THE PREVALENCE OF ALLERGIC RHINITIS IN COLLEGE STUDENTS AT KENYA MEDICAL TRAINING COLLEGE (KMTC) – NAIROBI

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DECLARATION

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I certify that this dissertation is my original work and that it has not been presented for approval in any other university

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DEDICATION

I dedicate this work to my mother, Mrs. Alice Gathiru, who through hard work and strong will saw us through our education after the sudden demise of our father.

I would also like to dedicate this work to my wife, Zipporah and son, Michael, for their patience, encouragement and support during my period of study away from home.

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ABBREVIATIONS

ACE	-	Anticholinesterase
AR	-	Allergic Rhinitis
ARIA	-	Allergic Rhinitis and its Impact on Asthma
CaMP	-	Cyclic Adenosine Monophosphate
CSF	-	Cerebrospinal fluid
ECRHS	-	European Community Respiratory Health Survey
FC_ER_1	-	High Affinity Ige Receptor
FESS	-	Functional Endoscopic Sinus Surgery
GERD	-	Gastrooesophageal Reflux Disease
HDM	-	House Dust Mite
ICR	-	International Consensus Report
IgE	-	Immunoglobulin E
IL	-	Interleukin
ISAAC	-	International Study of Asthma and Allergies in Childhood
KMTC	-	Kenya Medical Training College
KNH	-	Kenyatta National Hospital
MHC	-	Major Histocomaptibility Complex
NARES	-	Non-Allergic Rhintis with Eosinophilia
NPT	-	Nasal Provocation Test
OME	-	Otitis Media with Effusion
SPT	-	Skin Prick Test
TCA	-	Tricyclic Antidepressant
THO	-	Undifferentiated T Helper Cells
TH_1, TH_2	-	T Helper $Cell_1$, T Helper $Cell_2$
UON	-	University of Nairobi
US	-	United States

ABSTRACT

Background

Allergic rhinitis is one of the commonest atopic diseases world wide yet its epidemiology in Kenya remains sparse. Currently, there is only one questionnaire based study (ISAAC) in children documented

Objective

The primary objective was to determine the prevalence of allergic rhinitis in KMTC students, aged 18 – 50 years. The other objectives included: to determine the severity, pattern of symptomatology and the common aeroallergens involved in the group thus studied.

Method

The study was done in two steps. In stage 1, using a stratified random sampling, 423 students were screened for symptoms of allergic rhinitis based on ICR definition of rhinitis. In stage 2, the positive respondents (63 Students) were subjected to a physical examination and skin prick test to confirm allergic rhinitis.

Results

A point prevalence rate of 13% was reported with no sex or age predilection in the group thus studied. 81.8% of the students with allergic rhinitis had their daily activity affected to a certain degree. Sneezing (83.6%) was the commonest symptom and hypertrophied inferior turbinates (70.9%) the commonest physical finding. Patients with intermittent disease (73%) were the most, average age of onset was 15.2 years, seasonal peaks were in January, July and December and 36% of the students with allergic rhinitis had a family history of atopy. The commonest aeroallergen was the house dust mite (76.4%) and the least was Aspergillus Niger (1.8%).

Conclusion

Allergic rhinitis affects a significant proportion of the adult population with symptoms which have an impact on the lifestyles of these patients. The common aeroallergens are found within our immediate surroundings e.g. house dust mite, which can be controlled if patients are educated and proper, cheap environmental control measures are instituted.

1.0 INTRODUCTION AND BACKGROUND

1.1 INTRODUCTION

Rhinitis is defined as an inflammation of the nasal mucous membrane[1]. It is characterised by sneezing, nasal itchiness and rhinorrhoea. There are different types of rhinitis, some of which include:

(i) Infectious - due to bacteria, fungi, viral

(ii) Allergic

(iii) Vasomotor

(iv) Drug induced e.g. aspirin

(v) Occupational due to allergen at workplace

(vi) Hormonal rhinitis - related to puberty, pregnancy, some endocrine disorders e.g. hypothyroidsm.

The history of allergic rhinitis (AR) dates back to 1819 when John Bostock in London called his periodic affection of eyes and chest as catarrhus aestivus (summer catarrh)[2]. Later Charles Blackley of Manchester, a hay fever sufferer, confirmed that pollen was involved in causation of hay fever. He performed skin tests on himself using a sample of pollen and not only elicited a skin reaction but also provoked conjuctivitis, rhinitis and asthma.

AR is an IgE mediated hypersensitivity reaction to the nasal mucosa, but can also involve the paranasal sinuses since it is a continuous lining[3]. It may also be associated with symptoms in the eyes, palate and pharynx [4].

The old classification of AR used to group it into two clinical types; perennial and seasonal. Since some allergens regarded as seasonal in one geographical area were considered perennial in another and also due to reactivity to more than one allergen by some patients, this old classification has largely been abandoned[5]. The newer classification system based on ARIA guidelines (Allergic Rhinitis and Its Impact on Asthma) has been adopted[6]. It classifies AR according to its duration and severity of symptoms.

Table 1

Aria classification of AR

Intermittent	**Mild Rhinitis**
• Symptoms occur on less than 4 days in a week.	• No disturbances in sleep, leisure, school or work activities
• Or for less than 28 days at a time	
Persistent	**Moderate/Severe rhinitis**
• Symptoms occur on the majority of days of the week and more than 28 days.	• Disturbance to sleep, leisure, school or work activities.

1.2 BACKGROUND

1.2.1 ANATOMY OF THE NOSE

The nose is an important and the first respiratory organ which consists of two equal nasal cavities separated by a nasal septum[7]

Embryology

The nasal placodes which developed at the end of the 4th week of intrauterine life largely contributes to the development of the nose[8]

Gross Anatomy

The nose can be divided into the external nose, nasal cavity and the nasal septum for descriptive purposes.

a) External nose forms part of the face and is formed by cartilage and bones.
b) The nasal cavity extends from the external nose to the posterior conchae and is subdivided into two by the nasal septum[7,9]. The walls of each cavity are formed by a roof, floor, lateral wall consisting of three conchae and a medial wall[10].
c) Nasal septum is composed of a cartilaginous, bony and cuticular part.

The chief arterial supply to the nose is by the anterior and posterior ethmoidals, sphenopalatine, greater palatine and branches from facial artery[9]

The venous drainage is into the facial vein, pharyngeal plexus and pterygoid venous plexus[9].

The nerve supply is by the olfactory, anterior ethmoid, nasopalatine, posterior superior lateral nasal, greater palatine and anterosuperior alveolar nerves[9]

Lymphatic drainage of anterior half of nose drains into the sudmandibular nodes while posterior half drains into the retropharyngeal and upper deep cervical nodes[9]

Histology

The nasal cavity mucous membrane has 3 different histologic structures in 3 different areas namely- the vestibule, respiratory and olfactory area[4]

i. Vestibule : Is lined by modified skin with coarse hair and is a transitional area whereby keratinised epithelium gives way to pseudostratified respiratory epithelium[9].

ii. Respiratory area : Epithelium consists of ciliated pseudostratified columnar epithelium. It is thinner and has numerous goblet cells[11].

iii. Olfactroy area : This area occupies upper 1/3 of nasal septum and the area of superior concha. Its epithelium consists of the sustentacular, basal and olfactory cells. Bowmans glands are located beneath the olfactory epithelium and produce secretions that dissolve odoriferous gases.

1.2.2 PHYSIOLOGY OF THE NOSE

The chief functions of the nose includes; olfaction, filtration and humidification / warming of the air going to lungs[12].

Vocal resonance, self cleansing and protection of nasal mucous membrane by provision of moisture are other functions of the nose.

i. Olfaction

Odoriferous molecules which must be dissolved in mucous bind to receptors on cilia of olfactory neurons initiating a cascade of intracellular processes that leads to production of transmitted nerve impulses. Normally a concentration of an odoriferous substance has to change by approximately 30% before a difference can be detected[13].

ii. Filtration

The average human being inhales approximately 10, 000 litres of air in 24 hours[14], this contains a lot of particles. The vestibule vibrissae trap the large inhaled particles while the tiny particles and microbes are usually adhered to mucous. The mucociliary transport system transports these particulate matter towards the nasopharynx whereby they can either be expectorated or swallowed.

iii. Humidification

Air that reaches the lungs is at about 30^0C and at a relative humidity of 75-95%[12]. The vast arterio-venous shunts in the inferior turbinate plays an important role in air-warning.

The Nasal Cycle

There normally occurs a normal nasal blockage alternating between the two nostrils that most people notice especially at night when lying in bed. This involves a cycle of congestion and decongestion of the nasal lining and usually takes one to four hours although individual variations do occur[15]. This cycle is mediated by alterations in autoimmune tone of the nasal vasculature[16]

2.0 LITERATURE REVIEW

2.1 EPIDEMIOLOGY

Prevalence

Geographical differences in types, potency and overall aeroallergen burden may lead to varying prevalence of AR amongst countries and also within the same country[17]. A study done by Bauchan V et al[18] on the prevalence and rate of diagnosis of AR in Europe showed a prevalence of 17% in Italy and 29% in Belgium. Another study by Arnedo-Pena A et al[19] on time trend of prevalence of AR in school children in eight areas of Spain showed varied prevalence of rhinoconjuctivitis and nasal allergy with Madrid, Asturias and Bilbao showing higher prevalence than Barcelona, Castellon or Pamplona.

In addition to the above factors, the diagnostic criteria used in community surveys may also affect the reported prevalence, as shown by Wang et al[20].

World wide, the prevalence of AR has been estimated to be around 10-20%[3]. The prevalence of AR has been increasing over the last 3 decades[5], this has probably been due to the following (theories).

1. Atmospheric pollution, although not fully substantiated
2. Major change in gene pool predisposing people to excessive IgE production and thus increased expression of AR

A concurrent increase in prevalence of asthma has also been noted[5].

Age

AR is most common amongst 20-40 years[21] old persons, although it may occur in persons of any age[17]. The mean age of onset has been reported to be about 8 -11 years of age[17], whereas the peak age of AR has been reported to be 21 – 30years[21].

Sex

Some authors report no sex predilection[4,22,23], while others report childhood AR to be more common in boys than girls and others believe there is a male predominance[3].

Race

AR occurs in persons of all races with no racial predilection[22].

Morbidity and Mortality

AR per se is not a life threatening disease, but has significant morbidity from the disease itself, associated complications eg sinusitis and comorbid conditions like asthma. In fact some of the comorbid conditions and complications can be quite fatal eg severe asthma, anaplylaxis etc.

AR has also a substantial impact both on the economy and quality of life. Estimated worldwide cost on direct prescription medications has been quoted to be more than $6 billion per year[21]. Recent surveys in US reported more than 811, 000 missed workdays, 824, 000 missed school days and 4.23 million reduced activity days per year[4].

2.2 AETIOLOGY

The aetiology of AR is multifactorial, and includes both genetic and environmental factors.

2.2.1 Genetics

AR has been associated with a genetic predisposition. Atopic persons have a predisposition to develop allergic diseases which is genetically inherited. Recent evidence suggests autosomal dominant inheritance with a strong maternal influence[3]. A genetic linkage with a gene / genes on chromosome Hq has been implicated[1]. The chances of developing allergy in the children if one or both parents are allergic is 20% and 40% respectively[4]

2.2.2 Environmental factors

AR development also depends on exposure of the individual to environmental allergens. Inhalant allergens are usually the cause.

Montealegra et al[24] study data suggested that the type of environmental allergen one is exposed to (animal, plant or fungi allergen) in tropical areas, may not influence the clinical manifestation of allergic diseases especially allergic rhinitis. Environmental allergens can be subdivided into seasonal and perennial allergens for descriptive purposes:

i. Seasonal allergens

These cause symptoms in patients in particular seasons of the year when the allergen is plenty in the environment. The common causes of seasonal allergy include; grass pollen, tree pollen, weed pollen and fungi spores like aspergillus.

