 Family history

The diagnosis of AD in children is more likely if a family member or close relatives have AD, asthma or hay fever. [19]A genetic basis of this disease is also supported by the finding of a 75–85% concordance in monozygotic twins and as expected a lower 30% in dizygotic twins.[20]

1.2.3 Clinical features

At first atopic dermatitis is located at the extensor surfaces of extremities, face, trunk and neck. As the patients age increases, the disease then shows predilection for the flexural aspects of the antecubital and popliteal fossa.[14]

In infants, atopic dermatitis often appears as symmetric lesions on the ankles, feet, and flexor areas of the extremities, cheeks, forehead and the scalp. Perioral pallor makes the erythema prominent. The diaper region and the nose are spared. The skin shows an increase in skin lines and creases, and the palms have palmar hyper linearity.

In young children, atopic dermatitis is manifested by generalized dryness of the skin (xerosis generalizata)

Eczematous and exudative lesions are present which involves flexural creases of the extremities. Feet and hands are involved so is the face. There may also be toe involvement and nail changes that are easily misdiagnosed as tinea pedis and onychomycosis. In these patients, one may often see striking infraorbital folds and allergic shiners.

In adolescents it is more diffuse with an erythematous base. The face is frequently involved as are the flexural areas of the arms and legs. Generalized skin dryness is often

prominent with accentuated skin lines and nail involvement. Ichthyosis vulgaris is present in over 30% of the patients with hyper linear palmar creases and polygonal dry skin scales, especially on the legs. Hand and foot dermatitis may be the only manifestations of AD in this age group.[5]

The clinical diagnosis of AD requires the presence of at least three major features and at least three minor features.

The major clinical features of AD include pruritis, chronic or relapsing dermatitis, personal or family history of AD and typical distribution and morphology of the AD rash.[21] In infants the AD rash occurs on the face and extensor surfaces; however in older children and adults flexure lichenification is common.[21]

The minor clinical features of AD are in the eyes (cataracts usually in the anterior sub capsular region, Keratoconus and the infraorbital folds), facial pallor, palmar hyper linearity, xerosis and Pityriasis. Other features are white dermatographism, Ichthyosis, Keratosis pilaris, non specific dermatitis of the hands and feet, nipple eczema and positive type I hypersensitivity skin tests. Propensity for cutaneous infection, elevated serum IgE level, food intolerance, impaired cell-mediated immunity, Erythroderma and early age of onset are also minor features of AD.[21]

The most common diagnostic features are pruritis, chronic or relapsing dermatitis, and family history of AD, the typical distribution of the rash and age of onset. [14]

Most of the time pruritis can be very severe causing sleep disruption, irritability and stress. The pruritis can lead to scratching that may result in secondary skin changes like thickening of skin lines (lichenification). In infants and young children pruritis is most common on the scalp, cheeks chin and extensor surface of extremities while in older children and adults, pruritis is on the flexure surfaces.

1.2.4 Differential Diagnosis of Atopic Dermatitis

There are other skin conditions that mimic AD. These are neurodermatitis, psoriasis, seborrhoeic dermatitis, contact dermatitis, scabies and skin manifestation of immunodeficiency disorders.[19]

Neurodermatitis is characterized by scaly patches of the skin on the head lower limbs wrists or forearms caused by localized itch like an insect bite. It is rare in under fives with a psychogenic component.

Psoriasis is an autoimmune disorder affecting the skin and joints with areas of inflammation and excessive skin production with silvery-white appearance found on extensor surfaces of elbows and knees. Usually it occurs between 15 and 25 years old for the first time.

Seborrhoeic dermatitis is a yellowish greasy and scaly lesion affecting the scalp and face. If an individual is stressed, fatigue or there's change of season or infected with yeast, other parts of the body may be affected. It differs from AD in that the lesions are yellow and greasy compared to AD where the lesions are dry, scaly and itchy.

Contact dermatitis is a localized skin reaction that includes redness, itching and burning where the skin has become in contact with an allergen or irritant.

Scabies is an infectious skin disease characterized by superficial burrows and intense itch, blisters and pustules on the palms and soles of the feet, are characteristic symptoms of scabies in infants.

