A STUDY OF "CORRELATIONBETWEEN ABSOLUTE EOSINOPHIL COUNT AND ASTHMA CONTROL IN ADULT PATIENTS IN GOVT ROYAPETTAH HOSPITAL, CHENNAI - 14"

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for the Award of the Degree of

M.D. (GENERAL MEDICINE)
BRANCH – I

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April – 2015

BONAFIDE CERTIFICATE

This is to certify that **"A STUDY OF CORRELATION BETWEEN ABSOLUTE EOSINOPHIL COUNT AND ASTHMA CONTROL IN ADULT PATIENTS IN GOVT ROYAPETTAH HOSPITAL, CHENNAI - 14"** is a bonafide work performed by **Dr.G.NARAYANAN**, post graduate student, Department of Internal Medicine, Kilpauk Medical College, Chennai-10, under my guidance and supervision in fulfilment of regulations of the Tamil Nadu Dr. M.G.R Medical university for the award of M.D. Degree Branch I (General Medicine) during the academic period from May 2012 to April 2015.

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DECLARATION

I solemnly declare that this dissertation **"A STUDY OF CORRELATION BETWEEN ABSOLUTE EOSINOPHIL COUNT AND ASTHMA CONTROL IN ADULT PATIENTS IN GOVT ROYAPETTAH HOSPITAL, CHENNAI - 14"** was prepared by me at Government Kilpauk Medical College and Hospital, Chennai-10, under the guidance and supervision of **Dr.R.SABARATNAVEL.MD.,** Professor and Head of the Department of Internal Medicine, Government Kilpauk Medical College and Hospital, Chennai.

This dissertation is submitted to **The Tamil Nadu Dr. M.G.R. Medical University, Chennai** in partial fulfilment of the University regulations for the award of the degree of **M.D. Branch I (General Medicine)**.

Place: Chennai

Date: **(Dr. G.NARAYANAN)**

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ABSTRACT

A Study of "Correlation Between Absolute Eosinophil Count And Asthma Control In Adult Patients In Govt Royapettah Hospital, Chennai-14"

Introduction :

Bronchial Asthma is one of the most common disorders of inflammatory reactions of the respiratory tract, characterized by breathlessness, wheezing, minimal cough and expectoration.

Aim:

To study the correlation of absolute eosinophil count with asthma control in adult

Methods and methodology:

This study was conducted on the patient who attended the Asthma op in Govt Royapettah Hospital, Kilpauk Medical College, Chennai. The patients of 18-75 years were selected for the study. Both the sexes were included and who belong to different socio-economic groups was taken as per inclusion criteria. A total of 40 cases were selected minimum of study was 6months. The statistical analysis made here is based on the data obtained from 40 cases.