The dorminant seasonal allergen varies worldwide due to difference in geographical and climatic conditions e.g. commonest seasonal allergen in UK is grass pollen, USA, ragweed pollen and Japan, the Japanese Cedar[3].

ii. Perennial allergens

Min et al[25] in Korea showed an overall prevalence of perennial AR of 1.4%, the risk factors that were associated with an increased prevalence included; current urban residence, nasal septal deformity, chronic sinusitis with nasal polyposis and overcrowding; smoking showed no influence on prevalence.

Worldwide the commonest cause of perennial AR is the house dust mite (HDM) [3]. The species of which are; D. pteronysinnus, D. farinae and Euroglyphus maynei. HDM are found in areas with plenty of human skin scales vitz – beddings, carpets etc.

Experimental determination under laboratory conditions have shown optimal growth of D. pteronyssinus to occur at temperature of 25°C and 80% relative humidity of air[26]. The major mite allergen is the digestive enzyme cystein protease group 1 allergens eg Der P1 found in mite faecal matter[3].

Other perennial allergens include fur from domestic pets like dogs, cats, rabbits and also cockroaches.

2.2.3 Other allergens

a) Occupational allergens

These cause symptoms at the place of work and include:

- Flour in bakers
- Wood dust in carpenters
- Drugs in pharmaceutical workers or nurses
- Latex in surgeons, nurses

Buckland[27] et al showed that the prevalence of latex allergy in patients presenting with rhinitis is higher than in the general population.

b) Food and drugs

Food, although a rare cause of AR has been associated with it. Huang et al[28] showed that the prevalence of AR was associated with milk, liver and fruits. Liver was identified as the most significant predictor of rhinitis. Food induced allergy is more common in children than adults[3].

The mechanism of drug induced AR is unknown although a number of drugs have been implicated eg Aspirin, propranolol, ACE inhibitors

c) Pollution

Its role in causation of AR is controversial, but it is known to worsen symptoms in AR patients. It has also been implicated in the increase in prevalence of AR worldwide[29].

2.3 PATHOPHYSIOLOGY

Allergic rhinitis is a clinical example of a type I hypersensitivity reaction, in which an immediate hypersensitivity reaction occurs following exposure to an allergen[30]. This allergic response occurs in two phases [29]: -

i. Initial sensitization phase - This is characterized by IgE production and induction of a humoural response.

ii. Subsequent clinical disease (reactive phase) – it occurs as a result of repeated allergic exposures, and can further be divided into an early and late phase.

2.3.1 Sensitization phase

When genetically predisposed individual ingests or inhales an allergen, it is taken up by Antigen Presenting Cells: dendritic cells, which then migrate to lymph nodes and prime antigen specific undifferentiated T Lymphocytes (THO)[31]. THO cells in turn differentiate to either of the T helper cells (TH_1 and TH_2). It is thought that exposure of THO cells to IL – 4 stimulates its differentiation to TH_2, thus more IL-4 and IL-13 are produced by primed TH_2. IL-4 and IL-13 so produced promote production of specific IgE from B lymphocytes.

Before IgE production can occur, allergen specific B lymphocytes must bind to allergen, internalise, process and present its peptides in association with Major Histocompatibilty Complex (MHC) class II to TH_2. Two signals are involved for the preferential IgE production[3]: -

(a) IL-4, IL-13 produced by primed TH_2 act on B lymphocytes and promote class switching from Immunoglobulin M production[31].

(b) The binding of CD_{40} molecules on B lymphocytes and CD_{40} ligand on TH_2 cell surfaces is another signal for induction of IgE synthesis. IgE so formed gets bound via its Fc end to high affinity membrane receptors (FC_ER1) on tissue mast cells and circulating basophilis[30].

2.3.2 Reactive phase (clinical phase)

With subsequent exposure the polyvalent allergen gets bound to the Fab end of the IgE molecule and by cross-linking IgE molecules, mast cell activation occurs. Two major signals are involved in the release of mediators from mast cells[30],

(i) Transient increase of cAMP levels promotes the initial degranulation

(ii) Activation of phospholipase A_2 (PLA_2) initiate the synthesis of lipid based mediators from membrane phospholipids. Arachidonic acid generated from membrane phospholipids is metabolised by the cyclooxygenase and lipooxygenase pathways to give rise to prostaglandins and leucotrienes respectively.

Elevation of cytoplasmic free calcium is important for the assembly of microtubules and microfilament involved in intracellular transport of preformed granules to the cell surface[30]. The products of mast-cell degranulation include both preformed and newly synthesised mediators[31,32].

The preformed mediator includes;

Histamine : Vasodilation, increases capillary permeability, bronchoconstriction

Heparin : Anticoagulant

Tryptase : Activate C_3

B glucosamate : Splits off glucosamine.

Eosinophil and Neutrophil chemotactic factors

Newly synthesised mediators include;

i. Arachidonic acid metabolites;

(a) By lipooxygenase pathway

Leucotrienes C_4, D_4 - Vasoactive, bronchoconstriction, chemotaxis.

Leucotriene B_4 - Neutrophil chemotaxis and activation

Leucotriene E_4 - Enhances bronchial responsiveness and increase vascular permeability.

(b) By cyclooxygenase pathway

Prostaglandins D_2 - Vasoactive, Bronchospastic

Prostatglandin F_2 - Vasodilator, bronchospastic

Thromboxane A_2 - Spasmogenic

ii. Platelet Aggregating Factor - Increase vascular permeability, bronchoconstriction.

iii. Adenosine - Bronchoconstriction

iv. Bradykinin - Vasodilation, smooth muscle constriction

a) Early phase response

This occurs within 2 to 30 minutes after exposure to allergen and subsides within 1-2 hours. It is due to mediators derived from mast cells mainly histamines, tryptase, leucotrienes, kinins and prostaglandins[4]. It is characterized primarily by sneezing, nasal itchiness and rhinorrhoea.

b) Late phase response

This occurs within 2 to 8 hours, but may last upto 12 hours after exposure to antigen. It is mainly due to generated lipid mediators and infiltration of eosinophils, neutrophilis basophils, monocytes and T lymphocytes[30]. These inflammatory cells release their mediators

and thus prolong earlier reactions e.g. neutrophil and macrophage release lysosomal enzymes, lymphocytes release cytokines, eosinophils release eosinophil cationic proteins etc.

Eosinophils are prominent cells in nasal mucosa of allergic rhinitis patients. In a study done by Braunstahl et al it was found that the number of eosinophils in nasal and bronchial mucosa was significantly higher in rhinitis patients with or without asthma than in non symptomatic atopics[33]. The number of eosinophils in nasal mucosa usually increases following allergen provocation

A 'priming' effect in which subsequent smaller doses of specific antigens triggers a mucosal reaction has been observed [3,4,34]. This priming effect is thought to be due to an infiltration of inflammatory cells following initial exposure[3]. The mucosa also gets sensitized to other non-specific allergens to which it has not been exposed.

Other cytokines involved in the process of allergic reactions include;

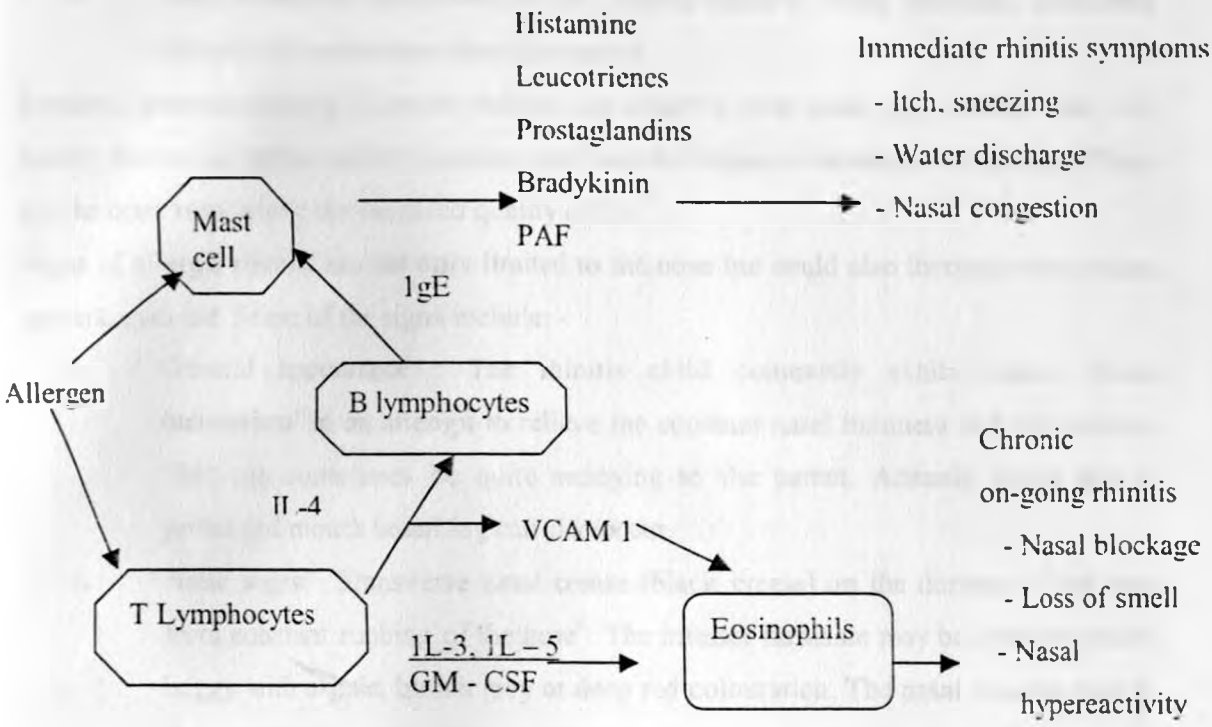
1L - 2 - has an autocrine action on activated T-Cell causing them to proliferate

1L – 5 - promotes maturation, chemotaxis, activation and survival of eosinophils

1L – 6 - promotes mucous production

Figure 1

Schematic representation of the mechanism of allergic rhinitis[3]



Vascular Cell Adhession Molecule-1(VCAM-1) - 1L – 4 stimulates the expression of VCAM – 1 on vascular endothelium which promotes eosinophil adhesion

GM - CSF - Granulocyte/macrophage colony stimulating factor

PAF - Platelet activating factor

CLINICAL PRESENTATION

The picture of symptomatology in allergic rhinitis depends on whether the disease is at its acute state or chronic (persistent) state. The dominant symptoms in the acute state include:

i. Paroxysmal sneezing of a frequency of about 10-20 sneezes at a time[4]

ii. Nasal obstruction associated with a watery rhinorrhoea and nasal pruritis

iii. Itchiness of the eyes, pharynx, palate

iv. Occasional bronchospasms

The dominant symptoms in the chronic (persistent) state include:

i. Viscus or purulent rhinorrhoea

ii. Less frequency of sneezing and allergic conjunctivitis

iii. Prolonged nasal congestion, nasal stiffiness and a post-nasal drip (PND)

iv. Chronic cough from frequent colds and PND

v. Other secondary symptoms include; loss of sense of smell and taste, associated sinusitis and eustachian tube dysfunction

Systemic symptomatology of acute rhinitis can occur in both acute and chronic state, are mainly due to the inflammatory response and include fatigue, sleepiness and malaise. These are the ones responsible for impaired quality of life[17].