Children with immunodeficiency disorders have a severe itch and generalized dermatitis. Repeated recurrent infections and failure to thrive help distinguish the eruption in immunodefficient patient from AD.[19]

1.3 Treatment

Successful treatment of AD requires a systemic, multipronged approach that incorporates skin hydration, topical glucocorticoids therapy, the identification and elimination of precipitating factors such as irritants, allergens, infectious agents, and emotional stress.

Treatment should be individualized to address each patient's skin disease reaction pattern including the intensity of the rash and trigger factors that are different in different patients.[21]

1.3.1 Bathing and Moisturizers

A daily bath with mild non drying soap for at least 15-20 minutes helps to moisturize the skin making it less itchy. Avoidance of vigorous scrubbing and use of soft towel to dry the skin is recommended within three minutes and cream application.[22] Reducing dryness of skin which is often associated with eczema; moisturizers should be used regularly and are effective when applied within five -10 minutes after bathing. [23]

1.3.2 Silk Clothes

Avoiding irritant clothes and using silk clothes is one of the important key treatments in patients with atopic dermatitis to reduce irritation which aggravates pruritis. [24]

1.3.3 Antihistamines

Antihistamines are H_1 blockers. They may be used to treat AD, particularly because of their sedative effects, which may help the patient sleep at night.[5] Pruritus which is refractory to moisturizers may be conservatively treated with antihistamines. [26]

1.3.4 Antibiotics

Antibiotics are used to treat secondary skin infections. May be applied directly to the skin as an ointment, but are usually more effective when taken orally. [26]

1.3.5 Topical Corticosteroids

Topical corticosteroids are used for treatment of flare ups. Their side effects can be reduced by optimizing the number of applications and using new preparations with improved and reduced side effects. [27]

The method of application influence potency of the active ingredient. Ointments are more potent than creams and have a greasy appearance. They should not be applied to open or oozing lesions and in intertriginous folds. [28]

Systemic corticosteroids are reserved for patients with severe treatment-resistant atopic dermatitis

1.3.6 Phototherapy

Phototherapy is used in children with severe forms of AD, but its usefulness is limited because of long term risks, inconveniences and expenses.[29] This may be administered as ultraviolet A (UVA), ultraviolet B (UVB) or combined UVA and UVB. Possible long-term side effects of this treatment include premature skin aging and skin cancer.[30]

1.3.7 Immunomodulators and Antineoplastics

Cyclosporine are effective in patients with refractory AD. A dose of 5 mg/kg per day produces a rapid clearing of lesions. Relapse often occurs when therapy is stopped, so it can be cycled with topical corticosteroids or other treatments to prevent rebound effect and produce a longer remission. However, the general immunosuppressive effects and the serious adverse effects like nausea, hypertension, hyperbilirubinemia, and renal impairment limit its use. [26]

Immunomodulators are new drugs that have been developed to help control inflammation which reduce immune system reactions when applied to the skin. They can be used in patients older that two years of age and have few side effects. They not only reduce flare-ups but also maintain skin texture and reduce long term use of corticosteroid.[31, 30] Examples of these medications are the calcineurin inhibitors; tacrolimus ointment and pimecrolimus cream.

Calcineurin inhibitors are newer Immunomodulators, alternatives to steroid which have same potency with cyclosporine but with fewer side effects. They block T-cell activation and suppress release of pro-inflammatory cytokines. Tacrolimus an example of calcineurin inhibitor reduces Langerhans cell activation of T cell and cytokine production in eosinophils, mast cells, and basophils[32].

1.4 ATOPIC DERMATITIS AND OTHER ATOPIC COMORBIDITIES

AD is associated with other atopic diseases in many patients[33, 34]. Studies have shown that up to 80% of patients with AD develop allergic rhinitis or asthma later in childhood or during adolescence[34].

