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Some Aspects of Asthma, Allergic Rhinitis and Atopic Dermatitis In School Children in Khartoum Province

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DEDICATION

This study is dedicated to the souls of my parents who did educate me, encouraged and supported my education during their lifetime

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ABSTRACT

A school survey enrolling 2176 pupils aged 6-13 years old in basic schools in Khartoum province was conducted during the period from October 2002 to February 2003 in 22 governmental randomly selected basic schools (out of 185 schools).

The aim of the research was to study the prevalence, severity and triggering factors, of asthma, allergic rhinitis and atopic dermatitis including the interrelations of the three atopic conditions. A detailed self administered adapted ISAAC questionnaire with supplemented questions was used. The parents and children at home completed the questionnaires. Physical examination and anthropometrics measurements were also recorded.

The cumulative prevalence for asthma, allergic rhinitis and atopic eczema were 13.1%, 22.7%, and 8.1% respectively, with respective 12 months period prevalence of 10.8%, 22.1% and 7.1% respectively. The 12 months period prevalence for exercise induced wheeze, nighttime cough, and rhinoconjunctivitis were, 4.6%, 8.6% and 11.4% respectively. The prevalences of severe asthma, allergic rhinitis and atopic eczema were,14.1%, 3% and 23.1% respectively.

Family history of atopy and father smoking were significant associations for children with asthma. Asthma affected males more than females while the converse were true for allergic rhinitis and atopic eczema. Common triggering factors for asthma were respiratory infections, dusts and cold air. For allergic rhinitis, common triggers were temperature changes and dusts. Extreme weather changes and sweating were most common triggers for dermatitis. The commonest signs were pigeon chest (28.9%), oedematous nasal turbinate (72.5%) and accentuated palmar crease (24%) for asthma, rhinitis and dermatitis respectively. About 55.7% of children with asthma had allergic rhinitis, while 16.2% had atopic dermatitis. All three: asthma, allergic rhinitis and atopic dermatitis occurred in 12.3% of the children.

Asthma is prevalent among Sudanese school children. There is a need for specialised asthma clinics for better management and health education of the patients. Further studies on asthma and other atopic conditions are recommended.

ملخص الاطروحة

أجري مسح مدرسي شمل 2176 تلميذا وتلميذة تتراوح اعمارهم ما بين 6-13 عام في مدراس الاساسي بمحافظة الخرطوم في الفترة مابين اكتوبر 2002م الي فبراير 2003م ضمت هذه الدراسة 22 مدرسة حكومية تم اختيارها عشوائيا بين 185 مدرسة.

هدف البحث هو دراسة امراض الربو الشعبي ، التهاب الانف الاستهدافي، والتهاب الجلد التاتبيى من حيث معدل الانتشار، درجة الوخامة والعوامل المؤهبة لكل من هذه الامراض بما في ذلك دراسة العلاقة (التداخل) بين الحالات التاتبيه الثلاث. تم استخدام استبيان "ISAAC" مفصل ومعدل مع اسئلة اضافية لهذا الغرض قام بملئه الوالدان(الآباء) والاطفال في منازلهم. كما انه تم اجراء الفحص السريري والقياسات البشرية للاطفال قيد الدراسة ثم تسجيل ذلك.

وجد أن انتشار التراكمي لكل من الربو الشعبي ، والتهاب الانف الاستهدافي والتهاب الجلد التاتبي، كان 13.1% ،22.7% و8.1% علي التوالي . اما الانتشار خلال 12شهر بالنسبة لكل من الربو الشعبي ، والتهاب الانف الاستهدافي والتهاب الجلد التأتبى 10.8%، 22.10%، 7.1%علي التوالي. وكما أن معدل الانتشار خلال 12 شهر بالنسبة لكل من الازير المصاحب للتمرين، السعال الليلي والتهاب الملتحمة والانف هو 4.6% ، 8.6%و 11.4% علي التوالي. وجد أن الربو الشعبي في الاطفال له علاقة هامة (ارتباط هام) مع وجود حالة تأتبيه في الاسرة وتدخين الاب. حالات الربو الشعبي وجدت عند الذكور اكثر من الاناث بينما وجد عكس ذلك بالنسبة لحالات الـ التهاب الانف التأتبيى او الاكزيما. وجد أن العوامل المؤهبه الشائعه لمرض الربو الشعبي هي الاخماج التنفسية،

التعرض للغبار والهواء البارد. أما التهاب الانف التأتبيى فان التغيرات في درجة الحرارة والغبار هما العوامل المؤهبة الاكثر شيوعاً. بينما كان التعرق (العرق) الطقس الاقصي هما اكثر العوامل المؤهبه الالتهاب الجلد الاستهدافي . العلامات السريرية الاكثر شيوعاً كانت الصدر الحمامي (28.9%)، و وزمة خزروفي الشكل الانفي (72.5%) وثنيه كفي بارزة (24%). وذلك للربو الشعبي التهاب الانف الاستهدافي والتهاب الجلد التأتيبى علي التوالي. وجد أن 55.7% من الاطفال المصابين بالربو الشعبي يعانون من التهاب الانف الاستهدافي بينما 16.2% منهم يعانون من التهاب الجلد التاتبيى. حدوث الربو القصبي، التهاب الانف الاستهدافي والتهاب الجلد التاتبي وجدت في 12.3% من الاطفال.

الربو الشعبي منتشر بين تلاميذ المدراس بالسودان وهنالك حوجه لعيادات متخصصة للربو وذلك من أجل المعالجة – الأفضل للمرض والتثقيف الصحي للمرض. كما ان هناك حوجه لمزيد من الدراسات في الربو الشعبي وحالات التاتبية.

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ABBREVIATION

AD	Atopic dermatitis
AR	Allergic rhinitis
IgE	Immuno globulin E
ISAAC	International study of asthma and allergies in children
N	Number
PEFR	Peak Expiratory Flow Rate

1. INTRODUCTION AND LITERATURE REIVEW

1.1. Introduction: Asthma, allergic rhinitis and atopic dermatitis (AD).

Asthma is the most common chronic disease in children [1,2]. It is a major global health problem, which exerts a substantial burden on the family, health-care services and the society as a whole [1,3].

Asthma can derange the child's activities such as playing or sporting events. Besides, it can disturb sleep patterns thus affecting the academic and career success of the child because of poor school attendance which is associated with asthma attacks.

The prevalence of asthma has increased globally in past three decades [1,2,4]. An increase in the prevalence of asthma has also been reported in the developing countries [5,6].

Strachan proposed a protective effect of infections on atopy by describing an inverse association between the number of older sibling and hay fever [7].

Bodner et al found similar protection against atopic diseases [8]. Repeated lower respiratory tract infections in early life are said to be associated with the subsequent development of asthma. In contrast, early episodes of other infections have

an inverse relation to the development of asthma and other respiratory symptoms [7]. The protective effect of high sibling number appears to operate after birth [9]. Mycobacterium tuberculosis, by inducing TH_1 type immune response and suppressing the development of TH_2 type immune response may reduce the risk of asthma [10].

Asthma impairs growth, [11,12] but most asthmatic children will eventually obtain their predicted adult level [13].

Allergic rhinitis is a common allergic condition in most communities. Its prevalence is 10-20% in the United States [14]. The prevalence of hay fever and allergic rhinitis has increased over the past 30 years with a higher prevalence in urban areas [15]. Allergic rhinitis comprises of disease to a variety of allergens. Allergy to pollen is known as hay fever or seasonal rhinitis [16].

Perennial allergic rhinitis is persistent and is mainly caused by house-dust mite, animal dander or fungal spores [14].

Family size and position have an inverse relation to hay fever similar to asthma [15].

Children are particularly affected by this illness. Nasal obstruction often leads to disturbed sleep pattern. It causes school absence. It also affects the patient's daily activities in

severe cases similar to that in patients with moderate to severe asthma. Disorders of dentition in young children may occur [14].

Disease process may affect the respiratory mucosa of both the nose and the chest, being similar in pathogenesis but with the production of different symptoms in the nose and the chest [17].

Atopic dermatitis (AD) is a pruritic chronic inflammatory skin disease, which commonly starts in early infancy and childhood, but can persist or start in adulthood. It has significant adverse effects on life [18].

Atopic dermatitis has a familial tendency and is associated with asthma and allergic rhinitis [19]. Asthma is high among patients with AD with a range of 20 to 50 percent [20,21]. Patients with AD have behavioural symptoms, fearfulness and sleep difficulty. The child behaviour problems and maternal stress are significant in the severely affected children. Restriction from mixing with peers and teasing by peers may exacerbate symptoms [22].

There have been concern about growth failure in asthma[11,12], and atopic dermatitis [23]. Patel et al found that like in children with asthma, children with AD despite growth delay, the normal adult height is attained[13,23]. The growth pattern in

asthma is thought to be related to the atopic state rather than asthma itself [23].

Asthma, allergic rhinitis and atopic dermatitis are multifactorial diseases resulting from various familial and environmental influences. The association of atopy in the parents and the child developing the same type is highest for atopic dermatitis than allergic rhinitis and the least for asthma [24]. The intricate play of the genes with each other and with the environment may ultimately determine whether an individual develops asthma for instance [25].

In Sudan few studies have been done on childhood asthma. Salbutamol by nebulization is the treatment of choice of acute childhood asthma [26]. Supervised corticosteroids was recommended in the treatment of moderate to severe persistent asthma[6]. The prevalence of asthma among school children in Khartoum was found to be 10.7 percent [27]. Urban – rural difference was found to be among Khartoum dwellers [28]. Cold air and dust were found to be the most common triggering factors [29] and severe asthma was reported in 16% of asthmatic children [30].

Atopic dermatitis was found to be the most common dermatitis in children [31].