Signs of allergic rhinitis are not only limited to the nose but could also involve other organs eg ears, eyes etc. Some of the signs include: -

i. General appearance : The rhinitis child commonly exhibit some 'facial mannerism' in an attempt to relieve the constant nasal itchiness and rhinorrhoea. This can sometimes be quite annoying to the parent. Adenoid facies due to prolonged mouth breathing can also occur.

ii. Nasal signs: Transverse nasal crease (black crease) on the dorsum of the nose from constant rubbing of the nose[4]. The inferior turbinate may be hypertrophied / boggy with a pale, bluish grey or deep red colouration. The nasal mucous may be thin, watery in acute cases or thick and purulent in chronic cases. Nasal polyps may occur secondary to acute rhinitis[4,22,23].

iii. Aural signs : Signs of otitis media with effusion secondary to eustachian tube dysfunction; air – fluid level or bubbles in middle ear. Signs of eczematoid otitis externa may be observed.

iv. Ocular signs : Conjuctival injection, cobblestoning of conjuctiva and excessive lacrimation may be seen in patients with associated allergic conjunctivitis. Ocular venous stasis results in allergic shiners (dark discolouration below lower eyelids and Dennie-Morgan lines (creases) in lower eyelids[23].

v. Throat signs: Features here include cobble – stoning of posterior pharyngeal wall due to follicular lymphoid tissue hypertrophy. Malocclusion of teeth and a high arched palate due to chronic mouth breathing can occur

vi. The risk of asthma in acute rhinitis patients has been discussed and furthermore some of the mediators released eg histamine, leucotrienes have a bronchospastic effect, thus it is necessary to examine the chest for rhonchi (bronchospasms)

vii. Other evidence of atopy should also be sort for eg atopic dermatitis[23]

Differential diagnosis of AR

These can be classified as follows:

i. Ciliary defects eg cystic fibrosis

ii. Rhinitis eg vasomotor, infective, hormonal, atrophic etc

iii. Mechanical obstructive causes eg deviated nasal septum, adenoids, foreign body.

iv. CSF rhinorrhoea

v. Others eg Agammaglobulinemia[22], aspergillosis, sinusitis, GERD, granulomas

Complications / cormobid conditions

Complications may follow allergic rhinitis per se or prolonged use of medication for it's treatment. These include; chronic fatigue, irritability, insomnia, poor concentration, drowsiness, OME, Asthma, Nasal polyps, orthodontic problems etc.

Greisner et al found out that AR often precedes (45%) or occurs at the same time (35%) as Asthma[35].

INVESTIGATIONS

The most important investigation in allergic rhinitis are laboratory tests which are done majority of times to confirm atopy. Imaging has a limited role and is not specific to allergic rhinitis. For instance, a CT scan will only show the presence of sinusitis, hypertrophied inferior turbinates, nasal mucosal thickening or nasal polyps which are not specific for the disease.

Laboratory test include;

i. A peripheral eosinophilia picked up in a haemogram is not diagnostic of allergic rhinitis. Other causes of eosinophilia include helminthiasis

ii. Nasal smear for cytology : Smear taken during active clinical disease are stained with Giemsa or MayGrin Wald stains[3]. Nasal eosinophilia is a feature of allergic rhinitis but not diagnostic. NARES (non allergic rhinitis with eosinophilia) also exhibits nasal eosinophilia.

iii. Nasal provocation tests: It is a challenge test to the nasal mucosa with an implicated allergen to see if nasal allergic symptoms will be triggered.

iv. Skin tests: These include prick, scratch and intradermal. The prick test is preferred and has largely replaced the scratch test. It is considered a standard allergy workup in

many centers, and depends on formation of a wheal and flare secondary to interaction of allergen and sensitized masts cells in skin[23]

The factors considered to affect the response in skin tests include;

Volumetric potency of the antigen, age and race of the patient, the distance between injection sites and time of day of testing, reactivity of skin and medication like antihistamine, anticholinergics, TCA and H_2 receptor antagonists. Therefore because of these factors a positive and negative control are usually added in the tests[23].

v. IgE count test

2.5.1 SKIN PRICK TEST (SPT)

This is the most commonly used skin test, and measures specific IgE attached to the mast cells on skin. It is usually the first recommended test and gives information on type of allergen a patient is sensitive to. It can be used in detecting both inhalant and ingested allergens. SPT is a relatively safe test, a retrospective study involving 18,311 patients showed 6 mild reactions over a period of 5 years[36], while another survey of allergy specialists reported

6 fatal reactions from 1945 to 1986[37]. A study done by Erickson of 939 subjects with allergic airway diseases showed that screening methods employing SPT using only 3 or 4 allergens could be used in detecting atopy in subjects with allergic airway diseases[38]. Its advantages are;

- It is a rapid and safe test[23]
- Simple and give quick results thus can be used as a screening tool for atopy in epidermiological studies[23]:
- Cheap
- Has a high specificity and sensitivity[22]
- Wide variety of antigen can be tested, both inhaled and ingested especially in multisensitive patients
- The procedure is virtually painless and is thus acceptable to children
- Glycerinated stock solutions have been noted to be more stable than the aqueous solutions used for intradermal tests.

Disadvantages

Are quite few and include;

- False negative and positive results, but this depends mainly on the technique of the performer

- Low grade sensitivities could be lost
- Does not allow quantitative analysis of sensitivity to allergens

2.5.2 Intradermal skin tests

These are of two types, namely: -

a. Single dilution intradermal testing

This type does not permit accurate quantitative assessment of sensitivity to antigen

b. Progressive dilution intradermal testing (skin end point titration)

Allows both quantitative and qualitative assessment of sensitivity.

2.5.3 IgE counts test

These measures concentration of specific IgE circulation in blood stream[3,4,25]. They include:

- RAST -Radioallergosorbent test
- ELISA - Enzyme linked immunosorbent assays

IgE counts tests although less sensitive than SPT are indicated when there is a risk of anaphylactic shock, where extensive eczema makes SPT impractical and when antihistamine medication or B-blockers cannot be stopped for various reasons before SPT.

Other non-validated test for allergic rhinitis include; basophil histamine release test, cytotoxic tests and leucocyte antibody test for related antigens.

2.6 TREATMENT

Modalities of treatment of AR include the following;

(i) Environmental control and allergen avoidance
(ii) Pharmacotherapy
(iii) Immunotherapy
(iv) Surgical intervention (minimal role)

2.6.1 Environmental control and allergen avoidance

These are aimed at preventing sensitization of an individual and also prevention of occurrence of an allergic reaction in response to IgE – antigen interaction by protecting from re-exposure to the allergen.

Some of the measures partaken include providing covers for mattresses and pillows, vacuuming carpets, use of arcaricides etc, as a way of controlling HDM exposure.
Animal allergens can be put at bay by confining pets outside or cleaning them frequently.

During pollen season one could try reduce outdoor activities or keep windows at home and the car shut. It is important to note that although these allergen avoidance measures are effective, their practical implementation can be difficult. Therefore, it is crucial to educate patients to understand the significance of such avoidance / environmental measures.

2.6.2 Pharmacotherapy

The following groups of drugs are used in the treatment of AR:

i. Antihistamines

These are H_1 receptors antagonists. The newer 2nd generations antihistamine eg cetirizine are less sedating compared to the older 1st generation antihistamines[29] eg chlorpherinamine. This is because the newer drugs are more H_1 receptor specific and penetrate blood brain barrier poorly[39].

Recently, metabolites of antihistamines eg disloratidine, fexofenadine have been used due to their safety profile. Antihistamines are effective in controlling sneezing, rhinorrhoea and nasal itchiness but less effective in nasal blockage[3,6].

ii. Glucocorticoids

They have anti-inflammatory activity and can suppress many stages of the allergic inflammatory repsonse, thus attenuating the production and release of various mediators[6].

Topical nasal steroids are the ones commonly used since they act locally with no systemic effects. Glucocorticoids are effective at controlling all symptoms of AR[6].
Examples of intranasal glucocorticoids include – buclesonide, beclomethasone, fluticasone, mometasone.

iii. Decongestants

These include both oral eg ephedrine and intranasal eg oxymetazoline

iv. Chromones (intranasal)

Sodium cromoglycate is a mast cell stabiliser[5,39]. It is also effective at controlling all symptoms of AR[29].

v. Anticholinergic (intranasal)

These are muscarine receptor antagonists and are effective in controlling rhinorrhoea.

vi. Antileucotrienes are leucotriene receptor antagonists eg monteleukast, zarfilaurkast.

2.6.3 Immunotherapy

It is a form of hyposensitization in which an allergen is given in gradually increasing doses until symptoms are relieved or a maximum dose is reached. It suppresses formation of specific IgE and increases titers of specific IgG antibody which act as blocking antibodies[4,23].

Hyposensitization has been shown to be effective in treatment of pollen induced allergic rhinitis[3,5].

Some of the indications of immunotherapy include[3,23]:

i. Failed pharmacotherapy or its intolerable side effects and failed allergen avoidance measures

ii. When a limited spectra of allergen sensitivity is involved, preferably sensitivity to one or two allergens

Contraindications include,[3,23].

i. Patient with autoimmunie disorder, immunosuppression, asthmatics and those on B-blockers. It is difficult to control anaphylaxis incase it occurs in patients on B-blockers.

ii. Patients with multiple antigen sensitivity.

2.6.4 Surgery

Has a minimal role in the management of AR[12], but compliments medical treatment eg turbinoplasty can be done to relieve obstructive symptoms of hypertrophied inferior turbinates, FESS in management of nasal polyps and sinusitis, deviated nasal septum corrected etc.

ARIA has designed a step wise approach for the treatment of allergic rhinitis; ARIA treatment guidelines[6].

2.7 PROGNOSIS

Most patients can live normal lives, despite the symptoms without medical treatment. Those who seek treatment respond well to intermittent symptomatic care.
Symptoms generally begin to regress at around the 5th decade[22].

2.8 RELEVANT STUDIES DONE

The prevalence rate studies of AR have been carried out worldwide using different methodologies. When interpreting prevalence data it is therefore important to take into account that reported figures may be biased by differences in classification, diagnostic methods and increased awareness of this condition[40].

Many questionnaires have been designed and used in epidemiological studies of AR, some of which include; ISAAC, ECRHS, Middleston Diary, Dirksen etc. ISAAC is a standard questionnaire used in epidemiological studies in childhood but no such international equivalent exists for adults.

2.8.1 Prevalence

World wide the prevalence of AR has been estimated to be 10-20% as discussed earlier[3]. Some studies have been based only on questionnaires while others have been backed by objective evaluation eg SPT, IgE counts etc.

Vichyanond et al[41] using ISAAC phase 1 protocol questionnaire on 3631 randomly selected university students from 6 universities in Bangkok aged 16 – 31 years found a prevalence of 26.3%. Huurre et al[42] in a follow up survey of Finnish urban age cohort (1967 birth cohort) from age 16 to 32 years found the prevalence of AR rose from 17.5% to 26% (Males from 18.7% to 27.8%, females 16.2% to 24.5%. Greisner et al[35] found a cumulative prevalence of self reported seasonal AR to be 41.5% while non-seasonal AR was 14% in former university students. Shahar et al[43] reported the prevalence of self-reported AR in an adult population in Israel to be 14%. Jones et al[44] also in a questionnaire based study in Nottingham for over 14 years old, reported a prevalence rate of 19.6%. Questionnaire based (self-reported) studies

although widely used lack in ability to differentiate non allergic from allergic rhinitis, ie lack an objective measure.