Bronchial asthma is an inflammatory disorder of the airways, which causes attacks of wheezing, shortness of breath, chest tightness, and coughing. Its prevalence varies worldwide from 1.6% to as high as 36.8% depending on its definition and it has been increasing about 50% each decade.[35] According to information collected by National Centre for Health Statistics of the Centre for Disease Control and Prevention in 2002, 12.2% of children had been diagnosed with asthma in their life time.[35] Studies done show that atopic dermatitis is frequently associated with bronchial asthma as it is one of the four components of atopic disorder.[35]Other authors suggest that atopy is a strong risk factor for developing asthma in children.[36, 37]

Allergic rhinitis is an inflammation of the nasal passages, usually associated with watery nasal discharge and itching of the nose and eyes. It affects approximately 20% of the population.[37]Allergic rhinitis can be associated with a number of co morbid conditions, including asthma, atopic dermatitis, and nasal polyps. [37, 38, 39]

Allergic conjunctivitis is an inflammation of the conjunctiva of the eye. It is itchy with lid swelling. Redness, irritation and watering of the eyes are other symptoms of allergic conjunctivitis. It is common in people with allergic disorders.[40] It affects approximately 25% of the general population and 30% of children with atopy.[41]

Atopic dermatitis being an autoimmune disorder, it is sometimes associated with vitiligo, pernicious anemia, diabetes mellitus, Hashimoto thyrioditis, and alopecia areata.

2. HYPOTHESIS

Atopic dermatitis is an uncommon dermatologic condition among children under five years attending public hospitals in Tanzania.

3. PROBLEM STATEMENT

The exact prevalence of AD in all age groups in Tanzania is not known although experience from clinics indicates that AD is common in Tanzania. Since the burden of AD is not known, there's a need to determine the prevalence of AD and its co-morbidities in children less than five years.

4. RATIONALE OF THE STUDY

In Tanzania there are no published studies that have been done to identify the burden of atopic dermatitis among children less than 5 yrs of age. Panya et al[42] did a study on the pattern of mucocutaneous disorders in HIV infected children attending care and treatment centers in Dar es Salaam 2007. It was found that 10.5 %of the children had atopic dermatitis. The study did not identify the magnitude of atopic dermatitis in the community.

Currently, the prevalence of atopic dermatitis among children less than 5yrs is unknown and its co morbidities like asthma, allergic rhinitis, and allergic conjunctivitis also are largely undetermined. So this study was to establish the magnitude of AD in children less than 5yrs in Dar-es-Salaam which will justify the need for proper management of these children with atopic dermatitis

5. OBJECTIVES

5.1 Broad objective:

To determine the magnitude and pattern of atopic dermatitis among children under five years attending public hospitals in Dar-Es-Salaam.

5.2 Specific objectives were to determine:

5.2.1 Prevalence of AD among children under five years attending public hospitals in Dar-Es-Salaam by age and sex.

5.2.2 Age of onset of AD among children less than five years.

5.2.3. Distribution of co-morbidities of AD among children less than five years with AD.

5.2.4. Distribution of positive family history of atopy in children less than five years with AD.

6. METHODOLOGY

6.1 Study design:

A Cross-sectional descriptive study.

6.2 Study area:

This study was conducted at the Reproductive and Child Health (RCH) clinics located at Mwananyamala, Amana, and Temeke Municipal Hospitals in Dar es Salaam, Tanzania.

Dar es Salaam is located along the Indian Ocean coast. It is divided into three minucipals namely Kinondoni, Temeke and Ilala, each having its own municipal hospital which receive patients referred from health centers and dispensaries located within the municipality. It has a total population of 2,942,436 and children less than five years being 451,115.

The total number of people in Kinondoni municipal is 1,257,366 and children less than five years 181,178 in Temeke municipal 886,529 and children less than five years 177,306, and in Ilala municipal is 798,541 and children less than five years are 92,631. (RCH zonal office, Dar es Salaam 2007).

In these hospitals, RCH clinics are run five days a week with an average daily attendance of 60 children under five years. The clinics run five days a week and on weekends and public holidays the children are attended at the general out patient department together with adults.

These RCH clinics attend all children who visit for vaccination, growth monitoring and those who are sick.

6.3 Study population:

All children under the age of 5 years attending RCH clinics in their fore stated municipal hospitals.