1.2. Historic backgrounds.

1.2.1. Historic background of asthma.

Asthma is an ancient disease. The Egyptians referred to a condition probably asthma (1550B.C). The Chinese writing “Neo ching” (1000BC) mentioned the plant “Ma Huang” from which ephedrine was later extracted[32].

Hippocrates (b.460 – 375BC) described asthma and eczema as well as allergy to goats cheese. The remedy for asthma was to encourage the flow of phlegm by means of emesis, purging and bleeding [32,33].

Auleus Celsius 25BC-AD40, made a thorough description of dyspnoea, asthma and orthopneoa in Treatise 'de Medica'[33].

The Word asthma was coined by Aretaeus of Cappadocia in 120- A180 [32].

Moses Maimmonides (1135-1204) physician of Sultan Saladin wrote the "treatise" on asthma in Arabic language. He also advised the sultan's son who was asthmatic[32,33].

Nicolas Monardes (1567) introduced the use of ipecacuanha[32]. Sir Walter Raleigh in 1559 introduced the use of tobacco in treating respiratory ailments. Felix Platter 1603 said that asthma was due to obstruction of small arteries or to nerve disturbances[33] . Thomas Willis (1680) classified asthma in to the "Pneumonic" and "convulsing varieties" [32]. Joan Von Helmont (1662) compared asthmatic attack to epileptic fit[32,33]. Jerome Cardan treated the asthma of Arch Bishop Hamilton of St. Andrew by avoiding feathers[32].

Description of asthma causes (tobacco smoke, dust, environmental factors) and the description of heredity in asthma was made by John Floyer in 1698.

Bernardino Ramazzini linked asthma to house dust mite (1713) [32].

William Wilthering published an account of spasmodic asthma (1786) while Franz D Reisseisen described bronchoconstriction in 1808[32,33].

Rene Laennec in 1816 invented the stethoscope and confirmed bronchospasm as an important feature of asthma. In 1860 Henry Salter described the pathology and treatment of asthma[33].

1.2.2. Historic background of allergic rhinitis.

Rhases in AD b. 865 described seasonal catarrh due to Roses in Persia[33].

John Bostock in 1819 made the first clear description of hay fever and asthma while John Elliotson in 1831 linked hay fever and asthma to exposure to pollen and contact with rabbit[32].

Both Morril Wyman (1872) and Charles Backley found that weed and grass pollens cause hay fever [32,33].

Anaphylaxis was described by Charles Richet in 1902 and in 1906, Alfred Wolf-Eisner described Hay fever as an anaphylaxis phenomenon. Clemens Pirguet coined "allergy" to denote hypersensitivity or a capacity for reaction.

In 1910, Samel Mettzen classified asthma and allergic rhinitis as allergic diseases.

Bernard Halpern in 1942 found that antihistamine was effective in treating hay fever but less effective in treating asthma[32].

1.2.3. Historic back ground of atopic dermatitis.

The word eczema is derived from the Greek word "ekzein" meaning to boil out or to effervescence. It originated in AD 543 and is applied to many skin diseases[34].

In 1892, Besniers first described a skin disorder typical of a topic dermatitis, while in 1902 Brocq used the term "diffuse neurodermatitis" [35].

In 1923 Coca and Cooke used the term atopy to describe hyperresponsiveness in human with genetic predisposition to manifest clinical allergic syndromes such as asthma and hay fever.

In 1933, Wise and Sulzberger included atopic dermatitis in the group of atopic disease[32,33].

1.3. Definition of asthma.

It has been difficult to precisely define asthma. Recently, asthma has been defined as a chronic inflammatory disorder of the airways, characterized by completely or partially reversed airway obstruction with or without therapy. Airway inflammation results from interactions of various cells, cellular elements, or persistent bronchospasm which causes symptoms like

wheezing, breathlessness, chest tightness and cough, especially at night or after exercise. The airflow obstruction is associated with bronchial hyperresponsiveness[36,37,38].

For community based studies, the international study of asthma and allergies in childhood (ISAAC) adopted a questionnaire in which asthma is diagnosed on the basis of symptoms only [2]. The ISAAC questionnaire has been validated. An Australian study found that consistent responses to the first two questions (Wheeze ever and wheeze in the last 12 months) had a sensitivity of 85% and specificity of 91%, and a positive and negative predictive values of 61%, 94% respectively[39].

1.4. Prevalence of asthma.

There is concern that the prevalence of asthma and its severity is increasing, especially in developed countries [4,39-42]. In developing countries, recent studies showed that the prevalence of asthma is increasing, particularly in cities[5,6,43,44]. There is global variation in asthma distribution, ranging from 1.6% to 36.8% of the population [44,45]. In the United Kingdom and Australia, the prevalence is 17-30% while low prevalence of (1-7%) was reported in Eastern Europe and China[44,45].

In Sudan, the urban-rural asthma prevalence was found to be 14.5 % and 9% respectively[28]. Two unpublished surveys found the prevalence of asthma to be 13% and 16% respectively [6,28]. In 1994, the prevalence of asthma was 10.7% among school children in Khartoum[27].

In Jordan, the prevalence of asthma was found to be 8.3% in schoolchildren [46]. The prevalence of asthma was 11.5% in Saudi Arabia[47], 10.5% in the Palestinian Authority [44]. The prevalence were found to be 13% in South Africa[48], 10.2% in Nairobi(Kenya) [49], and 10.2% in Nigeria[50] In Ethiopia, the prevalence of current asthma in children was 2.4% [43]. The prevalences were 33.2% in U.K [39], 22% in Australia [50],and 12% in Singapore[51]. There are variations in the prevalence of allergic diseases in different centres in the world[45,50].

1.5. Epidemiology of asthma.

Childhood asthma has a substantial morbidity and high mortalities in some countries. Western countries have higher prevalence than the developing countries[37,38,43,45,50]. urban dwellers are more affected than rural dwellers [43,44].

Boys are affected more than girls until puberty. Asthma can begin at any age. About 80-90% of children have first symptoms by 5 years of age[37].

Early transient wheezers, wheeze before age of 3 years with remission after the age of 6 years. They are at risk from maternal smoking. Persistent wheezers have a family history, serum immunoglobulin E(IgE) and other atopic diseases[38,52].

Reasons for increased prevalence include housing, increased exposure to house-dust mite, environmental pollution and may be diet [37,38], early onset respiratory viral infections, poverty, low birth weight, small house hold, and large family. Increased awareness of parents and physicians[53] and congenital small lung.

Asthma exerts a vast economic and social toll [54]. Most children have moderate to severe asthma. Few have severe perennial asthma. Severely affected children may have growth retardation, chest deformities and persistent abnormal lung function [37].

Risk factors of death are: under estimation of severity, delay of appropriate therapy, underuse of drugs, incompliance, stress, excessive, sudden exposure, recent admission and sudden severe airway obstruction in chronic steroid dependent patients.

1.6. Pathophysiology of asthma.

Airway inflammation is the primary problem in asthma. An initial step is the release of inflammatory mediators from mast cells, alveolar macrophages, T lymphocytes and epithelial cells upon exposure to allergic irritants, cold air or exercises[37,55,56,57].

The release of histamine and leukotrienes, causes bronchospasm termed the "early phase asthmatic response" [56].

The inflammatory mediators also activate eosinophils and neutrophils and their migration to airways where they cause injury. This "late-phase asthmatic response" results in epithelial damage oedema, mucous hypersecretion, and bronchial muscles hyperresponsiveness. Varying airflow obstruction leads to recurrent episodes of asthma symptoms [36,37,56].

1.7. Etiology of asthma.

The predisposition of wheeze is determined by interactions of: genetic, atopic and environmental factors.

Genetic influence: Asthma is partially inherited. Boys are more likely to have asthma than girls if one or both parents have asthma. The concordance for asthma is 19% among monozygotic twins and only 5% for dizygotic twins[58].

Atopy: this is the tendency to produce IgE antibodies in response to environmental antigens and manifest clinically as asthma, allergic rhinitis and eczema. It is partly autosomal dominant inherited through maternal line. The gene locus is on chromosome11q. Atopy is a consistent risk factor for asthma[58-60].

Respiratory infections: Respiratory viral infections are the commonest triggers of wheeze, Moderate to severe bronchiolitis is associated with an increase risk of asthma later. This theory is debatable[58].

Maternal smoking: Maternal smoking increases the severity of asthma symptoms. The association is stronger for boys and older children and is affected by duration of exposure [58,61].

Air pollution: The effect of chemical and particulate pollution remains controversial although, there is a correlation between the level of ambient pollution and asthma symptoms[58].

Socioeconomic factors: Racial discrepancy in prevalence was said to be related to environmental factors and poverty[58,62].

1.8. Clinical manifestations of asthma.

Acute episodes are often caused by exposure to irritants, noxious fumes, allergens and chemicals. Slow onset occurs with viral respiratory infections.

The symptoms and signs include, cough, wheeze, tachypnoea, dyspnoea, prolonged expiration, use of accessory muscles of respiration, chest hyperinflation, cyanosis, tachycardia and pulsus paradoxus.

In extreme respiratory distress, wheeze may be absent and there may be difficulty in talking or walking. Abdominal pain is common in younger children[63]. Vomiting may temporary relieve symptoms.

During severe obstruction there may be profuse sweating and low grade fever.

A barrel chest indicates chronic severe asthma. Harrison sulci and chest hyperinflation may be present in children with recurrent severe retractions[37].

1.9. Clinical patterns of asthma.

Asthma pattern varies greatly from individual to another even within an individual. The severity and frequency of exposure may change with time.

According to the national heart, lung and blood institute, expert panel report, guidelines for the diagnosis and management[3], and the ISAAC: rationale and methods[2], asthma may be broadly divided into mild, moderate and severe asthma[2,3,51].

1.9.1. Mild asthma:

Children with mild asthma have the following characteristics.