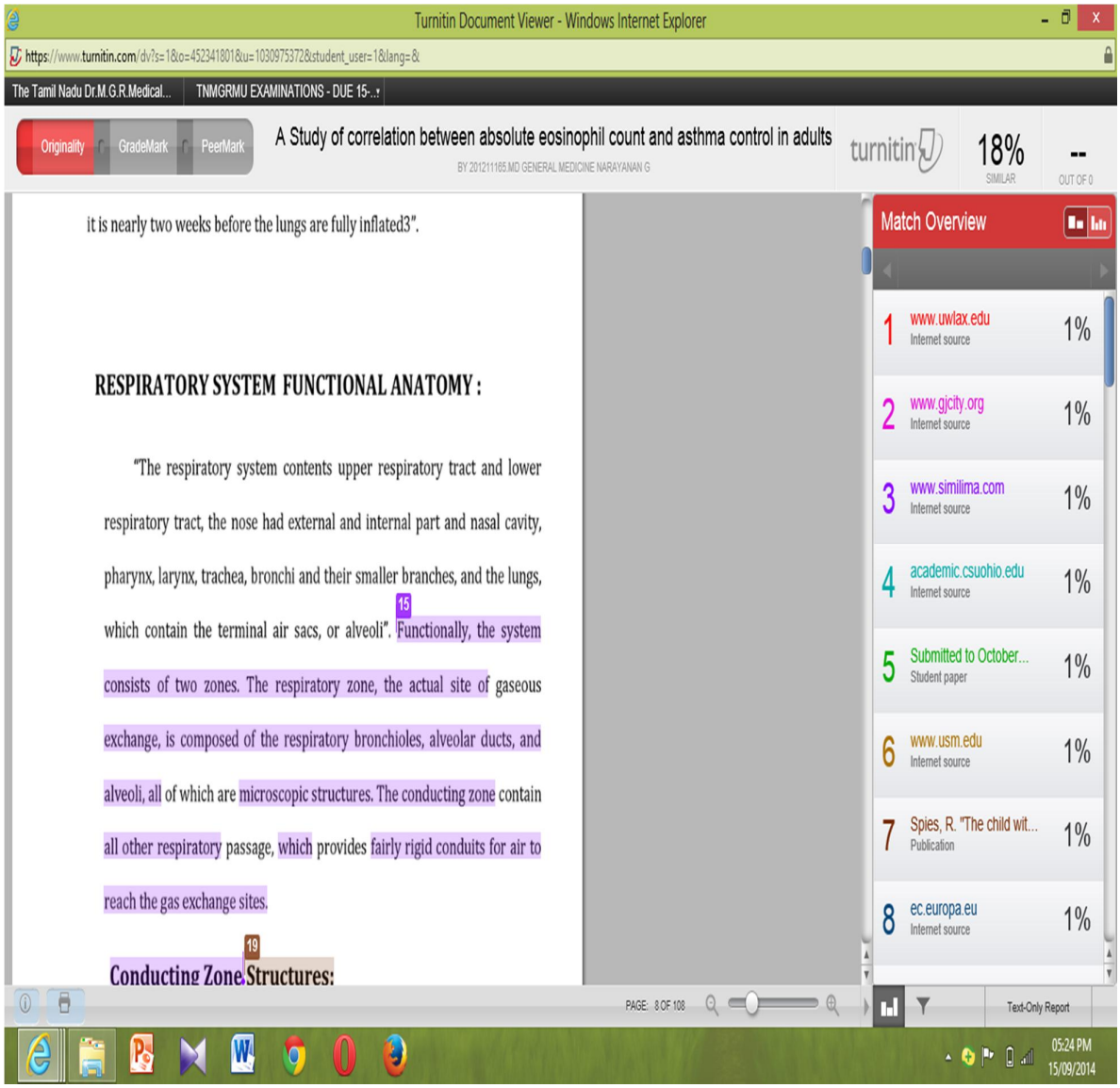
Results :

Fifty seven and half percent (57.5%%) of the asthmatic had increased eosinophil count(more than 450 cell per cumm) and forty two and half percent (42.5%) had normal absolute eosinophil count and p value 0.000.

Conclusion :

Our study shows that serum absolute eosinophil count have a significant impact(p value 0.000) on asthma control, higher levels correlating with poor control ,so increased dose or frequent steroid inhalation and dose adjustment, lower absolute eosinophil count well control of asthma.

Measurement of absolute eosinophil count can be useful in assessing the severity of such inflammation. So the evaluation of absolute eosinophil count is a useful baseline investigation and its serial measurements can be taken as a marker for adjusting the dose of inhaled steroids.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it is nearly two weeks before the lungs are fully inflated3".
RESPIRATORY SYSTEM FUNCTIONAL ANATOMY :
"The respiratory system contents upper respiratory tract and lower respiratory tract, the nose had external and internal part and nasal cavity, pharynx, larynx, trachea, bronchi and their smaller branches, and the lungs, which contain the terminal air sacs, or alveoli". Functionally, the system consists of two zones. The respiratory zone, the actual site of gaseous exchange, is composed of the respiratory bronchioles, alveolar ducts, and alveoli, all of which are microscopic structures. The conducting zone contain all other respiratory passage, which provides fairly rigid conduits for air to reach the gas exchange sites.
Conducting Zone Structures:
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CONTENTS