6.4. Inclusion and exclusion criteria

6.4.1 Inclusion criteria

All children from birth to 59 months attending RCH clinic in the 3 municipal hospitals.

All children whose parents/guardian consented for participation in the study

6.4.2 Exclusion criteria:

All children whose / parents guardian did not give consent to participate in the study. All children who did not fit in the criteria for AD.

All children whose parents claimed that in the past they had forms of AD but not clinically proven by the investigator.

6.5 Sample size estimation:

$$N = \frac{z^2pq}{d^2}$$

N is the sample size

Z is 5% significant level which is 1.96

P is the expected prevalence level worldwide which is 10%[3]

D is the desired precision which is 4%

Hence the minimum sample size is 214 children

Total of 301 children were included in the study.

6.6. Sampling technique/method

Monday and Friday were the days with a lot of children attending RCH clinic because they were given vaccination. So in each municipal hospital the author visited on these two days

in order to recruit more patients. Each RCH clinic within the municipal hospital was visited on Monday and Friday of the same week and the cycle repeated after all the three municipal Hospitals were visited. The other days of the week that is Tuesday, Wednesday and Thursday were distributed through the clinics by simple random selection. The investigator was introduced to the parents/guardians by the RCH nurse in charge in the waiting area. Health education on AD and the aim of the study was clearly explained to all the parents/guardians who brought their children. Children who were enrolled in the study were chosen by simple random technique. Numbers one and two were written in a piece of paper, well folded and were kept in a box. The box was thoroughly shaken before each caretaker and the caretakers were asked to take one piece of paper. Those with number two were recruited in the study while those with number one were attended to as other children in the clinic. Parents/guardians who were enrolled in the study were requested to give an informed consent verbally as well as written for their children's participation.

6.7. Data collection

A standardized questioner designed for the purpose of this study was used to collect information of the children who were enrolled in the study.

The information included:

- Demographic details: date, age, sex, residence,
- Clinical history: when and where did the 1st symptom appear, what was the 1st symptom?
- History of symptoms in either of the parents.

A thorough clinical examination was done by the principal investigator to assess the location, distribution of the dermatitis and its characteristics. The other eczematous conditions that never met the major and minor criteria were excluded.

The clinical criteria for bronchial asthma, allergic rhinitis and allergic conjunctivitis were as follows:

Bronchial asthma: shortness of breath, cough, wheeze, chest tightness and anxiety.

Allergic rhinitis: watery nasal discharge, itching of the nose, sneezing and nasal congestion.

Allergic conjunctivitis: intense itching increased tearing of eyes, redness, and swelling. (Refer to appendix I)

7.0 ETHICAL CLEARANCE AND ISSUES

Ethical clearance to conduct the study was sought from Research and Publication committee of Muhimbili University of Health and Allied Sciences. Permission was also requested from the district medical officers of the respective districts to conduct the study within the district. Data collection started after receiving permission. Consent was obtained from parents/guardian since the study participants were children.

For confidentiality purposes, numbers were used during data entry and analysis though questionnaire had names of participants. Care taker of each child filled the questionnaire and children were examined privately. All the children who were found to have atopic dermatitis were treated by being given appropriate medication according to the standard protocol of managing AD. Children seen at Amana hospital were referred to Amana skin clinic at Amana Municipal Hospital. Those seen at Temeke and Kinondoni were referred to Muhimbili National Hospital Skin clinic.

8.0 DATA MANAGEMENT AND ANALYSIS

All questionnaires were rechecked by the principal investigator and any missing information was sought out immediately.

Data entry and cleaning was done by the principal investigator. Data analysis was done using EPI Info version 6 and SPSS version 13. Chi square was used to determine if there was any association between variables and Fischer's test were used to determine if there's any association between variables which were less than 5. P value of less than 0.05 was considered statistically significant.

9.0 RESULTS

A cross-sectional descriptive study was done between June 2008 and January 2009 at Dar es Salaam Public Hospitals, RCH clinics to determine the magnitude and pattern of AD.

During the study period, a total of 301 children were recruited, 100(33.2%) from Ilala district, 102 (33.8%) from Kinondoni and 99 (33%) from Temeke municipals. In infants less than six months the AD was distributed on the cheeks while those older than two years AD was on the extensor surfaces of the extremities. One child had severe form of AD.