- Brief exacerbations.
- Wheezing episodes of 1-3 times a year.
- Normal school attendance or absence of 1-3days per year.
- Night time asthma symptoms 2 or more times per months.
- Normal daily activities or a little affected.
- Patient asymptomatic between attacks.
- Normal lung functions test between attacks with peak expiratory flow (PEF) 80% of predicted value.

1.9.1.a. Variants of mild asthma:

1.Exercise-induced asthma: this occurs in the majority of children because of their high level of activity. The symptoms start to peak at 5-15 minutes after exertion. It is more marked with exertion in cold dry atmosphere [58].

2.Noctural asthma: Children with asthma often complain of asthma and wheeze at night. It is due to an exaggerated normal circadian fall in lung function[58].

3.Impossible asthma: Some children will continue to have symptoms irrespective of appropriate therapy. Compliance is very important in the success of treatment[58]

4.Cough variant asthma: children may have chronic cough as the only manifestation of asthma. It may be due to hyperresponsiveness of cough receptors[58].

5. Drug (aspirin) induced asthma: This occurs after ingestion of drugs particularly aspirin and other non steroidal anti-inflammatory drugs[58].

1.9.2. Moderate asthma.

Children with moderate asthma have more frequent symptoms and often have cough and mild wheezing between more severe exacerbations. This type of asthma is characterized by:

- Wheeze episodes 4-12 times a year.
- Exacerbations may last for some days.
- Exacerbations may affect sleep.
- Nighttime asthma symptoms about one night per week .
- Daily activities are moderately affected.
- School absence of 4-12 days a year.
- Requires daily use of short β_2 agonists.
- PEF is 60-80% predicted value.

These children many experience cough and wheeze with moderate exercise. Therefore they need daily use of short β_2 agonists.

1.9.3. Severe (chronic) asthma.

Children with severe asthma have daily symptoms of wheezing and many have severe episodes. Severe asthma has the following characteristics.

- Continuous asthma symptoms.
- Frequent exacerbations more than 12 a year.
- Sleep affected one or more nights a week.
- Daily activities greatly affected.
- School absence more than 12 days per year.
- PEF is < 60% predicted value.
- Speech limitation during attacks.
- Have chest deformities.

These children have onset of asthma before 3 years of age they have other atopic diseases.

1.10. Acute severe asthma.

This is a clinical diagnosis defined by increasing severe asthma that is not responding to drugs that are usually effective. These patients have chronic steroid dependent asthma; have recurrent visits to emergency units. They have poor compliance and may have sudden onset of respiratory distress. On examination patients have tachycardia, tachypnoea, cyanosis and speech limitation. Patients may be agitated or lethargic. There is poor air exchange and there is use of accessory muscles of respiration[7].

1.11. Triggering factors of asthma.

Various allergic and specific stimuli, provoke the attacks of asthma. Asthma triggers include:

1.11.1 Allergens: like animal allergens (pets like cats , dogs , rodent. birds), house-dust mites, cockroaches, indoor fungi (mold, mildew) outdoor allergens (trees, grass, weed pollens, mold spores)[37,64].

1.11.2. Food and medicine sensitivities: Like non-selective beta blockers, aspirin and other non-steroidal inflammatory agents, food containing sulfites, (potatoes, shrimp, dried fruits) [37]

1.11.3. Respiratory infections: Such as ear and sinus infections [37,65]

1.11.4. Irritants: As cigarette smoke, fireplace smoke, perfumes, cooking odours, car exhaust, gas fumes, weather changes, air pollution, musty odours and weather conditions [64,66,67].

1.11.5. Chemicals: Industrial and occupational exposure to chemicals.

1.11.6. Physical activity: like exercise.

1.11.7. Mood changes: like excessive laughing or crying.

1.12. Diagnosis of asthma.

Asthma is a clinical diagnosis, which is based on the recognition of a pattern of symptoms, signs and history. [4,56] For an accurate diagnosis there must be:

a. Episodic symptoms of airflow obstruction.

b. Airflow obstruction or symptoms that are at least partially reversible spontaneously or pharmacologically [36]

c. Exclusion of alternative diagnosis.

Peak flow monitor for 1-2 weeks may help to confirm or negate the diagnosis. The challenge tests (histamine/methacholine)
may give conflicting results so their use is questionable [38].

In epidemiological studies, the ISAAC core questionnaire on wheeze is used. Asthma being diagnosed on a positive response to questions as wheezing ever and wheezing in the past year [2,3,9].

1.13. Differential diagnosis of asthma.

These include other causes of airway obstruction like: [36,37]

- Allergic rhinitis
- Allergic bronchopulmonary aspergillosis
- Bronchiolitis.
- Cystic fibrosis.
- Aspiration syndromes.
- Bronchopulmonary dysplasia.
- Foreign bodies in air way or oesophagus .
- Gastroesophageal reflux.
- Laryngomalacia.
- Primary cilliary dyskinesia.
- Subglotic stenosis.
- Vascular ring, right aortic arch.
- Tropical oesinophilia.

1.14. Management of asthma.

The National asthma education and prevention program (NAEPP) Expert panel (1997) recommended new guidelines asthma management [36,56].

There are two approaches the “step up” approach and the “step down” approach. The step down approach is the best, where treatment is started at higher than patients’ disease level of severity.

The correct diagnosis is necessary and treatment strategies is individualized [36,56,69,70].

The aims of treatment are:

1. Prevent symptoms.
2. Maintain near normal lung function, normal activities and sleep pattern.
3. Prevent exacerbations, eliminate triggers and reduce emergency visits.

4. Reduce dosage and side effects, use β_2- agonists less than twice daily, and use of short course of oral corticosteroids less than 4 times per year.

5. Satisfy patient and parents.

1.14.1. Asthma medications.

There are two categories [3,56].

1.14.1.a. Quick relief therapy:

a. *Short acting* β_2 *agonists*. Agents used for relieving and preventing bronchospasm. The beta$_2$ selective agonists include: albuterol, terbutaline and others. They have rapid onset of action and shorter duration of action. Ephedrine is an example of non-selective beta agonists. [56].

b. *Systemic corticosteroids*: used for short-term therapy for initial control and treatment of moderate to severe exacerbation. Duration of therapy is 3-10 days [3,36,37,56].

c. *Anticholinergic drugs:* Ipratropium inhibits vagal mediated bronchoconstriction. It is used as an adjunct to inhaled beta$_2$ agonists in severe exacerbation of asthma[36,56].

1.14.1.b. Long term control medication.

(a)Corticosteroids: These are potent anti-inflammatory agents used with all types of persistent asthma.

(b) *Cromolyn sodium and Nedocromil*: these have mild to moderate anti-inflammatory effect. They are safe. Both can prevent exercise induced bronchospasm (EIB).

(c) *Salmeterol and extended release albuterol*: they are used in combination with inhaled corticosteroid or other anti-inflammatory agents. Salmeterol is used in nocturnal and exercise induced asthma.

(d) *Theophylline:* this is a second or third line agent. It has adverse effects, potential drug interactions and need serum level monitoring. It is used in nocturnal asthma not controlled by high dose anti-inflammatory drug.

(e) *Zafirlukast and zileuton*. Both are used for treatment of chronic asthma in adult and children over 12 years. Montelukast is used in children 2 years and above[36]. Both are used as alternative in mild persistent asthma[3 ,56,57].

1.14.2. Step therapy for long term and quick relief of asthma in adults and children older than five years:

Here mild asthma is subdivided into mild intermittent and mild persistent asthma[36,37,56].

(a) Long-term control [56]*:*

Step1: Mild intermittent asthma. No daily medication needed.

Step2: Mild persistent asthma: Once daily medication: anti-inflammatory drug is needed:

- Low-dose inhaled corticosteroid or cromolyn or nedocromil
- Children usually begin with a trial of cromolyn or nedocromil.
- Zafirlukast may also be considered in patients 12 years old or sustained release theophylline is an alternative therapy.

Step 3: Moderate persistent asthma: two daily medications.

- Medium-dose inhaled cortecosteroid two daily medications.
- Low-to medium–dose inhaled corticosteroid and long acting bronchodilator.

Step 4: Severe persistent asthma: daily medications:

- High – dose inhaled corticosteroid and:
- Long –acting bronchodilator and:
- Oral cortecosteroid with daily dose not exceeding 60 mg.

(b) *Quick relief of symptom or exacerbations when* there are symptoms. Short acting inhaled $beta_2$ agonists as needed[56].

1.14.3. Management of acute severe asthma.

The goals of management are:

1. Assess severity of the attack.
2. Institute therapy to reverse bronchospasm.

3. Maintain oxygenation.

4. Continue or adjust therapy based on response.

5. Provide education and support to minimise the chance of recurrence.

PEFR: is measured to provide a baseline for therapy and latter to assess the response.

Oxygen: Initiate before completion of the initial assessment.

Nebulized salbutamol : given at 20 minutes' intervals and there after at hourly intervals depending on the response. In severe obstruction, nebulized salbutamol may be given continuously until obstruction is reversed or signs of toxicity supervenes.

Electrolytes imbalances: Modest hypokalaemia (due to salbutamol) does not require treatment except in patients who have hypokalaemia due to other causes.

Corticosteroids: should be started early as they reduce the need for hospitalization and may reduce the likelihood of death. Oral corticosteroids which be continued for at least a week. Inhaled therapy is started before oral therapy is stopped.

Level of care: the patient is monitored closely using, PEFR, arterial PCO_2, level of dyspnoea, used of accessory muscles and fatigue.

Intubation and mechanical ventilation: the decision is made before the patient is in extremis.

Discharge plan: patient education and management plan should be checked and amended. Patient should be told when to increase beta agonist therapy and inhaled corticosteroids, to initiate or increase oral corticosteriods and when to proceed for emergency care [71].

1.14.4. Follow up and patient Education.

Follow up is needed for good control and for assessing therapy strategy. Daily peak flow monitoring in those with moderate to severe persistent asthma and whenever exacerbations occur.