INTRODUCTION

Asthma has an alarmingly increasing incidence in the past few decades[1]and is one of the leading causes of morbidity in adults. Bronchial Asthma is a very common chronic disorder of the airways which affects individuals at almost any age and is an important cause of respiratory morbidity and mortality. The World health organization estimate is that there are 15 to 20 million people with asthma in India and affects 7% of the population and 300 million people worldwide[2]

Bronchial asthma is a predisposition to chronic inflammation of the lungs in which the bronchi are reversibly narrowed[3]It creates a substantial burden on individuals and families as it is more often under diagnosed & undertreated. Though effective screening, evaluation and management strategies are well established in developed countries, these are not fully implemented in India. Thus asthma appears to be increasing in prevalence despite considerable improvements in management and numerous drugs for treatment of asthma. So, it is essential to study the role of other extraneous factors influencing the control of asthma. There is various Allergens concern everyone, since it has become the part and parcel of life due to various factors involved. To an extent, no one is spared by allergens in atmosphere in which we live. All this allergens will precipitate the condition, leading to exacerbations of asthma where the

smooth muscle cells in the bronchi constrict the airways and become inflamed and swollen thereby breathing become difficult.

In modern system of medicine, drugs are used for long term prevention, starting with inhaler, corticosteroids and then long acting β-2 agonists if necessary.

Absolute eosinophil count is shown to be a risk factor for allergic asthma and can be used as a marker of disease severity in adult with asthma[4].So, in the present study our aim was to study of correlation between absolute eosinophil count and asthma control in adult patients[5].

These have prompted me to known a clinical study of Absolute Eosinophil count in bronchial asthma and to evaluate the effectiveness of medicine.

REVIEW OF LITERATURE

Framework:

1. Historical aspects of bronchial asthma

2. Magnitude of asthma in adults

3. Definition of asthma

4. Classificationof asthma

5. Pathogenesis

6. Absolute eosinophil count and its role in asthma

7. Diagnosis of asthma

8. Management of asthma

1. Historical aspects:

Asthma, though known for centuries is still an intriguing disease. The term asthma was derived from Greek word “casein” meaning sharp breath or to exhale with open mouth. This term was first used in Homer’s epic poem ‘Iliad’ in 800 B.C. It was called ‘sacred disease’ that was earned from a visit of gods. The word first appears in Homer’s “Iliad”. Hippocrates was the first to use it as a medical term in ‘corpus

hippocraticum'[6]in 450BC.In the 17th century, 'Bernardino Ramazzini[7]', identified and noted a connection between asthma and organic dust. In 19th century, initially it was considered as psychosomatic illness and was treated based on psychoanalysis. Later Henry Hyde Salter in his treatise "on asthma & its treatment" has refined the term asthma as "Paroxysmal dyspnoea of a peculiar character with intervals of healthy respiration between attacks." He found that aduquate amount of theobromine content in black coffee gives relief in asthmatic spasms.[8]At the end of 19th century physicians concluded that asthma was a distinct disease with specific manifestations.

DEVELOPMENT OF THE RESPIRATORY SYSTEM:

At about four weeks of development, the respiratory tracts begins as an elongated outgrowth of the foregut. "This out growth is called as the respiratory diverticulum[9]".The respiratory diverticulum have endodermal lining and its gives rise to the epithelium and glands of the trachea, bronchi and alveoli ducts and sac . The Musles, connective tissue and cartilage, in "relation to the organs of respiration are derived from splanchnopleuric mesoderm[10]".

As the diverticulum elongates, its distal end form the tracheal bud. It divides into bronchial buds.

By 24weeks, respiratory bronchioles will develop[11]. During 6to16weeks, lungs will be formed, except those of gaseous exchange.