Table 1: Population characteristic of children under the age of five years recruited in the study (n=301)

	Sex					
	Male		Female		Total	
Age (mon)	No	%	No	%	No	%
0 < 12	89	49.2	92	50.8	181	60.1
12 < 24	26	41.3	37	58.7	63	20.9
24 < 36	10	41.7	14	58.3	24	8.0
36 < 48	10	52.6	9	47.4	19	6.3
48 < 59	3	21.4	11	78.6	14	4.7
Total	138	45.8	163	54.2	301	100

Out of 301 children studied, there were 138 (45.8%) males and 163 (54.2%) females with a ratio of 0.85 to 1. Majority of children 181/301 (60.1%) were below one year, while the least 14/301 (4.7%) were in the age group between 48 to less than 59 months (Table 1).

Table 2: Age at onset of first symptom of AD. (n=39)

	Sex					
	Males		Females		Total	
	No	%	No	%	No	%
Age in mon						
0 >24	11	40.7	16	59.3	27	69.2
>24	5	41.6	7	58.3	12	30.7
Total	16	41	23	59.9	39	100

A total of 39/301 (13%) had AD. There were 16(41%) males and 23(59.9%) females. Children who developed AD in the first two years of life were 27(69.2%) (Table 2).

Table 3: Distribution of co-morbidities of AD according to age. (n=19)

	Co-morbidities							
	Bronchial Asthma		Allergic Rhinitis		Allergic Conjuctivitis		Total	
	No	%	No	%	No	%	No	%
Age (mon)								
0 < 24	3	19.0	9	56.2	4	25.0	16	84.2
24 < 59	2	66.7	1	33.3	0	0.0	3	16
Total	5	26.3	10	52.6	4	21.1	19	100

p=0.25 for Fischer's test

Among 39 children who had AD, 19 (49%) had associated co-morbidities. Five (26.3%) had bronchial asthma, 10(52.6%) had allergic rhinitis and only four (21.1%) had allergic conjunctivitis. About 84% were below two year of age (Table 3).

Table 4: Distribution of co-morbidities according to sex. (n=19)

	Sex					
	Male		**Female**		**Total**	
Co-morbidity	No	%	No	%	No	%
Bronchial Asthma	3	60.0	2	40.0	5	26.3
Allergic Rhinitis	6	60.0	4	40.0	10	52.6
Allergic Conjuctivitis	1	25.0	3	75.0	4	21.1
Total	10	52.6	9	47.4	19	100

p=0.591

Among 19 children with co-morbidities, 10 (52.6%) were males and 9 (47.4%) were females. Out of five children with bronchial asthma, 60% were males and 40% were females. The same applied to allergic rhinitis. However for allergic conjunctivitis out of four children 75% were females and 25% were males (Table 4).

Table 6: Distribution of children with AD and a positive family history of AD and associated co-morbidities according to age. (n=19).

	Family History						
	Bronchial asthma		Allergic rhinitis		Allergic conjunctivitis		Total
Age (mon)	Yes	No	Yes	No	Yes	No	
<24	0	3	6	3	2	2	16
>24	0	2	1	0	0	0	3
	0	5	7	3	2	2	19

10.0. DISCUSSION

A total of 39/301 (13%) children were found to have atopic dermatitis. Twenty (19.6%) out of 102 children had AD from Kinondoni District, 10 (10%) out of 100 children had AD from Ilala District and 9 (9.1%) out of 99 children had AD from Temeke District.

Males were 16 (41%) and females 23 (59%) with AD. There were more females than males because they were the majority recruited in the study. Children who had developed AD in the first two years of life were 27 (69.2%). Among these children, 15 (38.5%) developed the first symptom within the first 6 months of life. This could be because of immune dysfunction, epidermal barrier dysfunction or genetic predisposition of these young infants.