Several studies on AR have been done with the inclusion of an objective measure thus giving a more reliable and true state of prevalence. Bauchan et al[18] in a subset of subjects screened positive for AR performed a clinical diagnosis, specific IgE test along a disease specific questionnaire in adults in Europe. They reported a prevalence range of 17% in Italy to 29% in Belgium. Ciprandi et al[45] investigated 18 years old Lingurian conscripts in Italy by history, clinic visits, SPT etc and got a prevalence of 2.2%. Linnerberg et al[46] in their study on prevalence of skin test positive AR in Danish Adults reported a prevalence of 22.5%. Fedoseev et al[47] examined positive questionnaire respondents aged 16 to 98 years of St. Petersburg clinically, functionally and allergologically and reported prevalence of 9.9%. Olivieri et al[48] using a screening questionnaire, SPT and IgE determination in a population aged 20-40 years got an overall prevalence of self reported AR of 15.9% and a confirmed AR prevalence of 12.5%. AR commonly affected women above 35 years and men below 35 years of age. Ogino et al[49] studied nasal allergy in medical students of Osaka university, Japan from 1983 – 1987 using questionnaire, NPT and intradermal skin tests and reported a prevalence rate of 30%.

These studies although objectively done still shows a wide variation of prevalence rates. This could be due to differences in methodology employed and geographical factors. IgE counts used in some of the studies are usually less sensitive compared to SPT and relatively expensive to use in epidemiological studies. Aqueous solutions used for intradermal tests are also known to be more unstable compared to glycerinated solutions used for SPT, thus a risk exists of diminished volumetric potency of the allergens tested.

2.8.2 Symptomatology

Jones et al[44] reported a prevalence of nasal obstruction to be 16.9%, runny nose, 19.8%, 7.1% reported episodes of sneezing bouts and 13.7% had rhinosinusitis. Wang[20] reported prevalence of sneezing as 15.8%, rhinorrhoea, 11.7%, itchy nose, 10.6% and nasal blockage, 10.2%. Cirillo et al[50] reported sneezing as the most common nasal symptom in Italian conscripts. Gabriel Mhidze[51] in Tanzania found turbinate hypertrophy as the commonest presentation 20.8%) and epistaxis as the least common (0.7%) in primary school children.

2.8.3 Severity of AR

Montefort et al[52] reported severity of AR in maltese 13 – 15 years old as follows;

Not affecting daily activity 31.8%, daily activity affected a little 43.2%, daily activity affected moderately 13.7% and daily activity affected a lot as 2.9%.

2.8.4 Aeroallergens

Ogino et al[49] in their study on Japanese medical students found HDM and Japanese cedar the main allergens with a positive rate of 66.4% and 51.0% respectively. Baratawidjaja et al[53] in evaluating prevalence of regional aeroallergens using SPT in patients with asthma or allergic rhinitis in Indonesia, reported the highest sensitization rate to HDM (77.5%), followed by pollen and least fungal spores. AR patients were particularly highly sensitized to HDM. Gabriel Mhidze[51] in Tanzania using SPT found goat hair to be the commonest allergen (32.2%), followed by maize pollen (29.8%) and the least common was straw dust (11.4%) Raukas et al[54] reported the most prevalence sensitization using SPT amongst adults in Tallinin to be German Cockroach (15.5%). Monte Alegra[24] reported in patients with asthma, AR and atopic dermatitis, the prevalence of SPT to animal allergens to be highest followed by plant and fungal allergens.

2.8.5 Local data (Kenya)

The epidemiology of AR in Kenya is sparse just as is in the rest of Africa. Esamai and Anabwani[55] did an ISAAC phase 1 protocol study in Uasin Gishu district in13 – 14 years old primary school children. They found a cumulative prevalence of 32.4% and a prevalence rate of 25.3%. This study lacked an objective measure and therefore the figures obtained are possibly inclusive of both allergic and non allergic rhinitis.

Currently no documented literature is available concerning the prevalence of AR in the adult population in Kenya.

3.0 METHODOLOGY

3.1 GENERAL OBJECTIVE:

To determine the prevalence of allergic rhinitis among college students at the Kenya Medical Training College, KMTC.

3.2 SPECIFIC OBJECTIVES

1. To determine the prevalence and severity of AR in College Students at KMTC – Nairobi and correlate this to age and sex.
2. To determine the presentation and pattern of symptomatology of AR
3. To determine the common aeroallergens involved in the aetiology of AR in students thus studied.

3.3 STATEMENT OF PROBLEM

AR is one of the commonest atopic diseases worldwide, in USA has been reported to be the 2nd commonest after Asthma[21].

In KNH ENT clinic, a significant number of patients are being followed (managed) for AR. Despite AR being known to be a significant cause of morbidity and having a substantial economic impact[5], its epidemiology in Kenya is not well known.

3.4 JUSTIFICATION

The following reasons justify this study:

(i) The epidemiology of AR in Kenya is sparse. Currently only one questionnaire based (ISAAC) study in children has been documented. This study included an objective measure (SPT) in determining the prevalence of AR in college students at KMTC aged 18 – 50 years, which represents the wider adult population in the country.

(ii) Information will be relevant to Public Health Officials and clinicians in educating people on proper, effective and cheaper environmental control measures and allergen avoidance.

(iii) Once the prevalence of AR is known (using objective measure) further studies can be designed using the prevalence as a basis for calculation of sample size e.g surveys on economic and lifestyle impact of AR, Socio-dermographic factors studies etc.

(iv) Information obtained will also assist in establishing management protocols for AR.

3.5 SETTING

- Kenya Medical Training College (KMTC), Nairobi.

3.6 STUDY DESIGN

* The study was a descriptive cross-sectional study.

3.7 STUDY POPULATION

The study population was medical students from KMTC, Nairobi. KMTC is situated in the City of Nairobi just adjacent to the Kenyatta National Hospital. It offers certificate, diplomas and higher diploma courses in Health Sciences. There are about 15 different courses offered by the college including; nursing, clinical medicine, pharmacy etc.

According to the registrar's office the population of students is about 3000, with an age range of between 17 to 50 years. Majority of the students are secondary school leavers aged between 17 and 25 years. The remaining older ones are mainly in-service students. The choice of KMTC students as the study population was based on the following reasons: -

(i) The study group is an adult population

(ii) KMTC being a medical college, more reliable answers / responses to the questions from the students concerning symptoms, history of family allergies, triggers of allergies etc. were expected.

(iii) KMTC was a more cost and time effective place to carry out the study due to its vicinity to UON / KNH. A wider study would require more resources and time

3.8 CASE DEFINITION

Students with two or more of the nasal symptoms (based on International Consensus Report (ICR) rhinitis definition [20]); nasal obstruction, sneezing, nasal pruritis, watery rhinorhoea with or without conjunctivitis on most days and a positive SPT were considered to have AR

3.9 SAMPLING TECHNIQUE

- Stratified random sampling was used in the collection of data
- The students were stratified into 13 groups according to their departments and a simple random sampling done to select students from each department.

3.10 SAMPLE SIZE DETERMINATION

The minimum sample size required was calculated from the Kish formula as follows:-

$$n = \frac{Z^2 (P(1-P)}{d^2}$$

n = Sample size to be determined

Z = 1.645 at 95% C.L.

P = Prevalence of AR

d = Precision required by the investigation, in this case 0.05.

Because P is unknown, the recommended P of 50% or 0.50 was used.

$$n = \frac{(1.645)^2 \times 0.5.(0.50)}{(0.05)^2}$$

n = 271

Inclusion criteria

- Students aged 18-50 years
- Completion of a questionnaire
- Consent.
- Not to have taken medications as follows
    - 1st generation antihistamines for more than 3 days
    - 2nd generation antihistamines for more than 10 days, Astemizole for more than six weeks
    - H_2 receptor antagonist e.g. ranitidine - stopped on day of SPT
    - Not to be on adrenergic receptor antagonist or β-blocker
    - Medications with antihistamines properties e.g. TCA, anticholinergic stopped before SPT - on day.

Exclusion Criteria

- Nasal infection
- Structural (nasal) abnormalities
- Skin diseases e.g. eczema
- History of severe anaphylaxis
- Where unusual and rare allergies suspected eg latex
- Reactivity to negative control (SPT)
- Refusal to participate in SPT
- Food allergies
- Pregnancy
- Patients on immunossuppressive therapy e.g Steroids

3.11 DATA COLLECTION

3.11.1 Equipment and Materials

- Standardized commercial glycerinated extract purchased from a single vendor.

Seven allergens were tested including; Dermatophagoides Pteronyssinus (HDM), Bermuda Grass Pollens, Cockcroaches, Cat fur, Dog hair, Penicillium Notatum and Aspergillus Niger.

- Sterile lancet/needle
- Spirit, cotton wools, tissue paper
- Marker pen
- Controls histamine 1mg/ml, Normal saline
- Torch, nasal speculum, otoscope, stethoscope
- Adrenaline/Branular/injection needles/syringes
- Examination Gloves
- Coolant to maintain Allergens temperature at $2^0 - 8^0$ c

3.11.2 Technique of Data Collection

At the time of the study, only 13 departments of the 14 were available for the research. In one of the departments (Departments of records), the students were not available because they were either doing exams or had just completed and were scattered. The distribution of the students per department was as shown below;

Table 2

Department	Number of students
Pharmacy	460
Nursing	546
Clinical medicine	399
Orthopaedic technology	66
Community oral health	110
Physiotherapy	126
Dental technology	84
Occupational therapy	127
Medical imaging	118
Medical laboratory	183
Medical engineering	60
Environmental health	102
Health education	41
TOTAL	2402

Proportionate distribution of number of students per department for screening was used and simple random sampling done to select students per available class or classes allocated by the head of department. The study was done in two steps;

Stage 1: Screening step.

Using a questionnaire (Section A) 423 students were interviewed by the investigator and divided into two groups; the symptomatic and asymptomatic.

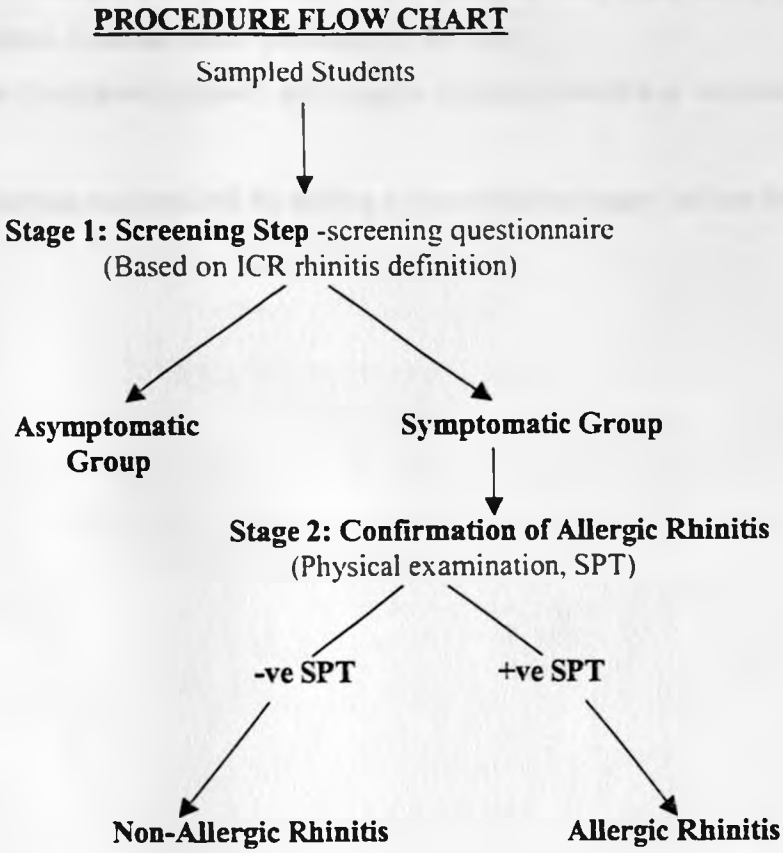
Sixty three students had two or more of the nasal symptoms (based on ICR definition[20]); nasal obstruction, sneezing, nasal pruritus, watery rhinorhoea, with or without conjuctivitis on most days and were considered to have symptoms of rhinitis.