Education is important as regards regimen, controlling of the exposure to triggering factors, compliance with treatment and satisfaction[56].

1.15. Complications of asthma.

These include pneumothorax, status asthmaticus with respiratory failure, fixed (nonreversible) airway obstruction and death [36.37].

1.16. Natural history and Prognosis of asthma.

Among infants, 20% have wheeze only with upper respiratory tract infections, and 60% of them have no wheeze by age 6 years. This “transient wheezers” have no allergy, but have

abnormal lung function [36.52]. Early wheezers who have allergies are likely to have wheeze at 6 years and above. Children who start wheezing after 6 years of age are likely to be wheezing by 11 years.

Before puberty, asthma occurs more in boys, while adulthood asthma is common in females. A boy is likely to have a family history and may have other allergies than a girl. In boys, asthma remits in adolescence. Some young adult improve with time [59].

Children with mild asthma with onset between 2 years and puberty have a 50% remission rate and only 5% have severe asthma. Those with severe asthma plus atopic diseases are likely to have persistent disease, 95% become asthmatic adults. Some children who grow out asthma may have recurrence later in life[72].

Young asthmatic patients have good prognosis. A poor prognosis occurs if asthma develops before 3 years of age, unless it occurs solely in association with viral infections [36].

1.17. Prevention of Asthma:

Control of environmental factors can reduce the incidence of asthma. There are three levels of prevention[73].

1.17.1. Primary Prevention.

Environmental factors: Bronchial hyperresponsiveness and atopy may be amenable to prevention. Atopy and perennial allergens exposure are linked to asthma. Using the population based strategy of prevention [73], reduction in the mean level of exposure to a risk factor may greatly reduce the incidence of asthma in the population.

1.17.2. Secondary and tertiary Prevention: Secondary prevention reduces the morbidity and mortality in known asthmatics.

Allergens avoidance: Encasing of mattresses with occlusive covers reduce symptoms and bronchial hyperresponsiveness [56, 73].

Avoidance of environmental pollution: Avoidance of exposure to tobacco can reduce both the acquisition and aggravation of asthma.

Use of drugs in Prevention:

Anti-inflammatory drugs used according to asthma severity to improve bronchodilation and reduce inflammation [73].

1.18. Definition of allergic rhinitis.

Allergic rhinitis is defined as an inflammatory condition of the nasal mucosa characterized by anterior nasal symptoms of pruritus, sneeze, discharge and stuffiness. There may be an associated loss of the sense of smell and taste[74].

1.19. Prevalence of Allergic rhinitis.

Allergic rhinitis is the commonest allergic condition, which affects 20-30% of the population. Allergic rhinitis prevalence has increased in the past years [16,41]. The prevalence of hay fever was found to have increased in the United Kingdom (UK)[75]. In the United States, allergic rhinitis prevalence is 10-20% of the population [14]. In the UK, the prevalence of allergic rhinitis was found to be 14% in one study [16]. In another study of UK 18.2% had recent rhinoconjunctivitis [75]. In Aberdeen the prevalence was 11.9% among children [41]. In Australia the prevalence was 19% [9]. In Singapore, the prevalence was 44% among children [51]. In Saudi Arabia, the prevalences were and 12.1%, 17% and 24% in Dammam, Riyad and Jeddah respectively [47]. In Kenya the 11months is allergic rhinitis was 25.3% in children [49]. While the prevalence was 25.6% in Nigeria [50]. In Sudan rhinitis was reported in 2.5% and 0.8% children with current asthma and non asthmatic respectively [27]. The prevalence of childhood hay fever shows great variation throughout the world ranging from 1.4 to 39.7^{o} percent [45,75].

1.20. Epidemiology of allergic rhinitis.

Seasonal allergic rhinitis is a seasonal disease in which the timing and repeated occurrence are characteristic. For perennial allergic rhinitis, there are many confounding factors, which make it often difficult to differentiate allergic from non-allergic type clinically.

Allergic rhinitis begins in early childhood, so younger people have higher prevalence than adults. The diagnosis and symptoms drops with age [74], males and females are equally affected.

1.21. Pathophysiology of allergic rhinitis.

Allergic rhinitis is a result of nasal mucosal inflammation. Normally the nose acts as a filter device (for polluted air and filter particles, irritant gases and allergens) and also as a humidifier. Rhinitis results from a local defense mechanism that tries to bar irritants and allergens from entering the lungs.

Tissue mast cells and basophils may bind with immunoglobulin E antibodies in atopic predisposed individuals. IgE is produced against specific allergens. Exposure of the allergens to mast cells and the bridging of the bound IgE result in degranulation of mast cells. Mast cells can release mediators without the IgE mechanism with non-allergic stimulators like alcohol, fumes and odours. The mediators released without the IgE mechanism are vasoactive and bronchospastic. Eosinophils respond to these mediators and their rupture is associated with the destruction of the basement membrane. Like in asthma there is the early phase response and the late phase response [14,37,74,77].

1.22. Clinical Manifestations of allergic rhinitis.

In allergic rhinitis, sneezing in a prominent feature. Sneezing is parochysmal, rhinorrhoea; nasal obstruction; and itching of the nose, palate, pharynx and ears.

Typically there is bilateral nasal obstruction which results from boggy oedema of the mucous membranes.

Characteristic mannerisms occur in many patients. Examples include allergic salute, here the nose is constantly pushed up trying to relieve obstruction. A transverse crease called “allergic crease” results from allergic salute. The child may develop “rabbit nose” which are wrinkles on the nose. Nasal obstruction causes a bluish discoloration of the lower eye lids known as “allergic Shiners”. Dark circles under the eyes called “Dennie-Morgan folds” result from venous stasis due to nasal obstruction. Mouth opening is common. The “long-face syndrome” where the face is elongated is seen in children with chronic allergic rhinitis [14,34,75].

1.23. Patterns of Allergic rhinitis.

There are two varieties of allergic rhinitis:

1. *Seasonal allergic rhinitis* : is commonly known as hay fever. It affects both the nose and the eyes causing rhino-conjunctivitis, which is the commonest allergic disorder. Patients often have headache and are usually irritable. There is a post-nasal mucous drip. This type responds well to treatment [37,74].
2. *Perennial Allergic Rhinitis:*This variety is characterized by the symptoms occurring throughout the year. It is caused by allergens to which there is continuous exposure. Despite perennial allergic rhinitis resembling seasonal allergy variety, there are differences in symptoms, diagnosis and treatment options. Perennial allergic rhinitis usually starts in young children and may even do so at 2-3 years of age, but the peak incidence occurs in adolescents and young adults. The main symptom being nasal obstruction. Sinusitis and serous otitis media are common. There is frequent headaches and the sleep pattern is disturbed [41.74].

1.24. Triggering factors allergic rhinitis.

Certain triggering factors are associated with either seasonal or perennial type.

Perennial symptoms are triggered by indoor aero-allergens which include, house-dust-mites, cat dander, dog dander, indoor moulds, cockroaches, feathers, and climate changes.

Triggers of seasonal allergic rhinitis include outdoor allergens like grass pollen, trees pollen and outdoor mould spores [74].

1.25. Diagnosis of allergic rhinitis.

On the basis of history it is often easy to diagnose seasonal allergic rhinitis because of its season timing. Perennial allergic rhinitis may be difficult to differentiate from the non allergic varieties. In straight forward history, no investigations are needed. The complete haemogram shows eosinophilia. The IgE value is obtained to evaluate atopy. The normal value of IgE doesn't exclude allergic rhinitis and a high IgE value is suggestive.

1.26. Differential diagnosis of allergic rhinitis.

The differential diagnosis include mechanical obstructive causes, inflammatory causes, immune deficiencies and other causes [14]. The deferential diagnosis include:

- Adenoid hyperplasia
- Unilateral choanal atresia.
- Foreign body.
- Deviated nasal septum.
- Nasal polyps
- Neoplasms.
- Vasomotor rhinitis
- Neutrophilic (infectious) rhinitis.
- Sinusitis
- Cystic fibrosis
- Ciliary dyskinesia.
- Gastroesophageal reflux
- Rhinitis medicamentosa (rebound effect of overuse of topical nasal decongesants).

1.27. Treatment of allergic rhinitis.

There are two main categories of treatment namely avoidance of allergens and drug therapy[37,74,78].

Avoidance of allergens: Avoidance of allergens may improve symptoms and reduce the need of drug therapy. For many patients the removal of a trigger have a dramatic effect, but it takes time for appreciation. Control of house-dust-mite by use of mattress cover is useful. Pollen is difficult to avoid because of daily activities. The pet should be removed from the family.

Drug treatment: A stepwise management guidelines is recommended depending on the severity of the disease.

Step 1: Mild intermittent allergic rhinitis. Most patients have mild intermittent symptoms. Oral or topical antihistamine with few side effects and less drug interactions is the main stay of treatment [78,79]. for predominant nasal blockage, intermittent topical decongestants is the drug of choice.

Step 2: Mild persistent allergic rhinitis. Anti-inflammatory drugs may be started if avoidance and first line therapy fail. Sodium chromoglycate used for allergic conjunctivitis and topical nedrocromil sodium for mild persistent symptom.

Step 3: Moderate persistent allergic rhinitis. Topical corticosteroids are most effective particularly for nasal blockage [80]. Topical nasal decongestant may be needed initially. Topical corticosteroids can be started before pollen season to prevent symptoms. It also prevents recurrence of nasal polyps[78].

Step 4: Severe persistent rhinitis: this is disabling and needs urgent effective treatment. Short term oral prednisolone (30mg/day) for 14 days is effective. Improvement is maintained by daily 2-3 times topical corticosteroids.

1.28. Complications and prognosis of allergic rhinitis.

These include the following[37,77].