The prevalence of atopic dermatitis varies from 5% to 20 % worldwide.[3] In African countries AD in children varies from 3% to 20% with severe forms being below 3%.[4] In this study, the prevalence of AD was 13% being similar to the findings of a study by D Sole et al [43]. This could be explained by the fact that both study populations had similar genetic predispositions. But Brazil has a different climate in different areas which might have influence the results as climate is a risk factor of AD. In contrast to my study, their study population was children aged 6-7 years and 13-14 years with a bigger sample size.

In this study, most of the children with atopic dermatitis were females, which is in contrast to a study done by Wichmann et al [44] involving 3926 children between the ages of 13-14 years. In the study by Wichmann, the proportion of children with AD was equally distributed among males and females. However both studies were conducted in the same geographic conditions.

In this study the prevalence of AD was found to be consistent with other studies done in developed countries[3] as well as those studies done in African countries[4]. This could be explained by the pathophysiology of the disease despite different geographical locations in

which studies have been done. It can also be explained by the fact that the study was done in urban area of Dar es Salaam. The results might have been different if it was done in a rural area.

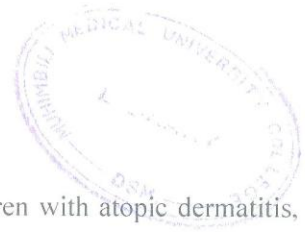
The finding of this study is similar to a study done by Nnoruka et al [45] who showed that the age of onset was within the first year of life. However, his study was a prospective one conducted for 2 years with a bigger sample size of 1019.

In agreement with other studies [5, 45] done in different countries of the world at different times, most of the children with atopic dermatitis were diagnosed before 1 year of age. In this study, majority of children with atopic dermatitis were below 6 months of age.

Majority of the children enrolled were of the age less than 6 months. In this group the prevalence of AD was found to be 45%.

Most children with co-morbidities were aged less than six months accounting for 84.2% as compared to older ages. The study has also shown that children in their infancy are more prone to AD and associated co-morbidities, a phenomenon which can be explained by the fact that the pathophysiology of AD and co-morbidities is the same.

In parallel with other studies [35, 36,37,38,39] done elsewhere in the world, this study has shown that atopic dermatitis is associated with other atopic disorders. Allergic rhinitis was the most frequent associated atopic disorder. It accounted for about 53% of the children, males being more affected than females. It was also found that male children get atopic dermatitis less frequently than their female counterparts. They suffer co-morbidities more frequently than females. This can be explained by the small sample size as there were less male children than females.



This study has also shown that out of those children with atopic dermatitis, 43.6% had a positive family history of either atopic dermatitis or other allergic disorders which is not so different from that reported by other authors. Nikol et al [26] found that 50%-60% of children had positive family history of atopic dermatitis.

Among children with atopic dermatitis, 48.7% had other allergic disorders. This corresponds to Nikol et al [26]. They reported that about 40%-50% of their children with atopic dermatitis were at risk of developing other allergic disorders.

This varies from the figures given by Krafchik [46]. He reported that only 30% of children with atopic dermatitis would develop asthma while 35% would get nasal allergies later in life.

Children younger than two years and who either one parent had AD, had an increased risk of the disorder. This is because AD is hereditary and it has a genetic predisposition component. Though this was not statistically significant.

10.1 LIMITATION OF THE STUDY

This was a hospital based study hence not representing a true picture of the community. It also recruited children attending public hospitals only thus children who attended private health facilities were not included in the study of which the prevalence could have been different due to different exposure.

About 20% of Tanzanian population is children below 5 years. The sample size was quite small to represent the number of children below 5 years in a big city like Dar es Salaam.

11.0. CONCLUSION AND RECOMMENDATION

The prevalence of atopic dermatitis in this study is 13%. The same applied to the age of onset, clinical manifestations and associated co-morbidities. These children should be identified and given treatment promptly as it can affect their lives.

This study was conducted in public hospitals only. A community based study is therefore recommended so as to determine the true prevalence of AD in Tanzania.