These students proceeded to answer section B of the questionnaire which addressed the pattern of symptomatology,type of allergens that triggered symptoms, family history of atopy, age of onset of symptoms and significant medical history of the student. They then proceeded to stage 2.

Stage 2: Confirmation of AR.

After a relevant physical examination and SPT, 55 out of the 63 students were confirmed to have AR. The important physical findings included; a pale oedematous nasal mucosa, hypertrophy of inferior turbinates, watery / mucoid rhinorrhoea, transverse nasal crease, allegic shiners and/or conjuctival injection

Figure 2



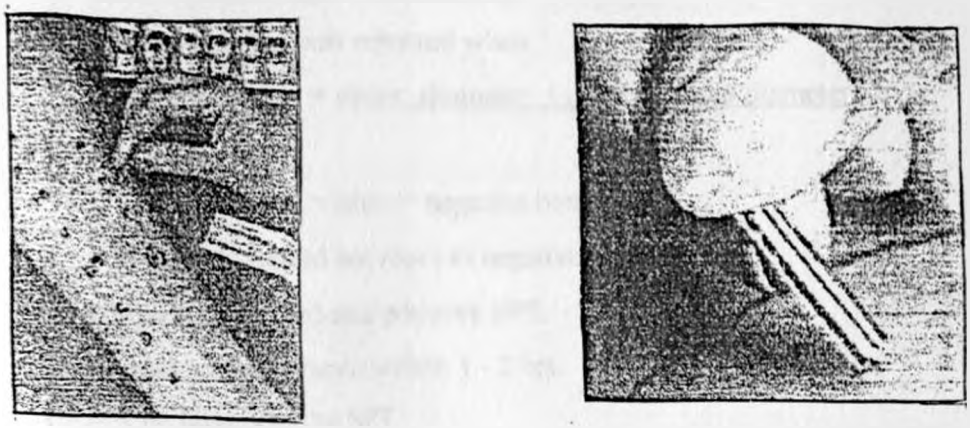
A total of 23 students were excluded from the study for the following reasons;

i. Eczema – 2 Students
ii. Severe Asthma on medication – 2 Students
iii. Failure to complete questionnaire, undergo a physical examination or SPT – 19 Students.

3.11.3 Test procedure (SPT)

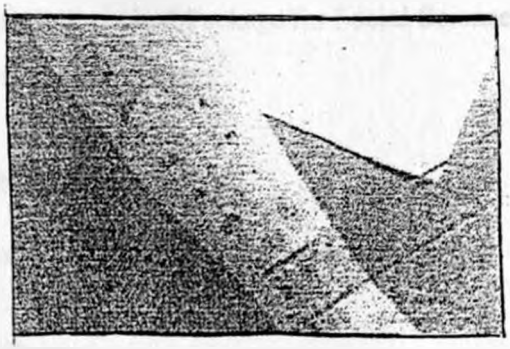
i. **Techniques of SPT**

- Volar aspect of forearm was cleaned with spirit swab and dots approximately 2 cm apart corresponding to number of allergens to be tested were marked off using a marking pen.
- A drop of allergen was put on dot and a prick to skin made using a sterile lancet/needles.A sterile needle per allergen was used.
- A positive (histamine 1mg/ml) and negative (saline) control was included in the testing.
- Excess allergen was removed by putting a tissue (blotting paper) on the drop.



Pricking skin

wheals



Measuring wheals

ii. **Interpretation of Results**

- Tests were read after 15-20minutes
- Positive reaction was reported when

  average diameter = $\frac{\text{major diameter + perpendicular diameter}}{2}$

  =3mm > negative control
- NB/patients should not react to negative control
- Atopy was defined as a positive SPT.

The wheal normally heals within 1 - 2 hrs.

Causes of false positive SPT

- Induction of bleeding
- Irritant reaction
- Tests too close together (<2cm) - non-specific enhancement through axon reflex from nearby strong reactions.

Causes of false negative SPT

- Insufficient skin penetration
- Allergen preparation of diminished potency
- Medication e.g. antihistamines
- Recent systemic anaphylaxis - SPT done more than a week
- Diseases diminishing skin response e.g. eczema

3.12 RECORD OF DATA

- Data is presented in pie charts, histograms
- Data was analyzed using Statistical Package for Social Sciences (SPSS)
- Assistance from a statistician was sort

3.13 QUALITY CONTROL –

Standardized commercial extracts from a single vendor was used.

3.14 STUDY DURATION:

The study was carried out from 6th to 18th February, a study duration of 2 weeks.

4.0 ETHICAL CONSIDERATIONS

- Clearance was given by KNH ethical committee and the Director of KMTC.
- Students safety was ensured by employing sterile procedures e.g. use of disposable sterile needles, use of antiseptic for cleaning skin test areas.
- Only students meeting inclusion criteria were tested (SPT)
- Confidentiality was protected.

5. STUDY LIMITATIONS

- Costs

One of the vendors could supply all the allergens that were required but the costs were prohibitive. The second vendor who was selected (Allergy Therapeutics, UK) does not supply allergen mixtures, so mould mix was substituted with individual fungal allergens like Penicillium Notatum and Aspergillus Niger and dog hair was used instead of Tropical Grass mix..

- Some students either refused completing the questionnaire or to undergo SPT.

- It was difficult to induct students in one department (Records) because the students were not available – they were either doing exams or had just completed and were scattered

- For safety reasons SPT could not be carried out in students with severe asthma on treatment or eczema, for which an ideal test would have been IgE counts.

6. RESULTS

6.1 PREVALENCE AND SEVERITY

6.1.1 PREVALENCE

(i) Prevalence of AR by Age

Table 3

Age	No. Screened	No. Positive	% Positive
18 – 22	226	29	12.8
23 – 27	111	15	13.5
28 – 32	34	6	17.6
33 – 37	27	2	7.4
38 – 42	23	3	13.0
43 – 47	2	0	0
TOTALS	423	55	13%

The prevalence rate of AR at KMTC is 13%. The majority (52.7%) were in the age group of 18 – 22 years who also formed a majority (53.4%) of the students inducted. Questionnaire based AR is 14.9% (63/423 x 100%)

Table 4

Study Group	Mean	STD Deviation	Median	Minimum	Maximum
Positive	24.24	5.167	22.00	19	40
Normal	24.31	5.725	22.00	18	46
Total	24.30	5.650	22.00	18	46

The mean age is 24.2, a median of 22 years and age range of 18 to 46 years.

Table 5
T- Test

	Study Group	N	Mean	STD Deviation	P
Age in Years	Positive	55	24.24	5.167	0.96
	Normal	368	24.31	5.725	

Despite the fact that majority of the students having AR were in the age group 18 to 22 years, the T-test showed no significance (P = 0.926) in the correlation of prevalence to age.

(ii) Prevalence of AR by Sex

Table 6

			Male	Female	Total
Study Group	Positive	Count	25	30	55
		%Within Study group	45.5%	54.5%	100%
	Normal	Count	177	191	368
		%Within study group	48.1%	51.9%	100%
Total		Count	202	221	423
		%Within study group	47.8%	52.2%	100%
Prevalence by sex	Positive	Count	25	30	55
		% within sex group	12.4%	13.6%	13%

Chi-Square Tests

Table 7

	Value	Df	Asymp Sig (2-Sided)	Exact Sig (2 Sided	Exact Sig (1-Sided)
Person Chi-Square	.134(b)	1	.714		
Continuity Correction (G)	.049	1	.825		
Likelihood Ratio	.134	1	.714		
Fisher's Exact Test				.773	.413
Linear-by-Linear	.134	1		.715	
Association					
No. of Valid Cases	423				

The prevalence of AR in females is 12.4% while in males is 13.6%. This shows a predominance of AR in males but from chi square tests ($x^2 = 0.134$; P = 0.77) this is not significant. Therefore, the prevalence of AR is not correlated to gender in the study group.

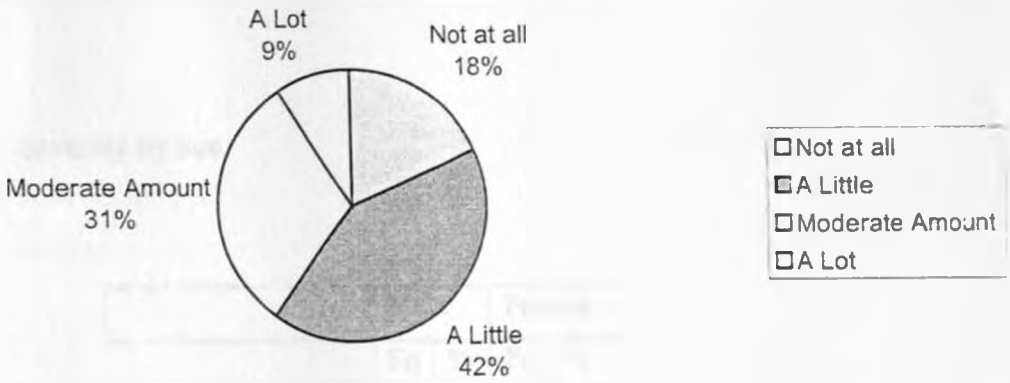
6.1.2 SEVERITY OF SYMPTOMS

(i) Table 8

Severity	Frequency	%
Not at all	10	18.2
A little	23	41.8
A moderate amount	17	30.9
A lot	5	9.1
Totals	55	100.0

Figure 3

PIE CHART REPRESENTING SEVERITY



Majority of students (41.8%) had the rhinitis symptoms interfering with their daily activities a little, while only in 9.1% was interference alot.

(ii) Severity by Age

Table 9

	Not at all		A little		Moderate		A lot	
Age in years	Fq	%	Fq	%	Fq	%	Fq	%
18-22	4	7.3	12	21.8	11	20	2	3.6
23-27	4	7.3	7	12.7	4	7.3	0	0
28 – 32	1	1.8	3	5.4	1	1.8	1	1.8
33-37	1	1.8	0	0	1	1.8	0	0
38-42	0	0	1	1.8	0	0	2	3.6

Severity of symptoms was more in the 18-22 years old age group, which was also the predominant age group in the study group. 21.8% and 20% of them had symptoms interfering with their daily activities a little and to a moderate amount respectively.

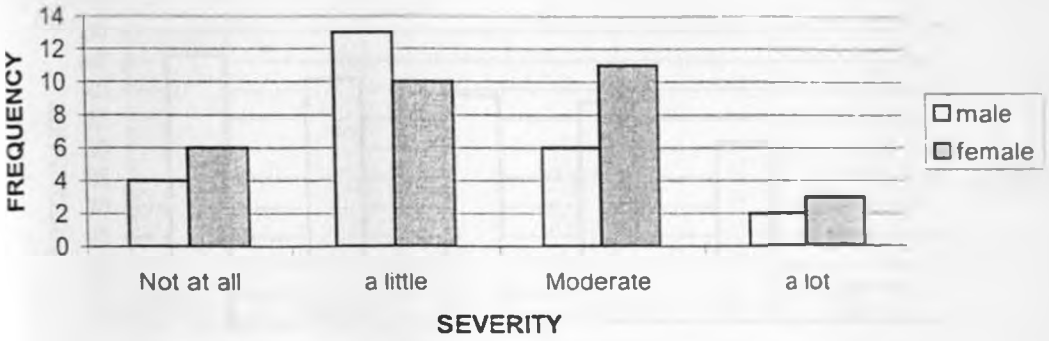
(iii) Severity by Sex

Table 10

	Male		Female	
	Fq	%	Fq	%
Not at-all	4	16	6	20
A little	13	52	10	33
A moderate amount	6	24	11	36.7
A lot	2	8	3	10

Figure 4

BAR GRAPH REPRESENTING FREQUENCY OF SEVERITY ACCORDING TO SEX



There were more females (20%) than males who had moderately severe symptoms. The males predominantly (52%)complained of symptoms interfering with the daily activities a little.