1- Epistaxis which is common.

2- Acute or chronic sinusitis.

3- Nasal polyps affect < 0.5 % of patients with allergic rhinitis [17,37].

4- Rhinitis medicamentosa.

There is no death due to allergic rhinitis but, allergic rhinitis is associated with severe morbidity.

1.29. Definition of Atopic dermatitis.

Atopic dermatitis is an inflammatory skin disease with chronically relapsing course, which is characterized by episodes of intense pruritus, papules, lichenification, and dry skin with susceptibility to cutaneous infections [81,82].

1.30. Prevalence of Atopic dermatitis.

Atopic dermatitis is a great global public health problem with a life time prevalence in children of 10-20% and a prevalence of 1-3% in adults. Its prevalence has increased over the past three decades in industrialized countries, but remains low in agricultural area like Eastern Europe, China and rural Africa. The prevalence are high in urban area and among higher social class [18,83]. Infants with atopic dermatitis tend to develop allergic rhinitis and asthma [19,37].

Reasons for the increase in prevalence include, high exposure to air pollution, small families with less exposure to infections, more pets, high maternal age, and a wider range of foods. The hereditary component being important [84].

In Sudan one study reported the prevalence to be 2.6% among children age 6-7 years and 12-15 years[27]. While in another study, the prevalence was reported to be 9.5% among children aged 2 months to 16 years [31].

In Singapore (1996) eczema was the least allergic disorder with a prevalence of 9.5% among children age 6-7 years and 13-15 years[51].

In Kenya (1996) the 12 months prevalence of atopic dermatitis was 14.4% among children[49]. The prevalence atopic dermatitis was 3.1%. in Nigeria[85].

In UK, Itchy flexural rash (eczema) in the past 12 months was reported to be 16.4% in children aged 12-14 years and 4% of children had all three symptoms of hay fever, eczema and wheeze[76,77]. In Aberdeen (Scotland) atopic dermatitis prevalence was 12% [41]. In south Africa the prevalence was around 7% [45].

1.31. Pathogenesis of Atopic dermatitis.

Atopic dermatitis results from the interactions of susceptible genes, host's environment, pharmacological abnormalities and immunological factors[17,86].

Genotypes and phenotypes: The risk of a child developing atopic disease is 70% if both parents have the same disease and 30% if the parents have different atopic disease. For atopic dermatitis a high concordance rate of 77% in monozygotic twins (15% in dizygotic twin) was found [87].

Biochemical Abnormalities: Patients with atopic dermatitis have dry skin which itches extremely to various stimuli. An abnormality in essential fatty acids metabolism may be linked to the dry skin and the disturbed cellular immune response.

The abnormal skin response to pharmacological stimuli are due to raised secretions of vasoactive mediators.

IgE dysregulation. There is dysregulation of IgE production. An abnormal lymphokine production is related to the immunological parameter in atopic eczema[86].

Immune response cell interactions dysregulation. Within the atopic eczematous skin, vigorous allergens directed immune response partly mediated by TH_2 cells are present.

House dust mite allergen. Reduction in house dust mite load leads to improvement in atopic eczema.

Food allergy. This is an uncommon trigger in atopic dermatitis[87].

Environmental factors seem to be as important as genetic factors in determining childhood eczema[83].

1.32. Clinical Manifestation of atopic dermatitis.

Atopic dermatitis mostly begins between 2 and 6 months of age. In some, it starts even before 2 months of age. It can start at any age. About 60% and 90% are affected at 1 year and 5 years of age respectively. Atopic dermatitis tends to remit by 3-5 years; and in some, mild to moderate disease may persist [37].

The clinical features include: itching, macular erythema, papules or papulovesicles, eczematous areas with crusting, lichenification and excoriation, dryness of the skin and secondary bacterial infections. There are three age-related stages [81,86,87].

Infantile phase: starts from 2 months to 2 years. The skin lesions affect both cheeks, but may occur anywhere on the skin. The extensor surfaces of the knees are affected when the child begins to crawl. The papules are itching, may exudate and become crusted. Lymphadenopathy occurs with secondary bacterial infections.

Childhood Phase: Its starts from 2 months to 12 years. it involves the flexures of the elbow and knees, the neck, wrist and ankles. The erythaematous papules tend to lichenify.

Adult Phase: This starts from puberty. There is lichenification of the flexures and hands. The lips, face, upper arms and back may be affected.

Atopic hand eczema: This is common in children. There is diffuse lichenified eczema of both hands. The nails are often involved resulting in pitting and ridging[81].

Associated findings in atopic dermatitis include: keratosis pilaris, accentuated palmar creases, lichenification, Dennie- Morgan folds, allergic shiners, transverse nasal crease, pallor around the mouth, nose and ears, white dermographism, cataract and keratoconus[87].

1.33. Triggering Factors of atopic dermatitis.

Many factors induce the exacerbations of atopic dermatitis: the triggers are[22,87]:

Contact irritants: These are: soap, solvents, wool clothing, mechanical irritants, preservatives and perfumes.

Aaeroallergens: like house dust mite, moulds, pollen and animal dander[88,89].

Infections: Microbial agents like staphylococcus aureus, pityrosporum, yeasts and candida, as well as viral infections.

Food allergy: This is caused by immunological mechanisms. Food allergy tends to lessen with age. Allergy to eggs and cows milk is transient while that to peanuts is persistent [22,87,90].

1.34. Diagnosis of atopic dermatitis.

Atopic dermatitis has a wide clinical spectrum ranging from mild form such as pityriasis alba or hand eczema to major forms like erythrodermic rash. Pruritus and chronic or relapsing eczematous lesion with typical shape and distribution are essential for diagnosis [18]. Atopic dermatitis is a clinical diagnosis [88].

Hanifin and Rajka adopted a diagnostic criteria consisting of major and minor characteristics. At least 3 major and 3 minor characteristics are need for diagnosis [18,87].

The ISAAC protocol however, adopts only 5 characteristics for diagnosing atopic dermatitis:

1- An itchy rash with relapses.

2- A rash anytime in the past year.

3- Typical distribution of the rash.

4- Clearing or not clearing of the rash.

5- Eczema ever diagnosed.

1.35. Differential Diagnosis of atopic dermatitis.

Other conditions which mimic atopic dermatitis include the following [18.37,86.88]:

Seborrhoeic dermatitis, scabies, primary irritant dermatitis, allergic contact dermatitis, infectious eczematoid dermatitis, ichthyoses, phenylketonuria, acrodermatitis enteropathica, hyper-IgE(Job) syndrome, histiocytosis X, Wiskott-Aldrich syndrome, X-linked agammaglobulinemia, and biotinase deficiency.

1.36. Severity of Atopic Dermatitis.

Lack of objective measures makes the classification of severity of AD difficult [88].

Many parameters such as: extents of skin involvement, the degree of erythaema, the extent, number and depth of excoriation and or erosions, the degree of lichenification, response to therapy and affection of daily activities. These parameters are difficult to gauge[88,91,92].

The investigator's global assessment (IGA) used 3 parameters: Erythema, papulation or infiltration, and oozing/crusting to classify AD into mild, moderate, severe and very severe types using a six points scale which ranges from 0 score (clear) to 5 scores (very severe) [92].

Mild AD. This has a score of 0-2 and is characterized by:

- Clean skin with signs of inflammation of AD (0 score).
- Just perception of erythema, papulation/ infiltration of the skin (1 score).
- Mild erythema, papulation/infiltration of the skin (2score).

Moderate AD: This has 3 scores and is characterized by:

- Moderate erythema, papulation/infiltration of the skin (3 scores).

Severe (very severe) AD: This has a 4-5 scores and characterized by:

- Severe erythema and severe papulation/infiltration of the skin (4 scores).
- Severe erythema, and severe papulation/infiltration with oozing or crusting of the skin (5 scores).

Hanafin used: (1) erythema (2) induration and (3) Pruritus as parameters for scores to grade response to topical agents. The minimum score was 0 and maximum score was 3 for each parameter. The total sum of the scores of the three parameters at minimum of the 0 scores to a maximum of 9 scores. AD was then classified into mild, moderate and severe types[93].

The ISAAC protocol classified the severity of AD on the basis of the person response to 2 parameters only:(1) Persistence of the rash and (2) the frequency of night awaking per week due to itchy rash[51,74].

1.37. Management of Atopic dermatitis.

The knowledge of the nature of the disease and avoidance of triggering factors are important in management [88].

General measures: use of emollients and avoiding irritants. House dust mite should be avoided. Food allergy leads to eczema in only 20% of the children [86].

Topical corticosteroids: these are the mainstay of therapy [18,86,88]. Fluticasone propionate cream 0.05% was said to be safe in treatment for 4 weeks even in children as young as 3 months [94].

Cyclosporin: This is approved for short duration to treat severe refractory disease [85,87,88].

Tractolimus ointment : For short duration is effective [18,82].

Antistaphylococcal antibiotics: These are useful in poorly controlled patients [87,95,96].

Antiviral drugs: used to treat herpes simplex infections [18].

Phototherapy. Natural light is useful, but sunlight and high heat or humidity trigger the disease. Ultraviolet (UV) A combined with UVB is superior to UVB therapy alone. Phototherapy with psoralen and ultraviolet A (PUVA) is restricted to patients with extreme severe disease and is not recommended in children[18,82,87]. The medium dose UVA_1 cold -light phototherapy is more effective than UVA – UVB phototherapy in treatment of severe atopic dermatitis[97].

Antihistamines reduce Pruritus, so systemic steroids in rarely used.

Interferon gamma: This down regulates TH_2 cell function with resultant improvement [18].

Other treatment such as the Chinese herbal therapy are said to be of benefit in severe disease [18,98].

1.38.Complications of Atopic dermatitis.

Atopic dermatitis has several complications:

Psychological implication: The disease disturbs the child's sleep pattern. The child may be barred from others and other children may tease him. These result in behavioural difficulties.