12. REFERENCES

1. Fennessy M, Coupland S, Popay J et al The epidemiology and experience of atopic dermatitis during childhood :a discussion paper on the implication of current knowledge for health care, public health policy and research Journal of epidemiol community health 2000;54:581–589.
2. Sampson HA, Food sensitivity and the pathogenesis of atopic dermatitis Journal of the Royal society of Medicine 1997:90 (supp 30) 2-8.
3. Weston WL, Howe W. Epidemiology, clinical manifestations and diagnosis of atopic dermatitis version 16.1 January 2008 http://www.uptodateinc.com/patients/content/search.do?search=epidermiology+and+clinical+manifestation+of+atopic+dermatitis&source
4. Melaku K Atopic eczema in school children. Ethiopian J. Health Development 2000: 14 :105-108
5. Dockery GL what you should know about atopic dermatitis Podiatry Today Sept 2005 ISSN 1045-7860 Volume 18 Issue 9:46-54
6. Atopic dermatitis genetics and epidemiology. www.dermatitisfacts.com/atopic-dermatitis-eczema-genetics-epidemiology.php accessed March 2008
7. Friedman J Y, Reed SD, Weinfurt K et al. Parents reported preference scores for childhood atopic dermatitis disease states 2004, 4:21 doi: 10.1186/1471-2431-4-21 www.biomedcentral.com 1471-2431/4/21
8. Cooper K D, Atopic dermatitis: recent trends of pathogenesis and treatment. J. Invest dermatology 1994; 102:128-37
9. Hamid Q, Boguniewics M, Leung D Y. Differential in situ cytokine gene expression in acute versus chronic atopic dermatitis. Journal of clinical investigation 1994; 94: 870-6
10. James JM, Kagey SA, Sampson HA, Patients with severe atopic dermatitis has activating circulating basophils. Journal of Allergy and Clinical Immunology 1993; 91: 1155-62.

11. Fauler J, Neuman C, Tsikas D, Enhanced synthesis of cysternyl Leukotrienes in atopic dermatitis British journal of dermatology 1993; 128: 627-30.

12. Motala C, The Alisa Handbook of Practical Allergy, Allergy society of South Africa Atopic dermatitis page 1 www.allergysa.org. accessed October 2007

13. Leung D Y Atopic dermatitis: the skin as a window into the pathogenesis of chronic allergic diseases. Journal of Allergy Clinical Immunology 1995:302-18

14. Australian society of clinical immunology and allergy. Atopic dermatitis www.allergy.org.au/content/view/158/300. Accessed May 2008

15. Beltrani VS, the clinical spectrum of atopic Dermatitis. Journal allergy clinical Immunology 1999; 104: S 87-98

16. Burks AW, James J M, Hiegel A, et al Atopic dermatitis and Food hypersensitivity reactions J. pediatrics 1998; 132:132-6

17. medpedia, atopic dermatitis http://wiki.medpedia.com/atopic_dermatitis. Accessed June 2008

18. Bieber T, the New England journal of medicine. Atopic Dermatitis: 2008 number 14 358: 1483-1494. content.nejm.org/cgi/content/short/358/14/1483

19. Correale CE, Walter C, Murphy L et al, American Family Physician: Atopic dermatitis: A review of Diagnosis and treatment 1999 Am Fam phycian 1999;60:1191-210 published by AAFM Sept 1999 www.aafp.org/afp/990915ap/191.html. accessed June 2008

20. Kuhnyari A, Egyud K, Szaboi I et al Prevalence of atopic dermatitis among children under 19 in an East-Hungarian agricultural county. Clinical & Developmental Immunology, June–December 2006; 13(2–4): 395-399 accessed May 2008

21. Leung DYM, Tharp M, Boguniewics M, Atopic dermatitis Fitzpatrick's Dermatology in general. International edition 5th edition volume 1:1473 published by the Mc Graw-Hill 1999 published in the USA pg 1464-1480

22. Keepkidhealthy.com. http://www.keepkidshealthy.com/welcome/treatmentguides/eczema.html. accessed May 2008

23. Rupert M. What goes with eczema? British journal of dermatology 2004; 150: 127-131 www.talkeczema.com/webdocs/features/what_goes_with_eczema.php

25. Stanway A. Atopic dermatitis, eczema. DermNet dermnetnz.org/dermatitis/atopic.html. accessed May 2008

26. Nicol, Heer N, Managing atopic dermatitis. The American journal of primary healthcare April 2000 volume 25 issue 4. accessed May 2008