6.2 PRESENTATION AND PATTERN OF SYMPTOMATOLOGY

6.2.1 Symptoms and Signs

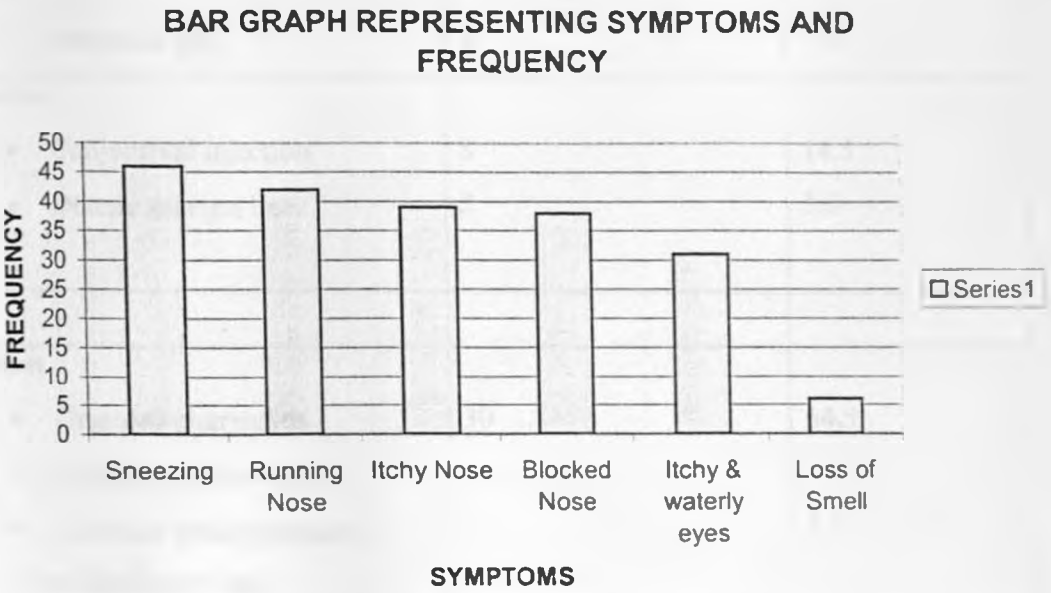
a. Symptoms

Table 11

Symptoms	Frequency	%
Sneezing (>3 spells) with no cold or flu	46	83.6
Runny nose with no cold or flu	42	76.4
Itchy nose with no cold or flu	39	70.9
Blocked nose with no cold or flu	38	69.1
Itchy watery eyes	31	56.4
Loss of sense of smell	6	10.9

The most common symptom was sneezing (83.6%), followed by runny nose (76.4%), itchy nose (70.9%) and blocked nose (69.1%). The least common was loss of sense of smell (10.9%).

Figure 5



b. Signs

Table 12

Physical Findings	Frequency	%
Allergic shiners	1	1.8
Transverse nasal crease	6	10.9
Nasal turbinates		
• HIT/boggy. pale	39	70.9
• Deep red	1	1,8
• HIT/Deep red	1	1.8
• Atrophic turbinates	1	1.8
Nasal mucous		
• Thin watery	33	60
• Thick purulent	8	14.5
Colour of mucusa		
• Pale	27	49.1
• Red	18	32.7
• Bluish	9	16.4

Sense of smell • Hyposmia	5	9.1
Ears • Retracted TM	4	7.3
Ocular • Conjuctival injection • Dennie morgan lines	8 2	14.5 3.6
Throat • Granular pharyngitis • Tonsillar hypertrophy • Granular pharyngitis and Tonsillar hypertrophy	30 1 3	54.5 1.8 5.5

No abnormalities were detected in the lungs, neck or skin.

The most common physical findings was a pale hypertrophied inferior turbinates (70.9%). This was followed by thin watery nasal mucous (60%), granular pharyngitis (54.5%) and pale nasal mucosa (49.1%). The least common were allergic shiners, deep red inferior turbinates, atrophic turbinates and tonsillar hypertrophy each with 1.8%.

6.2.2 Pattern of Symptomatology

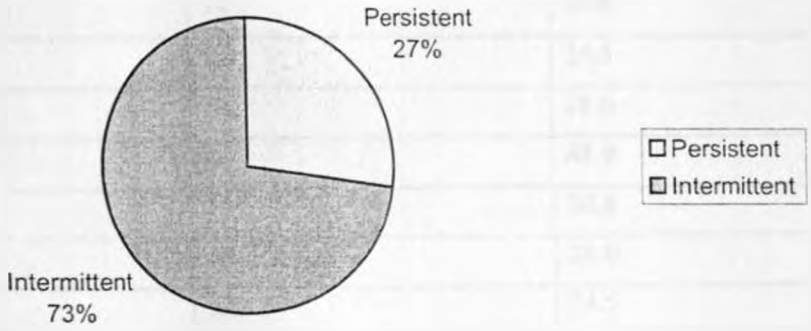
a. Duration of symptoms

Table 13

Duration of symptoms	Frequency	%
Problems at any one time last > 1 month	15	27.3
Problems at any one time last < 1 month	40	72.7

Figure 6

PIE CHART REPRESENTING DURATION OF SYMPTOMATOLOGY



Most of the students had intermittent disease (72.7%) while the rest had persistent disease (27.3%) according to ARIA classification of AR.

b. Age in Years of onset

Table 14

Age in years	Frequency	%	Age in years	Frequency	%
5	2	3.6	17	5	9.1
7	1	1.8	18	3	5.5
8	1	1.8	19	1	1.8
9	2	3.6	20	3	5.5
10	1	1.8	21	1	1.8
12	3	5.5	22	1	1.8
13	1	1.8	23	1	1.8
14	4	7.5	26	1	1.8
15	10	18.2	Don't know	8	14.5
16	6	10.9			

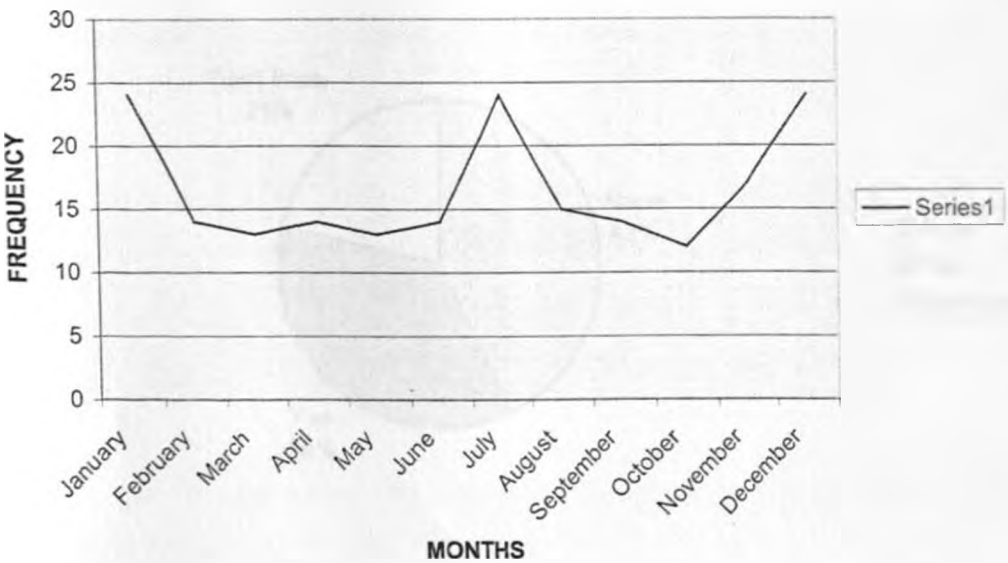
The average age of onset was 15.2 years. Those who had onset from childhood (≤ 12 years) were 18.2%.

c. Months (Seasonal variations)

Table 15

Month	Frequency	%
January	24	48.9
February	14	28.6
March	13	26.5
April	14	28.6
May	13	26.5
June	14	28.6
July	24	48.9
August	15	30.6
September	14	28.6
October	12	24.5
November	17	34.7
December	24	48.9
Don't know	6	10.9
Throughout the year	9	18.4

GRAPH REPRESENTING FREQUENCY BY MONTHS



The symptoms of rhinitis were most common in the months of January, July and December all with 48.9%.

The least common month was October with 24.5%.

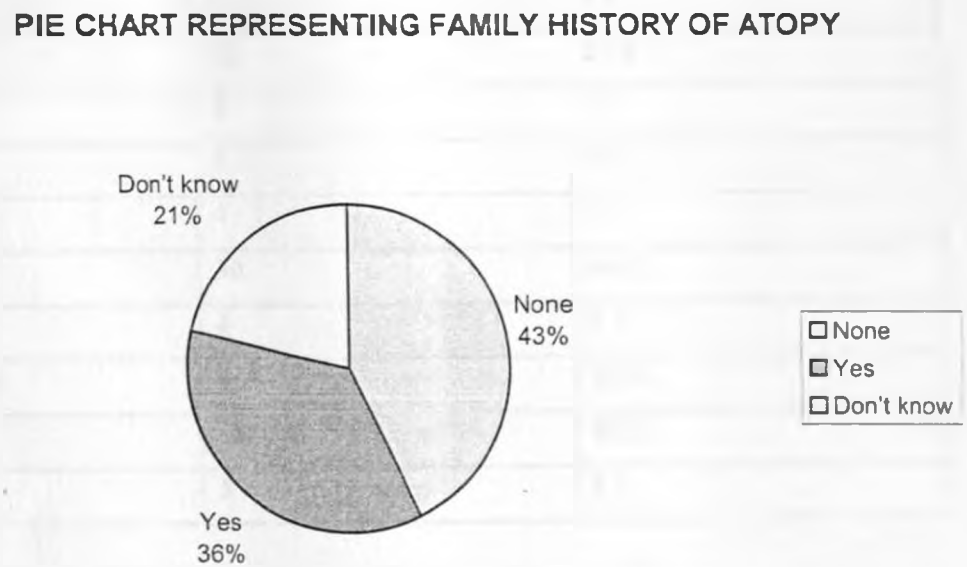
10.9% did not know the months their symptoms occurred in the last 12 months.

d. Family history of atopy

Table 16

Family history of atopy	Frequency	%
• None	24	43.6
• Yes	20	36.4
e.g.		
- Asthma	10	81.1
- allergic rhinitis	5	9.1
- food allergies	3	5.4
- Eczema	1	1.8
- Drug allergies	1	1.8
Don't know	11	20

Figure 8



36.4% of the students with allergic rhinitis had a family history of atopy. Asthma (81.1%) was the commonest allergic condition noted in the families.