Growth retardation: This may be due to the disease only or both tropical and oral corticosteroids may cause stunting of growth.

Bacterial infections: Secondary bacterial infection with *staphylococci* and *streptococci* is an integral part of the disease as well as a trigger [(88)].

Viral infections: Herpes simplex viruses and vaccinia viruses cause acute generalized infections. Molluscum contagiosum infection is common.

Ocular abnormalities: These take the form of Dennie-Morgan fold, Keratoconjunctivitis, Keratoconus, cataract and rarely retinal detachment [(81)].

1.39. Natural history and prognosis of atopic dermatitis.

Atopic dermatitis (AD) improves with age. The majority of the children improve by age of 5 years, others will have lifelong problems.

About one third of children with AD develops asthma or hay fever. Children who develop AD before 6 months of age have a greater risk of developing asthma by 2 years of age.

Relapses of atopic dermatitis are less frequent and severe in adult life. Adults with past history of atopic dermatitis may develop chronic hand dermatitis following exposure to irritants or solvents.

Severe atopic dermatitis occur in children who develop the disease before 6 months and those whose disease is associated with food allergies.

A poor prognosis is associated with early age of onset, a family history of atopic dermatitis, associated asthma or allergic rhinitis, female sex, extensive dermatitis in childhood and persistent dry or itchy skin [37,81].

Justification

Asthma and other atopic diseases are the commonest causes of chronic ill health in children, giving rise to considerable morbidity, school loss, and health care costs.

There is a global increase in the prevalence of asthma and other atopic conditions. In the Sudan, few studies have been done on Asthma. The study of Asthma should include other

atopic conditions as most of these conditions overlap with each other.

OBJECTIVES

1. To determine the prevalence of asthma, allergic rhinitis and atopic dermatitis in school children.
2. To identify the triggering factors of childhood asthma, allergic rhinitis and atopic dermatitis and to evaluate their association with asthma.

3. To evaluate the clinical severity of asthma, allergic rhinitis and atopic dermatitis.

2. Materials and Methods

2.1. Study design:

Descriptive, analytic, cross sectional community based study.

2.2.Study area:

The study was conducted in basic schools in Khartoum Province. There were 185 Governmental basic schools in

the Province. A number of 22 basic schools were randomly selected from the 185 basic schools in the Province.

2.3. Study duration:

The data was collected from 10th October 2002, to 20th February 2003

2.4.Study population:

School children of 22 basic schools in the Province basic education schools. There were 185 Governmental basic schools in the Province.

2.5.Sample size:

The calculated sample size was 2172 children but 2176 students were included in the study.

The sample size was calculated by the formula:

$$N = Z^2 P \frac{(1-p)}{d^2} \times \text{Design effect}$$

Where:

N = Sample size.

Z = Statistical certainty(1.96) at 95% level of confidence.

P= Prevalence (13% = 0.13).

Q = 1-p (probability of failure)= 0.87.

d2= Designed margin of error = 0.02.

d= Design effect (1-2).

2.6. Inclusion criteria:

School children in the basic education schools aged 6-13 years. The age group for the basic schools is 6-13years.

2.7. Exclusion criteria:

Children less than 6 years old and those 14 years and above. Children who refused or whose parents or guardians refused their inclusion in the study.

2.8. Study technique:

2.8.1. Consent.

Written consent was obtained from the Authorities in the State Ministry of Education of Khartoum State. Consent was also obtained from the educational offices in the localities and from the headmasters of the 22 randomly selected schools. Informal consent was obtained from the student and from the parents or guardians.

2.8.2. Research team:

This included the author, headmasters and teachers of the selected schools, and a statistician.

2.8.3. Research tools:

An adapted ISAAC questionnaire with supplemented questions on triggering factors, family history of atopy, parental smoking, education and occupations were used. The ISAAC

questionnaire has been tested and verified, consistent responses to the first two questions (wheeze ever and wheeze in the past 12 months), had sensitivity of 85% and specificity of 91% and a positive and negative predictive values of 61% and 94% respectively [39,76]. The author however was unable to test and verify.

Eleven school were assigned for boys and 11 school for the randomly selected 22 schools. These schools were randomly selected for each age group. About 100 students were randomly selected from each school. All students of specific age group who gave their consent were issued the questionnaire. The sample was collected by multistage stratification sampling for gender, age group and the localities. The questionnaires were completed by the parents and the students at home and then were returned to the Headmasters or headmistress. The author then collected the questionnaires. The author directly interviewed some of the older children.

Children who had symptoms suggestive of asthma, allergic rhinitis and eczema, were examined and their physical signs were recorded. Their weights and heights were recorded. The PEFR were taken from children with a history of wheeze or asthma and were recorded.

2.9. Measurements:

The weights, heights of the children were measured and recorded. PEFR were measured from children with history of asthma and were reported.

2.9.1. Weight:

The child was weighed with light dresses and without shoes. The weight was measured in Kg and the reading taken to the nearest 0.1Kg. Using the Speedo Terraillon high precision measuring scale (made in Ireland) The weights were plotted on the centile charts. A child was said to be under weight if his/her weight was below the 3rd centile.

2.9.2. Height:

This was measured with the child standing straight, the head straight and looking horizontally, with the hands hanging by the sides and the lower limbs straight with the feet put together and without shoes. The height was marked on the wall and measured in cm and the reading taken to the nearest 0.1cm. The height was plotted on the centile chart. A child was considered stunted if his/her height fell below the 3rd centile.

2.9.3. PEFR:

This was measured by the Mini Wright Peak Flow Meter in L/min. Peak expiratory flow in the fastest rate at which air can move through airways during a forced expiration starting with fully inflated lungs. The PEFR was taken with the child standing up and holding the peak flow meter horizontally. The child was told to take a deep breath and seal his/her lips round the mouth piece and then blow out as fast and as hard as possible. After the child was familiar with the procedure and had successfully performed an example he/she blew the peak flow meter three times. The best of the three readings was recorded for the child. The reading were compared with the graph derived by John Cotes [100]. Using this graph the percentage of the predicted value was calculated. There is a similar reference values of lung function test in normal Sudanese children[101]. Asthma was defined as a variation of > 20% (decrease) of the predicted value.

2.10. Case definitions and severity.

2.10.1. Asthma definition: Current asthma was defined as a history of wheeze or symptom of asthma and a diagnosis of

asthma in the past 12 months while lifetime asthma was defined as a history of wheezing ever in the past.

2.10.2. Severity of asthma.

This was assessed by two approaches:

a. Severity according to self reported symptoms. Asthma was classified into three levels of severity using the ISAAC classification [51,73]. A scoring method of severity was used similar to that used by Zureik et al[102]. Five variables were used which included [61,74,102,103].

- Number of wheezing attacks in the past year: 1-3 attacks (mild), 4-12 attacks (moderate) and > 12 attacks (severe).
    - *Number of days of school absence:* 1-3 days (mild), 4-12 days (moderate) and > 12 days (severe).
    - *Nights woken by wheeze per week*: Nil (mild), < 1 night (moderate), >1 nights (severe).
- Interference of wheeze with daily activities: A little (mild), a moderate amount (moderate) and a lot (severe).
    - *Speech limitation during attacks*: Nil (mild), present (Severe).
    - Each of the first four variables were given three levels of increasing severity of scores

(1,2,3). The fifth variable had two levels scores (1or2). The total scores ranged from 5 to 14. The levels of severity was classified as:

- Mild asthma (5-7scores), moderate asthma (8scores) severe asthma (>9 scores).
- b. Severity according to the PEFR measurement.
- *Peak expiratory flow rate:* > 80% predicted (mild), 60-80% predicted (moderate), < 60 % predicted (severe).
- The severity of scores ranged from 1- 3 scores. Mild asthma (1score), moderate asthma (2scores) and severe asthma (3 scores).

2.10.3: Definition of allergic rhinitis.

This was defined as a history of rhinitis ever and rhinitis or a diagnosis of hay fever in the past 12 months [51,74].

2.10. 3.1. Severity of allergic rhinitis.

This was classified on the basis of interference with daily activities [51,74].

Mild allergic rhinitis: No or little interference with daily activities.

Moderate allergic rhinitis: moderate interference with daily activities.

Severe allergic rhinitis: A lot of interference with daily activities.

The classification had an increasing levels of severity scores. Levels of severity were:

Mild allergic rhinitis (0-1 score), moderate allergic rhinitis (2 scores), severe allergic rhinitis (3scores).

2.10.4. Definition of atopic dermatitis.

Atopic dermatitis was defined as the presence of a chronic itchy rash ever and chronic rash and a diagnosis of eczema in the past 12 months. Lifetime AD was defined as a chronic itchy rash ever with a typical distribution or AD diagnosis ever.

2.10.4.1. Severity of atopic dermatitis.

The severity was assessed on 2 parameters [51,74]. Scoring system similar to investigator's global assessment score [93], Hanafin simplified scoring [94], and the grading score of Rajka

and Langeland for severity of atopic dermatitis [104]. The greater the scores the severer the disease [105]. The parameters were:

a. Persistent rash without clearing in the past 12 months. This had a total scores of 1 score. No persistent rash (mild) 0 scores. Persistent rashes (severe) 1 score.

b. Number of nights kept awaken by rash per week in the past 12 months. This was expressed as: Never awoken (mild) = 0 score, awaken < 1 night a week (moderate)= 1 score, and awoken > 1 nights a week (severe) = 2 scores.

c. This had 3 levels of severity with increasing numbers of scores: mild = 1 score, moderate = 2 scores and severe = 3 scores.

The overall total scores ranged from 0 to 4 scores. The level of severity were: mild AD (0 score), moderate (1 score) and severe AD (3 scores).