27. Akdis CA, Akdis M, Bieber T, et al atopic dermatitis: a review of recent advances in the field 2006; 61 8: 969-987 cme.medscape.com/viewarticle/586561 published 2009

28. Buys L, M Treatment options for atopic dermatitis 2007 Am Fam physician 2007; 75:523-8,530

29. Spergel J M, Schneider LC, Atopic dermatitis: The internet Journal of asthma, allergy and immunology 1999 volume 1:1 www.ispub.com/journal/the_internet_journal_of_asthma_allergy_and_immunology_volume_1...1_/4/.../atopic dermatitis. accessed January 2008

30. Cono J et al, Atopic dermatitis/eczema National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS): NIH Publication No. 03-4272 www.athealth.com/consumer/disoder/eczema.html accessed January 2008

31. Buck M, L Topical macrolactam Immunomodulators for atopic dermatitis: Pediatric pharmacotherapy 2002; 8 5 www.healthsystem.virginia.edu/alive/pediatrics/pharmnews/200205.pdf. accessed May 2008.

32. Guenther L: Topical Calcineurin Inhibitors—Efficacy and Safety in Atopic Dermatitis http://www.skintherapyletter.ca/fp/2007/3.2/3.html accessed May 2008

33. Diepgen T L, Radulescu M: cause, types and specific localizations and treatment

of atopic dermatitis http/eczema.dermis.net/content

34. Scott M. Dry skin chapter. dermatitis. atopic dermatitis Familypracticenotebook.com: fpnotebook.com/DER/Dry/AtpcDrmts.htm. accessed December 2007

35. Liu AH, Covar RA, Spahn JD et al. Childhood Asthma In Kliegman RM, Jenson HB, Behrmann RE et al. Nelson textbook of pediatrics 18th ed. Saunders an imprint of Elsevier 2008: pgs953-954

36. Wjst M, Boakye D. Asthma in Africa. PLoS Med 4(2):e72doi:10.1371/journal.pmed.0040072

37. Boguniewicz M, Allergic disorders. In: Hay W, Levin MJ, Sondheimer JM et al. Current diagnosis and treatment in Pediatrics. International Ed, 18th ed. Lange medical books/Mc Grawhill 2003; 1049-1060. accessed January 2007

38. Stoppler MC. Hay fever (allergic rhinitis) http://www.medicinenet.com/hay_fever/article. accessed January 2008

39. Milgrom H, Leung DYM. Allergic rhinitis In Kliegman RM, Jenson HB, Behrmann RE et al. Nelson textbook of pediatrics 18th ed. Saunders an imprint of Elsevier 2008; pg949-952

40. Nordqvist C, what is allergic conjunctivitis? What causes allergic conjunctivitis? htpp//www.medicalnewstoday.com/articles/157692.php. accessed January 2008

41. Boguniewicz M, Leung DYM Ocular allergies. In In Kliegman RM, Jenson HB, Behrmann RE et al. Nelson textbook of pediatrics 18th ed. Saunders an imprint of Elsevier 2008;pg 978

42. Panya M: The pattern of mucocutaneous disorders in HIV infected children attending care and treatment centers in Dar es Salaam. (Dissertation): Muhimbili University of Health and Allied Sciences, 2007.

43. Sole D, Camelo-Nunes, Wandalsen GF et al Prevalence of atopic dermatitis and related symptoms in Brazilian schoolchildren: results from the International study

of asthma and allergies in childhood (ISAAC) phase 3. Journal of Investigation, Allergology and clinical Immunology 2006; 16: 367-376

44. Wichmann J, Wolvaardt JE Maritz et al association between household living conditions and eczema in adolescents living in Limpopo province, South Africa Current Allergy & Clinical Immunology, August 2007 Vol 20, No. 3

45. Nnoruka EN, current epidemiology of atopic dermatitis in south eastern Nigeria International journal of dermatology 2004; 43: 739-44

46. Krafchik BR. Atopic dermatitis updated Apr 2006 emedicine.medscape.com/article/1049085-overview-70k