6.3 ALLERGENS

6.3.1 Types of Allergens and other environmental factors that trigger rhinitis symptoms

a. Questionnaire Based

Table 17

Allergens	Frequency	%
House dust	44	80
Pets		
• Cats	2	3.6
• Dogs	2	3.6
• Rabbits	2	3.6
Grass	6	10.9
Trees	4	7.3
Flowers	17	30.9
Moulds	5	9.1
Weeds	12	21.8
Foods	3	5.5
Drinks	4	7.3
Medication	4	7.3
Smoke	30	54.5
Stress	4	7.3
Cold weather	46	83.6
Perfumes	10	18.2
Others	5	9.1

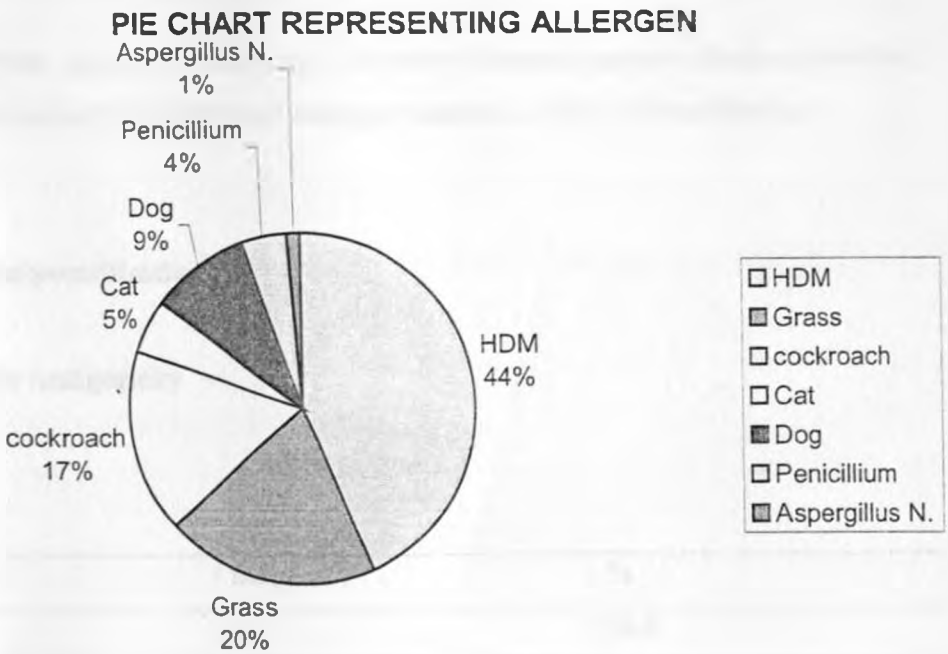
The most common allergens and environmental factors reported to worsen rhinitis symptoms were; cold weather 83.6%, house dust 80%, smoke 54.4%, flowers 30.9% and weeds 21.8%.

b. SPT based allergen

Table 18

Allergen	Frequency	%
House dust mite (HDM)	42	76.4
Grass	19	34.5
Cockroach	16	29.1
Cat	5	9.1
Dog	9	16.4
Penicillin Notatum	4	7.3
Aspergillus Niger	1	1.8

Figure 9



The most common allergen was house dust mite (76.4%), followed by grass (34.5%) and cockroach (29.1%). The least common was Aspergillus Niger (1.8%).

6.3.2 Reactivity of Allergens

Table 19

Allergen	Range (cm)	Average (cm)
House dust mite	0.3 – 1	0.6
Grass	0.3 – 0.8	0.49
Cockroach	0.3 – 1	0.46
Cat	0.3 – 0.5	0.4
Dog	0.3 – 0.5	0.37
Penicillium Notatum	0.3 – 0.4	0.35
Aspergillus Niger	0.4	0.4
Histamine (positive control)	0.3 – 0.7	0.49

House dust mite, grass and cockroach which were the most common allergens identified, also showed a higher reactivity with average diameters of 0.6, 0.49 and 0.46 cm respectively.

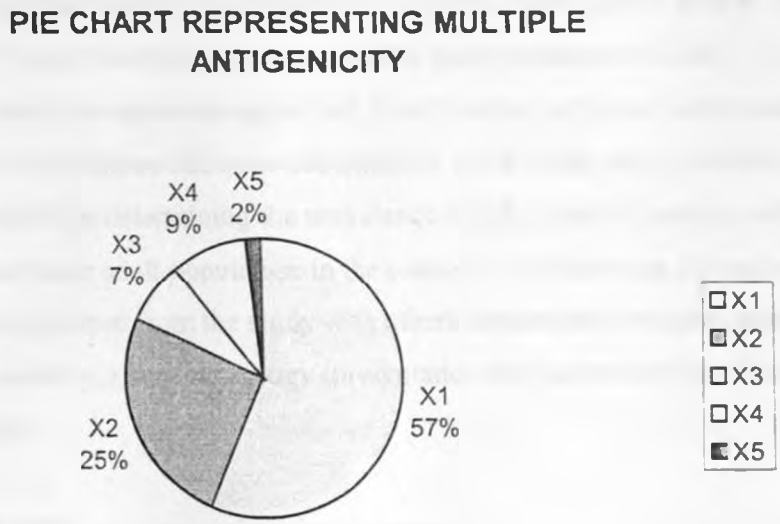
6.3.3 Polysensitisation

a. Multiple Antigenicity

Table 20

Multiple	frequency	%
X1	31	56.4
X2	14	22.5
X3	4	7.3
X4	5	9.1
X5	1	1.8

Figure 10



56.4% of the students reacted to a single antigen while 43.6% reacted to multiple antigens.

b. Combination of Multiple Antigenicity

Table 21

Types	Frequency	%
HDM + Cockroach	6	10.9
HDM + Grass + Cockroach	3	5.5
HDM + Cat	2	3.6
HDM + Dog	2	3.6
HDM + Grass	2	3.6
HDM + Grass + Cockroach + Penicillium	2	3.6

The most common multiple antigen combination was HDM and cockroach (10.9%).

7. DISCUSSION

The present study was designed to determine the prevalence of allergic rhinitis among 18-50 years old students at KMTC Nairobi. It correlated prevalence of AR to age and sex, severity of AR, determine the presentation and pattern of symptomatology and common aeroallergens involved in the group thus studied. It was carried out over a 2 week period from 6th to 18th February at KMTC Nairobi and the results represent a point prevalence of AR.

Currently in our country the epidemiology of AR is very sparse, only one questionnaire based study (ISAAC) in children has been documented. The present study included an objective measure (SPT) in determining the prevalence of AR in adults (college students) which represents the wider adult population in the country. The following discussion compares the results obtained from the study with others documented focusing mainly on the prevalence of AR, severity, symptomatology (presentation and pattern) and the common aeroallergens involve.

7.1 PREVALENCE

7.1.1 PREVALENCE OF AR

The present study got a prevalence rate of 14.9% (15%) by questionnaire, the definition of rhinitis was based on ICR[20]. Olivier et al[48] using a screening questionnaire got an overall prevalence of self reported AR to be 15.9%. D.Y Wang[20] using ICR definition of AR got a prevalence of 13.1% based on self reported questionnaire. Shahar et al[43] reported the prevalence of self reported AR in Israel to be 14%.

The prevalence of SPT positive AR in this study was 13%. Ciprandi et al [45] using SPT found a prevalence of 2.2% and a follow up study 7 years later reported a prevalence of 10.15% [50] Linnerberg et al [46] reported a SPT prevalence in Dannish adults of 22.5%.

The reported data on prevalence is varied from as low as 2.2% to as high as 41.5%.

The difference in prevalence as discussed earlier could be due to the following;

1. **Methodology**
    a. There is no standard definition of rhinitis in epidemiological studies.
    b. Questionnaire or self reported studies generally give a higher prevalence rate.

c. Studies which included IgE counts in confirmation of atopy eg Bauchan et al [18] had the advantage of capturing some AR patients who naturally could have been excluded from SPT e.g eczema patient, history of severe anaphylaxis etc.

Generally tests for confirmation of atopy did not seem to play a significant role in explanation of the difference of prevalence rate of the present study and other objectively done studies.

2. Country/Geographical Factors

In our society (developing countries) the level of awareness of AR is limited by the reduced/limited access to health information, and also environmental pollution is relatively lower in these countries which are currently stepping up industrialization.

Most of the studies done in the western countries e.g Huure et al [42] in Finland (26%) etc have shown a relatively higher prevalence compared to the present study.

According to the "Hygiene hypothesis", the cleaner the environment the higher the prevalence of AR, thus it is expected the more urbanized (developed) an area is the higher the prevalence.

3. Duration of Study

The present study reports a point prevalence of 13%. Ogino et al [49] studied nasal allergy in medical students in Japan from 1983 to 1987 and reported a prevalence of 30%. The longer the study in terms of duration the higher the chances of influence by factors like season aeroallergen load.

4. Age Group

The age bracket included in the adult prevalence studies show marked variability and hence could have affected the prevalence rates obtained.

The present study had a prevalence of 13% with an age range of 18-46 years.

Fedoseev et al [47] inducted respondents aged 16 to 98 years and reported a prevalence of 9.9%. Ogino et al [49] got a prevalence of 30% in medical students. The studies that restricted themselves to the peak age group (20 – 40) years tend to give a higher prevalence rate, while those inducting the elderly show a lower prevalence.

5. Sample Size

The size of the sample did not seem to affect so much the differences or variation in prevalence. The study screened 423 students and got a prevalence of 13%, while D.Y Wang[20] inducted 4602 subjects,got a prevalence of 13.1% and Ciprandi et al [45] inducted 2876 conscripts and got a prevalence of 2.2%.

7.1.2 PREVALENCE IN RELATION TO SEX

The present study reports a prevalence of 12.4% in females and 13.6% in males. The slight predominance of prevalence in males however was not found to be statistical significant (chi square – 0.134; p=0.77). The sex predilection of AR still remains a controversial issue with most literature not agreeing on sex predominance.[3,4,22,23]

Min Y.G et al [25] got an overall prevalence of perennial AR to be 1.14%, with a prevalence of 1.18% in females and 1.08% in males. Huure et al [42] in a follow up survey of Finnish urban age cohort found the prevalence of AR to have risen in males form 18.7% to 27.8% while for females from 16.2% to 24.2%.

7.1.3 PREVALENCE IN RELATION TO AGE

Despite the fact that the majority of the students with AR were in the age group 18-22 years (53.4%), which was also the main group in the study sample population, there was no statistical significance in prevalence of AR correlated to age (T test, significance = 0.926). This could have been as a result of a small age range inducted in the study (18-47yrs), if the age group included also children and the elderly probably a peak age would have been identified..

7.2 SEVERITY

This study reports severity not affecting daily activity as18.2%, daily activity affected a little 41.8%, a moderate amount 30.9% and a lot 9.1%.

Monte fort et al [52] reported severity in AR as follows; not affecting daily activities 31.8%, daily activity affected a little 43.2%, moderately 13.7% and affected a lot 2.9% .

These two studies concur that AR does affect daily activities a little, predominantly, at 41.8% and 43.2% respectively and a small percentage are affected a lot, 9.1% and 2.9% respectively. The present study also showed that females complained of more severity (moderately 36.7%) than males (a little 52%).

7.3 PRESENTATION AND PATTERN OF SYMPTOMATOLOGY

7.3.1 SYMPTOMATOLOGY

1. Symptoms

The present study presents sneezing as the most common symptom 83.6%, followed by runny nose 76.4%, itchy nose 70.9% and blocked nose 69.1%. It concurs with studies by D.Y Wang [20] and Cirillo et al [50] that reported sneezing the most common nasal symptom.

2. Signs

The most common finding in the present study was inferior turbinate hypertrophy (70.9%) followed by thin watery nasal mucous (60%) and granular pharyngitis (54.5%)

Gabriel Mhidze [51] in Tanzania found turbinate hypertrophy the commonest presentation (20.8%).

7.3.2 PATTERN OF SYMPTOMATOLOGY

(i) Duration of symptoms

In the present study, students who had persistent symptoms were 27% while those with intermittent symptoms were 73%. Greisner et al [35] found a cumulative prevalence of self reported seasonal AR as 41.5% and non seasonal AR as 14%.

The two studies concur that intermittent disease is more prevalent than persistent disease.

(ii) Age of Onset

The onset of AR is common in childhood, adolescence and early adulthood with a mean age of onset of around 8-11 years [17].

The current study gives a mean age of onset as 15.2 years. Those who had onset from childhood (≤12 yrs) were 18.2%. This figure could have been influenced by partially, lack of knowledge of actual time of onset, indeed 14.5% of the students did not know the time of onset of symptoms.