During 16 to 26 weeks, lung tissue becomes highly vascular and respiratory bronchioles, alveolar ducts and alveoli develop[12].

At 30th week, mature alveoli develop before birth, remaining will develop afterbirth. Infants[13] born before this time tend to exhibit infant respiratory distress syndrome resulting from inadequate surfactant production. During fetal life, the lungs are filled with fluid and all respiratory exchanges are made by the placenta. Vascular shunts cause circulating blood to largely bypass the lungs. At birth, the filled fluid pathway empties, and therespiratory passageways are filled with air. As the PCO2 in the baby's blood rises, the respiratory centres are excited, causing the baby to take its first breath. "The alveoli becomes inflate and begins to function in gas exchange, but it is nearly two weeks before the lungs are fully inflated[14]".

RESPIRATORY SYSTEMFUNCTIONAL ANATOMY:

"The respiratory system contents upper respiratory tract and lower respiratory tract, They are the nose and nasal cavity, pharynx, larynx, trachea, bronchi and their smaller branches, and the lungs, which contain the terminal air sacs, or alveoli[15]". Functionally, the system consists of two zones. The respiratory zone, the actual site of gaseous exchange, is

composed of the respiratory bronchioles, alveolar ducts, and alveoli, all of which are microscopic structures[16]. The conducting zone contain all other respiratory passage, which provides fairly rigid conduits for air to reach the gas exchange sites.

Conducting Zone Structures:

The right and left main (primary) bronchi are formed by the division of the trachea approximately at the level of T7 in an erect position. Each bronchus runs obliquely in the mediastinum before plunging into the medial depression (hilum) of the lung on its own side. The right main bronchus is shorter , wider and more vertical than the left and is the more common site for an inhaled foreign object to become lodged[17]. By the time incoming air reaches the bronchi, it is warm, cleansed of mostpathogenes, dust, and saturated with water vapour.

Once inside the lungs, each main bronchus subdivided into lobar (secondary) bronchi,and then lungs had three on the right and two on the leftlober bronchi and then each are supplying one lung lobe. The lobar bronchi branch into third-order segmental (tertiary) bronchi, which divide repeatedly into smaller and smaller bronchi (fourth-order, fifth-order, etc.). Overall, there are about around 23 orders of branching air passages in the lungs. Smaller passages are more than 0.1 cm in diameter are called bronchioles (“little bronchi”), and the tiniest of these, the terminal

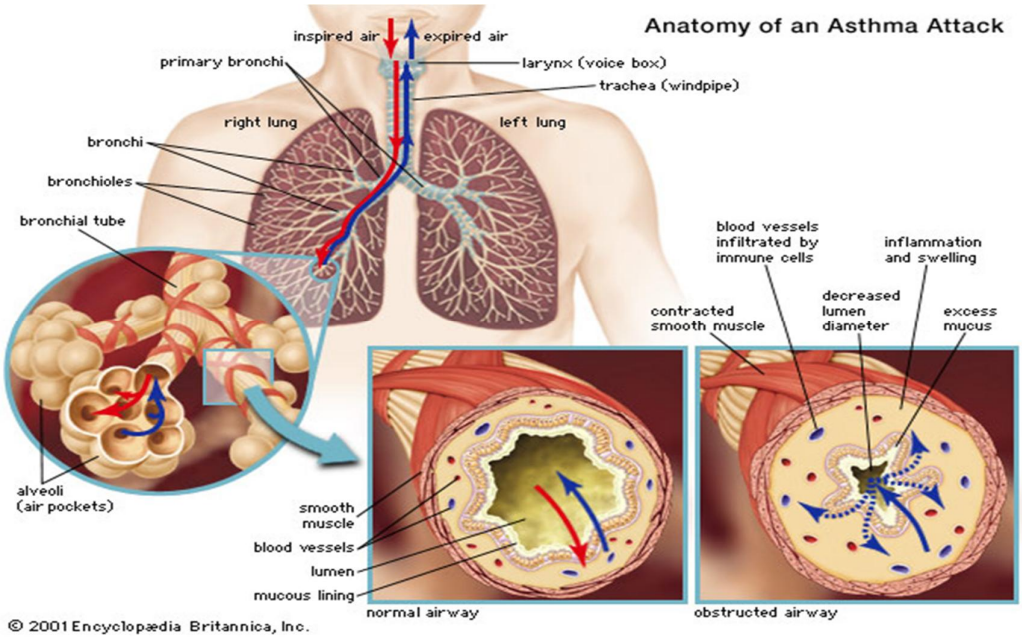
bronchioles, are less than 0.05 cm in diameter. Because of this branching pattern, “the conducting network within the lungs is often called the bronchial or respiratory tree[18]”.