2.11. Statistical methods:

The data was entered into the computer software. The statistical package for social sciences system(SPSS) was used for analysis. Chi-square test was used to compare the data. A

p.value of less than 0.05 was considered statistically significant.

3. RESULTS

3.1. Prevalence (percentage) of asthma and its related symptoms.

A total of 2176 students in 22 basic schools in Khartoum Province took part in the study. These 22 schools were randomly selected from the 185 basic schools in Khartoum Province. Both genders and age groups were equally distributed. The ages of the students ranged from 6-13 years. The schools were randomly selected for each age group. A self completed adapted ISAAC questionnaire with supplemented questions on parents' education, occupation, smoking, family history of atopy, and triggering factors were completed by the parents. The heights and the weights percentiles as well as the signs of atopy were included.

Table 1. Shows the cumulative and current (i.e. wheeze in the past 12 months) prevalence of self reported asthma and its related symptoms. Lifetime asthma (wheeze ever) prevalence was 13.1% while current asthma prevalence was 10.8%. The prevalence of exercise induced wheeze, persistent nighttime cough and asthma ever were 4.6%, 8.6% and 6.4% respectively.

3.2. Age and gender distribution of current childhood asthma.

Children 8 years old had the highest prevalence of 1.6% . Children 12 years old had a self reported prevalence of 1.5%. The lowest prevalence of 1.2% were reported by children six years old.

The over all male to female ratio was 1.1: 1 (Figure 1).

3.3. Distribution of asthma prevalence by residence.

The prevalence of asthma were 9.4%, 10.0%, and 10.8% for residence A, B and C respectively. Residence A included the *Gireif Garb* and the *Irkuweit* residential areas. Residence B included *Khartoum* 2, *El amarat, Sagana, Sahafa* and *Jebra* residential areas while C represented the *Hila El Jadida*, *Rimela, El-Alamab Nasir and Shagara* residential areas (Figure2).

There were no significant differences in residence distribution of asthma prevalences (P. value 0.32) as seen in Table 2.

3.4. Relation of asthma prevalence and parents' education.

The prevalence of asthma increased with increasing levels of education of the parents. About 235(10.8%) of children had asthma, and 42% of them had fathers who were university graduates (Figure 3a).

Regarding mothers' education, children whose mothers had secondary education had the highest prevalence of 37% (Figure 3b). Both fathers' education (P= 0.52) and the mothers' education (P.value 0.22) were statistically insignificant.

3.5. Relation of childhood asthma to fathers' occupation.

As shown in Figure 4, children whose fathers were skilled labourers and professionals had asthma more than children whose fathers' were unskilled labourers, unemployed, or fathers' had other occupations. This was found to be statistically insignificant (P= 0.06).

3.6. Relation of family history of atopic conditions and paternal/any household smoking to childhood asthma.

Paternal and maternal asthma were shown to be related to the risk of a child having asthma. Family history of AR and atopic eczema were also related to childhood asthma. Fathers, smoking as well as any household smoking were found to be confounding factors of childhood asthma.

These were found to be significant in all the variables or risk factors. Maternal smoking was excluded because very few mothers smoked (Table 3).

3.7. Triggering factors of current childhood asthma.

Respiratory infections (79.6%), common cold (79.1%) and in door/out-door dusts (73.2%), and cold air (70.6%) were the commonest triggering factors.

Insecticides and exercise were reported as triggering factors in 51.2% and 48.9% of the children respectively.

Foods triggered asthma in 9.8% of the children while drugs (4.3%) were the least triggering factors (Figure 5).

3.8. Signs and severity of asthma:

3.8.1. Signs of asthma severity.

The signs of severity of asthma were pigeon chest (28.9%), barrel chest (5.1%) and Harisons' sulci (1.7%). Pigeon chest was the commonest sign. No signs were found in 151(64.3%) children with asthma(figure 6).

3.8.2. Severity (%) of current childhood asthma.

Of the 235 children with asthma in the past 12 months, 69.6% has mild asthma, and 16.1% had moderate asthma about 14.3% of the children had severe asthma (Table 4).

3.9. Weights distribution percentiles of children with current asthma.

The weights of most children were between the 3rd and 97th percentiles. Few children had their weights above the 97th percentile. No child had his/her weight below the 3rd percentile. The weights were concentrated around the 50th percentile, but more children had their weights below the 50th percentile (Figure 7).

3.10. Heights distribution percentiles of children with current asthma

As seen in figure 8, the majority of children had their heights between the 3rd and 97th percentiles. Four children had their heights above the 97th percentile while two children had

their heights below the 3rd percentile. One child had his/her weight just below the 3rd percentile.

3.11. Distribution of asthma medications taken by male and female children.

Figure 9 shows that 75(31.9%) male children received asthma medication occasionally. About 28(11.9%) male children received asthma medication frequently while 14(5.6%) male children took asthma medication regularly.

As for females, 81(35.5%) of them took asthma medication occasionally, while 12(5.1%) received medication frequently, and 17(7.2%) received medication regularly.

In both sexes, 156 (66.4%) received medication occasionally, 40(17.0%) frequently and 31(13.2%) received medication regularly. Female children (17.2%) took asthma medication regularly more than male children (5.6%).

About 8(3.4%) children of both sexes did not receive any asthma medication in the past 12 months.

3.12. Prevalence (%) of allergic rhinitis and related symptoms.

Rhinitis ever was reported by 493(22.7%) of children. Current rhinitis was reported by 480(22.1%) and an associated allergic rhinoconjunctivitis occurred in 249(11.4%) of the children. Hay fever ever was reported by 31(1.4%) of children (Figure 10).

3.13. Age and gender distribution of allergic rhinitis.

As shown in Figure 11, allergic rhinitis was most prevalent among children 10 years old of both gender with prevalence of 3.6% (78 children). Children 11 years and 12years old had equal prevalence of 3.3% each.

Female children had allergic rhinitis more than males. Allergic rhinitis was most common among male children 12 years old.

In female the highest prevalence noted at 10 and 12 of years of age.

3.14. Triggering factors of allergic rhinitis.

Temperature changes and dusts were the commonest triggering factors accounting for 64% and 61.5% respectively. Cigarettes smoke was a triggering factor in 166(34.6%) of the children. Trees or grass pollens were the least triggering factors (14.8%).

The number of triggering factors were more than the number of children because a child could have more than one triggering factors (Fig 12).

3.15. Signs and severity of allergic rhinitis:

3.15.1. Signs of allergic rhinitis.

Oedematous nasal turbinate was the commonest sign (77.5%). Allergic shiner was seen in 155(32.3%) children. Horizontal nasal crease was only seen in one child. The actual number of children was 480, but the number of signs detected were 694 signs. This was because some children had more than one sign (Figure13).

3.15.2. Severity of allergic rhinitis.

As demonstrated in Figure 14,about 85% of the children had mild disease while 12% had moderate allergic rhinitis and only 3 % of the children had severe allergic rhinitis in the past 12 months.

3.16. Prevalence (percentage) of self reported atopic dermatitis and related symptoms.

Chronic itchy rash ever(not scabies) was reported by 176 (8.1%) children. Current atopic dermatitis was reported by 154(7.7%) children. Only 95(4.3%) children had atopic eczema ever (Table 5).

3.17. Atopic dermatitis and related symptoms prevalence (%) distribution according to father occupation.

The prevalence (%) of AD and related symptoms were higher in children of privileged parents like professionals merchants and least in children whose fathers were unemployed or fathers had other non specified occupations (Table 6).

3.18. Age and gender distribution of atopic dermatitis in the past 12 months.

Figure 15, indicates the age and sex distribution of children with AD. Male children had the highest prevalence at 9 and 13 years old, while female children had the highest prevalence at 11 years of age. The female male ratio of AD was 1.4:1.

3.19. Frequency distribution of triggering factors of atopic dermatitis.

Extreme weather (hot /cold) was the commonest triggering factor as reported by 71(46.1%) children. Sweating triggered atopic dermatitis in 39 (25.3%) a children. Again grass or trees pollens were the least triggering of factors (Fig.16)

3.20. Signs and severity of atopic dermatitis:

3.20.1.Signs of atopic dermatitis.

Of the 154 children who reported current atopic dermatitis, signs were found in only 121 children. The commonest sign was accentuated palmar crease (24.0%). Then lichenification of the antecubital and popliteal fossae (20.1%). Lichenification of the ankles or wrists (2.6%) were the least signs detected. About 33 (21.4%) children had no signs of atopic dermatitis (Figure 17).

3.20.2. Severity of atopic dermatitis:

Table 7 shows that, in children who had atopic dermatitis, 62.2% had mild AD, while 10.7% had moderate AD and 23.1% of the children reported that they had severe AD as assessed by clearing of the rash in the past 12 months and the number of nights woken per week by rash.

3.21. Gender distribution of asthma, allergic rhinitis and atopic dermatitis.

As shown in figure18, asthma was most common in males (51.5%) while allergic rhinitis and atopic dermatitis were more prevalent in females with percentages of 50.8% and 47.8% respectively for allergic rhinitis and atopic dermatitis in females.

The prevalences of allergic rhinitis and atopic dermatitis in males were 49.2% and 42.2% respectively.

3.22. Relation of asthma and related atopic conditions.

The prevalence of asthma was 10.8%. A total of 235 children had asthma. About 131 (55.7%) of the children had both asthma and allergic rhinitis. A combination of asthma, allergic rhinitis and atopic dermatitis were noted in 29 (12.3%) children

The prevalence of allergic rhinitis was 22.1%. About 74 (31.5%) of the children had both allergic rhinitis and atopic dermatitis (Figure19).