(iii) Months

Three peaks of symptomatology frequency was noted in the months of January, July and December in the present study. January and December are relatively dry months in our weather calendar and this could be associated with an increase in amount of outdoor allergens e.g. dust, grass pollen, fungi spores etc July is a cold season and most people spend time indoors, therefore, probably increase their exposure to indoor allergens especially house dust mite. It is also possible that those students with AR have a co-existing vasomotor rhinitis that worsens their symptoms in the cold season.

Esamai's [55] data in prevalence of AR in primary school children in Uasin Gishu did not support seasonal variations. Montefort et al [52] noted seasonal symptoms peaked in February, March, and April in Maltese children, lowest in summer and started rising in September.

(iv) Atopy

Genetic predisposition is a known associated aetiological factor in AR as discussed earlier. 36% of the students gave positive history of allergy in their family. Asthma was noted to be the commonest allergic condition.

7.4 ALLERGENS

i) Types

This study reported a high sensitization rate to house dust mite (76.4%) followed by Bermuda Grass Pollen (34.5%) and cockroach (29.1%). The least common was Aspergillus Niger (1.8%). Raukas et al [54] and Ogino et al [49] found the commonest allergen to be German cockroach (15.5%) and house dust mite (66.4%) respectively.

The findings of the study tally well with other tropical areas where studies done e.g. Baratawidjaja et al [53] and Monte Alegra et al [24], have confirmed animal allergen to be more prevalent, followed by plant then fungal allergen.

Generally the dorminant seasonal allergens varies world wide because of differences in geographical and climatic conditions.

(ii) Polysensitisation

Polysensitisation was seen in 43.6% of the students with AR. The commonest combination of allergens was HDM and cockroach. At least 18.2% of the students had polysensitisation to 3 or more allergens and thus would not be ideal candidates for immunotherapy.

8. CONCLUSION

The prevalence of AR in students at KMTC is 13%, there was no sex or age predilection in the group thus studied. 81.8% of the students with AR had their daily activity affected to a certain degree due to the symptoms of AR, of these 9.1% had their daily activity affected a lot.

From the above it does appear that AR affects a significant proportion of the population and does have some impact on the lifestyles of these patients. Thus the results of the study should act as a stimulus for better management of this condition and employing measures that can control the aetiological factors.

The age of onset of AR was 15 years, commonest symptoms sneezing and runny nose, symptoms peaked in January, July and December and 36% of those with AR showed family history of atopy. The commonest allergen was HDM and Bermuda Grass pollen, with 43% of those with AR showing polysensitisation.

In summary, AR affects a significant proportion of the adult population with some impact on their lifestyle and hence the economy at large. The research findings should be of help especially to public health officers and clinicians in creating awareness on allergic rhinitis and how to implement proper, cheap and effective environmental control measures and allergen avoidance.

9. RECOMMENDATIONS

There requires a consensus on the epidemiological definition of rhinitis so that prevalence gotten from the studies can be comparable.

A standardized adult questionnaire and methodology in prevalence surveys needs to be designed eg an internationally accepted adult questionnaire (equivalent to ISAAC), inclusion of confirmation of atopy etc.

Identification of allergen sensitization should be included as part of management of AR patients so as to be able to identify those that can benefit from immunotherapy and also in allergen avoidance. This information will also be of great importance to public health officers in implementing proper and cheap environmental control measures and patient education in general.

We need to identify fully and document the common aeroallergens in our environment and if possible manufacture them locally to ease patient's identification of allergens and facilitate further research in AR.

Further studies need be done in the following areas:

Socio-demographic and risk factors of AR e.g. geographical areas, occupation, etc.

Impact of AR on economy and lifestyle

Association with comorbid factors e.g. asthma.

Large series study e.g. Nation wide survey on AR.

10 APPENDICES

10.1 CONSENT EXPLANATION/GENERAL PATIENT INFORMATION

General Patient Information

We would like to seek your consent to participate in a study aimed at knowing the prevalence and understanding the nature of allergic rhinitis (AR). We would like to know more about this disease in our set up so that we can be able to manage it better.

How do you participate?

1. We will ask some questions to find out if you have rhinitis, understand how the disease developed and what factors might have played part in the development.
2. Participants with symptoms of rhinitis shall undergo a relevant clinical examination and skin prick test (SPT) to confirm AR.
3. We will compare the results of your test with other participants.

How does your participation affect you?

1. If you are not symptomatic for rhinitis then no further investigation or examination will be carried out.
2. Participants who are symptomatic for rhinitis shall undergo a SPT which is quite a safe procedure and is done to confirm and determine the allergens involved in causation of AR.
3. Treatment will be advised on participants confirmed to have AR eg: -allergen avoidance measures shall be advised to the identified allergens.
4. All information given will be confidential.
5. The study does not reveal individual identity.

Are there any hidden dangers in participation or non-participations?

1. Participants with history of severe anaphylaxis, pregnancy, eczema, or on immuno-suppressive drugs shall be excluded from the study since they will not undergo SPT for safety reasons.
2. If you object to any part or the whole of this study you are free to refuse to participate.

What do we do with the information we get?

1. The information we get will not only be of immediate benefit to the participants with allergic rhinitis on how to manage their allergic condition but will also help us in the long run in fighting the disease.
2. Like all scientific information, we will seek to share our findings with other people undertaking similar studies. Therefore, we may publish our findings in scientific journals or present them at meetings.
3. If you require to discuss this matter with family, friends or associates you are free to do so and we will be ready to answer any questions. If you are satisfied with our explanation and are willing to participate, please sign the consent form below.

10.2 CONSENT FORM

I ..... ID No..... study No. ..... of ..... do, hereby, consent to participate in the study aimed at determining the prevalence of allergic rhinitis in college students at Kenya Medical Training College, Nairobi. The nature of the study has been explained to me by Dr..... and no material gain has been suggested in order for me to be included in this study.

Signed ....
(Self/Guardian) Date

Investigator Dr. .....

....
(Signature) Date

10.3 STUDY PROFORMA 1

QUESTIONNAIRE

Personal information

Date:

Name :

Age: Sex:

District :

Length of time lived in Districts: Home Address:

QUESTIONS

The following questions are about your health

Answer questions with a tick unless instructed otherwise

e.g. Yes [✔]

No [ ]

All questions are about problems, which occur when you don't have a cold or flu.

SECTION A

1. Have you ever had the following nose problems when you don't have a cold or flu in the past 12 months?

	Yes	No
Itchy nose	[ ]	[ ]
Runny nose	[ ]	[ ]
Sneezing spells (>3 sneezes in a row)	[ ]	[ ]
Blocked nose	[ ]	[ ]

2. Has this nose problem been accompanied by itchy watery eyes?

Yes	No
[ ]	[ ]

3. Do the above nose (± eye) problems last more than 1 hour for most days

Yes	No
[ ]	[ ]

SECTION B

4. Do the above nose (± eye) problems at any one time last for

	Yes	No
More than 1 month	[ ]	[ ]
Less than 1 month	[ ]	[ ]

5. In which month/months of the past 12 months did the nose problem occur?

January [ ] March [ ] May [ ] July [ ] Sept. [ ]
February [ ] April [ ] June [ ] Aug. [ ] Oct. [ ]
November [ ] December [ ]

6. How old were you when your eye or nose trouble started? (age in years)

.....

7. Have you lost your sense of smell? Yes [ ] No [ ]

8. Do you known of anything that you think caused or worsened your eye or nose problem? Tick as appropriate

House dust	[ ]	Weeds	[ ]
Pet - Cats	[ ]	Foods	[ ]
- Dogs	[ ]	Drinks	[ ]
- Rabbits	[ ]	Medication	[ ]
- Others (specify).....			
Grass	[ ]	Smoke	[ ]
Trees	[ ]	Stress	[ ]
Flowers	[ ]	Cold weather	[ ]
Molds	[ ]		

9. In the past 12 months how much did the nose problem interfere with your daily activities?

Not at all [ ]
A little [ ]

A moderate amount [ ]

A lot [ ]

10 Are you taking any medication for your eye or nose problem? Yes No

[ ] [ ]

If yes, which type?

Antihistamines[ ] Injections (immunotherapy) [ ]

Drops/sprays Other medicines [ ]

Steroids [ ] (Specify).....

Decongestants [ ]

Anticholinergics [ ]

Sodium cromoglycate [ ]

11. Is there a family history of allergy e.g. Asthma, eczema, food or drug allergies etc

Yes No I don't know

[ ] [ ] [ ]

If yes, specify .....

12. What other medications, other than the above have you been taking over the last 3 months?.....

13. Are you suffering or being treated for any other illness?

	Yes	No
Hypothyroidism	[ ]	[ ]
Connective tissue disorder	[ ]	[ ]
Immunosuppression	[ ]	[ ]
Ciliary Dyskinesia	[ ]	[ ]

Others (Specify) .....

14. Do you have any other significant medical history e.g. history of anaphylaxis etc?

Yes No

[ ] [ ]

If yes, describe.....

10.4 STUDY PROFORMA 2
PHYSICAL EXAMINATION FINDINGS AND SPT RESULTS

1. General facial features

	Yes	No
- Allergic shiners	[ ]	[ ]
- Transverse nasal crease	[ ]	[ ]

2. Nose

	Yes	No
i. Nasal turbinates		
- HIT/Boggy, pale, bluish gray in colour	[ ]	[ ]
- Deep red	[ ]	[ ]
- Normal	[ ]	[ ]
ii. Nasal Mucous		
- Thin watery secretions	[ ]	[ ]
- Thick + purulent	[ ]	[ ]
iii. Colour of nasal mucosa		
- Pale	[ ]	[ ]
- Bluish	[ ]	[ ]
- Red	[ ]	[ ]
iv. Nasal septum		
- Deviated	[ ]	[ ]
- Septal perforation	[ ]	[ ]
- Normal	[ ]	[ ]
v. Other masses		
- Polyps	[ ]	[ ]
- Tumours	[ ]	[ ]
vi. Sense of smell		
- Normal	[ ]	[ ]
- Anosmia	[ ]	[ ]
- Hyposmia	[ ]	[ ]

3. Ears

TM: - Retracted	[ ]	[ ]
- Air-fluid level / bubbles	[ ]	[ ]
- Normal	[ ]	[ ]

4. Ocular

- Conjunctival injection/excess tears	[ ]	[ ]
- Dennie – Morgan lines	[ ]	[ ]
- Normal	[ ]	[ ]

5. Throat

- Granular pharyngitis	[ ]	[ ]
- Tonsillar hypertrophy	[ ]	[ ]
- Malocclusion of teeth	[ ]	[ ]
- High arched palate	[ ]	[ ]
- Normal	[ ]	[ ]
	Yes	No

6. Neck

- Lymphadenopathy	[ ]	[ ]
- Thyroid disease (Goitre)	[ ]	[ ]

7. Lungs

- Rhonci	[ ]	[ ]
- Normal	[ ]	[ ]

8. Skin

- Atopic dermatitis	[ ]	[ ]
- Normal	[ ]	[ ]

SKIN PRICK TEST RESULTS

	Test	Major θ	Minor θ	AVG θ	Remarks
1.	Control				
	- Positive				+ve
	- Negative				-ve
2.	Allergen tested				
	i)				
	ii)				
	iii)				
	iv)				
	v)				
	vi)				

11.0 BUDGET

Item Description	Estimated Amount
Stationery/Printing Expenses	10,000.00
Materials (Gloves, spirit, cotton wool, tissue paper, adrenaline, branular, syringes, prick needles)	12,300.00
SPT kit	105,000.00
Secretarial Services	5,000.00
Data Analysis	5,000.00
Ethical committee	500.00
Contingency (10%)	14,000.00
TOTAL BUDGET	**152,800.00**

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