Respiratory Zone Structures:

It is composed of a respiratory bronchioles, alveolar ducts, atria,sac and alveoli. There are about more than “300 million alveoli in the two lungs[19]“and each alveolus has an average diameter of about 0.02 centimetres. The alveolar walls are extremely very thin and between the alveoli is an almost solid networks of interconnecting capillaries.

The Respiratory Membrane:

The walls of the alveoli are composed primarily of a single layer of squamous epithelial cells, called type I cells, surrounded by a basement membrane. The alveoli external surfaces are densely covered with a “cobweb” of pulmonary capillaries. Together, “the alveolar and capillary walls and their fused basement membranes form the respiratory membrane, a 5-µm-thick air-blood barrier that has gas on one side and blood flowing past on the other”.



Gas exchanges occur mainly by simple diffusion across the respiratory Membrane,(o2) oxygens passes from the alveolus into the blood and CO2 leaves the blood to enter the gas-filled alveolus.

2. Magnitude of asthma:

Asthma has become an increasingly common problem with a global prevalence of 1-18% in different countries. It is estimated that around 300million people, worldwide, suffer from asthma. The prevalence increases by 50% every decade.

Studies show that the prevalence of asthma in Asian countries is around 2-4% as compared to high prevalence in countries like United Kingdom, Canada, Australia, New Zealand and other developed countries. The economic burden of asthma is estimated to be 1% of all

disability adjusted life year (DALY) lost worldwide. A worldwide increase in the prevalence of asthma is being reported at an alarming rate of 5% per year. In India the estimated prevalence is 5-10%.[20]

Asthma may have its onset at any age before puberty and is twice more common in boys in the pre pubertal age[21]. But the sex ratio equalized by age 30. Fifty percent of all asthmatic children are asymptomatic from 10-20 years of age but recurrences are common in adulthood.

The cost of treating asthma in patients places a considerable burden on the health resources owing to the cost of medications, hospitalizations and the time spent by parents or caretakers in looking after them.

BRONCHIAL ASTHMA

3.Definition:

The manner in which asthma has been defined has changed significantly over time.

- In 1950's asthma was defined as a disease characterized by airflow obstruction that could resolve spontaneously or following therapy[22]

- In 1960's asthma was viewed as an episodic disease in which airflow obstruction was caused by bronchial hyper responsiveness[23].
- In 1970's the concept of preventing bronchospasm and managing disease progression was considered.
- In 1990's asthma was redefined as a chronic inflammatory disease characterized by reversible airflow obstruction and bronchial hyper responsiveness.[24]

Asthma is "a chronic inflammatory disorder of the airways in which many cells and cellular elements play a vital role, in particular the mast cells, eosinophils, T lymphocytes, macrophages, epithelial cells ,other inflammatory mediator[25]." In susceptible individuals, the inflammation causes recurrent episodes of wheezing mainly early morning, breathlessness, chest tightness and cough mainly dry in nature particularly at night or in the early morning. These symptoms are usually associated with widespread but variable airway obstruction that is often reversible either spontaneously or with treatment. "The inflammation also causes an increase in the existing bronchial hyper responsiveness to a variety of stimuli". [26]

Asthma can be classified as

Three groups based on etiology

- Atopic (IgE mediated, triggered by allergens),
- Non Atopic (non IgE mediated, triggered by infection),
- Mixed and exercise or aspirin induced

ALLERGIC ASTHMA

Asthma is characterized by paroxysmal attacks of difficulty in breathing Accompanied by a sense of suffocation. In between the episodes the patient is symptom-free. An asthmatic attack can be provoked by a variety of factors which include allergens, emotional factors, physical strain, infections and exposure to sudden changes in temperature, climate and weather factors.