4.DISCUSSION

4.1. Prevalence of asthma, allergic rhinitis and atopic dermatitis.

In this study, the cumulative prevalence of childhood asthma was 13.1%, while current (12 months) asthma prevalence was 10.8%. This result is similar to that found previously in Sudanese school children who had asthma prevalence of 10.7% [27]. The prevalence of asthma in this study is higher than 8.3% in Jordan[47], 10.5% in Palestinian Authority [44], 10.2% in Kenya [47], 10.2% in Nigeria [50], 2.4% in Ethiopia [43], 5.9% and 4.7% in Zimbabwe [5], and Ghana [5], respectively. Like many developing countries, asthma prevalence in Sudanese children was less than those in developed countries like the United Kingdom [39], Australia [50], and Singapore [51]. The prevalence is comparable to (11.2%) in Saudi Arabia[47],and13% in south Africa [48]. Several studies [41,67,76] have shown increases in asthma prevalence in the same regions or area with time. The increase with time was not shown to be significant in this study, when the present prevalence was compared to a previous study [27].

Asthma was common in males than in females (male to female ratio of 1.1:1) in this study which is in line with many studies [41,44,51,76]. Regarding parents' education' asthma was common among children who parents where well educated and less in children with whose parents were illiterate. Asthma was also common in children whose fathers were either skilled labourers and professionals. It was common among children whose fathers were skilled labourers. This is because skilled labourers were likely to have a better earning because of private jobs. The 12 months prevalence of allergic rhinitis (AR) in this study among Sudanese school children in Khartoum province is 22.1%.

This prevalence is less than 24% in the UK study [16], 44% in Singapore [51], 25.3% in Kenya [49], and 25.6% in Nigeria [50].

The prevalence of allergic rhinitis in this study is higher than, 11.9% in Aberdeen children[41], 19% in Australia [9], but was within the prevalence range of 12.1- 24% in Saudi Arabia [47]. When compared with the previous prevalences of 2.5% and 0.8% of AR in children with asthma and non asthmatic children respectively [27], this study showed a significant increase in the prevalence of allergic rhinitis.

AR prevalence in Sudanese children is within the world's variation range of 1.4-39.7% [45, 75].

Atopic dermatitis prevalence in the past 12 months was found to be 17.1% in this study. This prevalence of life time atopic eczema prevalence of 22% in Australia [9] 12% in Aberdeen [41], 9.4% in Singapore [51], 16.4% in UK [75] 14.4% in Kenya [49], 14.2% in Leipzig (East Germany) [106]. The prevalence was comparable to that in South Africa [45], but more than that in Nigeria [85].

As in the UK study [83], atopic eczema was more prevalent among children whose fathers were professionals, merchants, civil servants, and skilled labourers. The prevalence was low among children whose fathers' were and skilled labourers or unemployed.

Atopic dermatitis was found to be more common among females, this is similar to studies [75,76].

4.2. Severity of asthma and asthma medication.

About 14.3% of the children had severe asthma, 16.1% had moderate severe asthma and 69.6% had mild asthma as assessed by symptoms combined with PEFR. The signs of severity was found in 35.7% of the children corresponding to severe asthma and no signs were detected in 64.3% indicating mild asthma.

Also in this study, 13.2% of children took asthma medication regularly daily which also indicates the percentage of children with severe asthma.

Mild asthma was the commonest variety of asthma in this study. This is consistent with the report by Chang et al [55]. Similarly milder asthma was common among children as demonstrated by Goh et al [51] and Shamssain et al [76]. The percentage of children with severe asthma was highest than that reported by Shamssain et al [76]. In this study, the percentage of severe asthma was less compared to a hospital based previous study [30]. This could be explained by better health services and health awareness in developed countries that control asthma better than in the developing countries.

It was found that 3.4% of the children with asthma in the past 12 months did not receive any asthma medication. In the UK study [39], a similar percentage of 3.4% of children who had four or more attacks of wheeze did not receive asthma medication.

4.3. Signs and severity of allergic rhinitis and atopic dermatitis.

The most common sign of allergic rhinitis was oedematous nasal turbinate, then allergic shiners. Atrophic nasal mucosa was the least sign detected.

Nasal polyps constituted only 1% and was noted in 5 children out of the 480 children who had current allergic rhinitis. This finding of low frequency of nasal polyps is consistent with the report that nasal polyps are very rare in children and they occur in patients who have allergic rhinitis with cystic fibrosis as noted by Becker [14] and Katz [77].

In this study, the severity of allergic rhinitis was 3% which is higher than that in UK children [75]. The study in Singapore

[51]reported a percentage severity of 3.3% which similar to the severity in this study although it is slightly higher. In both studies [51,75], severe allergic rhinitis was much less compared to moderate and mild allergic rhinitis. This study too had very small percentage of children with severe allergic rhinitis similar to the UK and Singapore studies [51,75].

4.4. Relation of family history of atopic conditions, parental smoking and childhood asthma.

The relation of family history of atopic conditions and smoking to childhood asthma was seen to be significant among children with asthma, a finding comparable to several other studies (Ponsonby et al [9], Dold et al [24], and Postman et al [25].)

Al-Frayh et al [47], and Soussan et al [61] studies showed higher prevalence of asthma among children with family history of asthma, allergic rhinitis and AD. Parental smoking or any household smoking was also associated with the risk of childhood asthma.

4.5. Triggering factors of asthma, allergic rhinitis and atopic dermatitis.

The most common triggering factors of asthma in Sudanese children in this study were common cold, respiratory infections, dusts, cold air, and exercise in a descending order. This finding is equivalent to Abuekteish et al [46], who found that respiratory infections and cold environment, exercise and dust were triggering factors in asthma. In another study [67] colds were the commonest trigger factor followed by exercise. Moreover, dusts, weather, air pollution and strong smell were the most found to be the prevalent triggers. [64], in a previous study [29], on triggering factors of asthma in Sudanese children, cold air and dust were commonest triggering factors.

In this study, temperature changes, dusts, insecticides, cigarette smoke and exhaust fumes were the most triggering factors in decreasing frequency for allergic rhinitis, while pollens were only a triggering factor in 14.8% of cases. This is in contrast to a report by Ross et al [16]. This could be explained by the fact that children in Khartoum, are likely not to be exposed to pollen as there are few trees and few gardens.

Regarding atopic dermatitis, extreme weather, sweating , wool clothing and skin infections were the commonest triggering factors. Foods were a triggering factors in only 17.5% of the children. Food allergy was said to be implicated in one third to one half of children with atopic dermatitis [87]; it was also reported that the role of food allergy in provoking atopic dermatitis was controversial [88].

4.6. Weights and heights of children with asthma.

There have been concern about growth failure in asthma [11,12]. In this study, the weights of all the asthmatic children were all above the 3rd percentile for weight. Only 2 children had their heights below the 3rd percentile. This results is consistent of Caffarelli et al [63], who found no significant difference in height and weight between children with asthma and control group. Powel et al [12], also noted no growth retarding effect on children with asthma.

4.7. Relation of asthma with other atopic conditions.

It is shown that, allergic rhinitis is the commonest allergic condition among Sudanese children, while atopic dermatitis is

the least atopic condition in this study. This confirms with Goh et al study [51], as well as the UK study [75].

In this study, the prevalence of asthma in children was found to be 10.8%. About 55.7% of the children with asthma had allergic rhinitis, while 16.2% of the children with asthma had atopic dermatitis combined. Only 12.2% on the children had all three conditions combined which is higher than 4% children having a combination of asthma, allergic rhinitis and atopic dermatitis as reported by Shamssain et al [76]. About 5.2% children had a combination of the three atopic conditions in study carried by Austin et al [75].

CONCLUSION

The cumulative and the 12 months prevalences of asthma were 13.1% and 10.8% respectively. Asthma was more prevalent in males. The peak of prevalence was in children 8 years old followed by those 12 years old.

There was an increase in asthma prevalence with increasing levels of parents' education, however, it was most prevalent in children whose mothers had secondary education. Asthma was most prevalent in children whose fathers were

skilled labourers and professionals. There was no significant difference in residence distribution.

Family history of atopy and smoking were strongly related to childhood asthma. About 14.3% of the children had severe asthma and pigeon chest was the most common chest deformity.

The weights and heights of most asthmatic children were between the 3rd and the 97th percentiles. Eight (3.4%) children with asthma did not receive any asthma medication.

The cumulative and the 12 months prevalence of allergic rhinitis were 22.7% and 22.1% respectively. It was most prevalent in females and among children 10 years old. About 11.4% of the children had associated rhinoconjunctivitis. Allergic rhinitis was severe in 3% of the children and oedematous nasal turbinate was the commonest sign.

The cumulative and 12 months' prevalence of atopic dermatitis were 8.1% and 7.7% respectively. It was most common in children whose parents were professionals and merchants. It was severe in 23% of children and the commonest signs were accentuated palmar creases (24%) and lichenification of the antecubital and popliteal fossae.

The most common triggering factors for asthma were respiratory infections, common cold, dust, cold air, insecticides and exercise, while temperature changes, dusts, insecticides, cigarette smoke and exhaust fumes for allergic rhinitis. For atopic dermatitis common triggering factors were extreme weather, sweating, wool clothing, skin infections and soaps.

In children with asthma, 55.7% of them had allergic rhinitis while 16.2% had atopic dermatitis. Allergic rhinitis and atopic dermatitis was found in 31.5% of the children. A combination of asthma, allergic rhinitis and atopic dermatitis was noted in 12.3% of the children.

RECOMMENDATIONS

1. There is a need to have specialized asthma clinics because it affects 10.8% of the children.
2. Doctors should educate parents and children about asthma with emphasis on avoidance of triggering factors and compliance with treatment.

3. As allergic rhinitis occur in 55.7% of children with asthma, doctors should treat allergic rhinitis concomitantly with asthma.
4. Parents who smoke should be advised not to smoke indoors as cigarette smoke is both and a risk and triggering factor for asthma.
5. Further studies on atopic conditions are recommended. These studies could include: aspects like the psychosocial impacts on children and their family; and the economic burden for instance.

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