Allergy is said to be the cause in a significant percentage of asthmatic population. Allergy to chemicals, drugs, dust-mites, pollens, moulds, animal-dander, food items and insect bites/stings etc. can provoke an asthmatic attack. In most of the cases of asthma, typical type 1 reaction is seen. Thus ‘asthma is a variable obstructive but reversible disease which affects the air passages.

PREVALENCE:

An estimated 20 million Americans suffer from asthma, and 50% of asthma cases are “Allergic asthma”. “About one half of the cases develop before age of 18 and another third occur before age 40”[27].

ALLERGEN INDUCED ASTHMA:

In atopic asthmatic individuals with experimental inhalation of allergen leads to development of different types of reaction.

Immediate asthma (early reaction)

Airflow limitation begins within minutes of contact with the allergen, reaches its maximum in around 15-20 minutes and subsides byarounds 1 hour.

Dual and Late Phase reactions:

Following an immediate reaction many asthmatics develop more prolonged and sustained attack of airflow limitation that responds less well to inhalation ofbronchodilator drugs. Isolated late phase reactions with no preceding immediate responsecan occur after the inhalation of some occupational sensitizers, like isocyanates.

Recurrent asthmatic reactions:

The development of the late phase reaction is associated with an increase in the underlying level of airway hyper-responsiveness such that individuals may showcontinuing episodes of asthma on subsequent days[28].

ETIOLOGY:

Etiology factors of asthma are of two types. Some factors, called inducing factors cause initial development of asthma, whereas some other factors provoke an episode inpredisposed individuals suffering from asthma and these are called provoking factor or triggerfactors[29].

INDUCING FACTORS:

Genes:

Genetic factors are important inducing factors, where atopic individuals go in to risk factor for developing allergic asthma. A distinct gene for atopy on chromosome 5q,11q and 12q has been identified[30].

PREDISPOSING FACTORS:

Allergens:

Exposure to a specific allergen will precipitate an attack of asthma, particularly in young people having asthma. House dust mite is the

commonest allergen. Pollens, molds, animal dander and cockroaches are other common allergens provoking asthma. This typeof asthma is named as ‘allergic asthma’.

Infection:

It is well recognized that viral infections commonly cause attacks of asthma. Secondary bacterial infection is widely held to occur and perpetuate the inflammatory reactions which give rise to prolonged airway narrowing[31].

Environment:

Many patients with allergic asthma experience worsening of symptoms on contact with cigarette smoke, car exhaust fumes, strong perfumes or high concentrations of dust in atmosphere.

Allergic asthma exacerbations increase in both summer and winter, air pollution, episodes associated with climate temperature inversions, NO2 in presence of high concentrations in summer, No2 and So2 in the winter[32].

Occupation:

Its exposure to chemical agents (such as epoxyresins in plasticFactories or toluene diisocyanate in varnishes and paints) that either induces bronchoconstriction directly or via degranulation of mast

cells. This is called occupational asthma. It is characterized by a cyclic history, with symptoms becoming prominent towards the end of a work shift and an increase of symptoms after leaving the work site[33].

Exercise:

It is provoked by various forms of exercise such as running or climbing stairs[34].

Food:

Among Indians, ice and cola drink are reported to be more sensitive and causes bronchoconstriction. In some cases egg, milk and wheat also act as a triggering factor for allergic asthma[35].

Psychological Factors:

It is well known emotional factors may been influence asthma, but there is no evidence that patients with the disease are any more psychologically disturbed than theirnon-asthmatic peers[36].

Nocturnal:

It refers to attacks that predominantly occur during the night. It has been related to recumbency, a diminished mucociliary clearance, nocturnal airway cooling, exposure tonight time allergens, gastro esophageal reflex and a ‘morning dip’ in circulatingepinephrine and cortisol.

Drugs:

Beta blocking drugs, cholinergic drugs used for myasthenia gravis, and Prostaglandins (PGF2) usedforinducingabortion and drug induce gastritis areknowntoinduced bronchoconstriction[37].

NON ATOPIC ASTHMAOR IDIOSYNCRATIC ASTHMA[38]:

It also called as Intrinsic or late – onset asthma.

A significant fraction of patients with asthma present with no family or personal history of allergy, with skin tests was negative and with normal serum levels of IgE and therefore have disease that cannot be classified on the basis of currently defined immunological mechanisms. These patients are said to have idiosyncratic asthma or non-Atopic asthma.

Non-Atopic asthma, is divided into two groups

i. Mechanical obstruction groups
ii. Bacterial infection groups

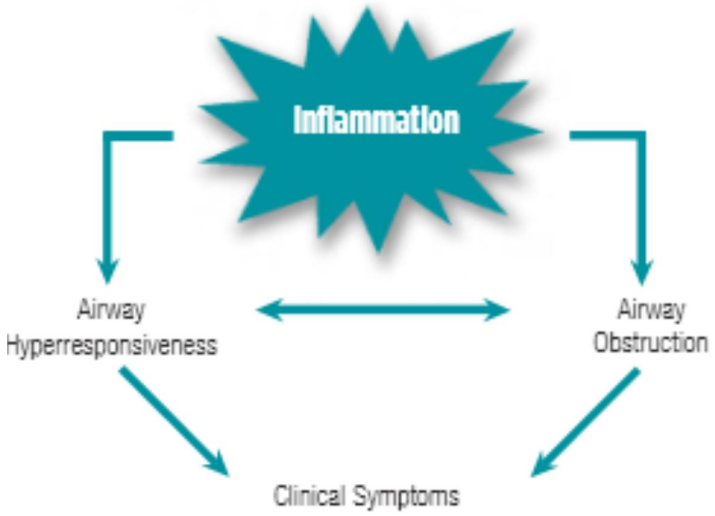
It is essential to having a clear idea of the immunological fundamentals, if one has to offer optical treatment to patients with allergic asthma, because this disease is partly due to the harmful effects of the immune system.

5. Pathophysiology:

The complex mechanisms involved in pathophysiology of asthma include:

1. Airway inflammation
2. Intermittent airflow obstruction
3. Airway hyper responsiveness

Asthma Pathogenesis



Airway narrowing is the final common pathway for all the symptoms and physiological changes in asthma. Many factors are associated with narrowing of airways.

Airway obstruction is mainly due to

- Edema and inflammation reponses of mucous membrane lining the airways
- Increased secretions of mucus and inflammatory cells
- Contraction in the smooth muscles of bronchi
-

Airway obstruction is usually diffuse but not uniform. In many adults with asthma, both larger and smaller airways are obstructed, though some patients may have exclusive smaller airway disease.

Airway obstruction results in increased resistance to airflow and decreased flow rates contributing to hyperinflation. Overinflated lungs compensate for pulmonary obstruction to some extent, but when the tidal volume exceeds the pulmonary dead space, it leads to alveolar hypoventilation.

The pulmonary blood flow is affected in hyperinflation because of increased intra alveolar and intra pleural pressure leading to uneven pulmonary circulation. Thus increased intra alveolar pressure along with decreased ventilation and perfusion, leads to V-P (ventilation-perfusion) mismatch in various lung units.

Ultimately all these factors contribute to the development of hypoxia. CO_2 retention occurs only in the later stages when the obstruction is more severe.

HYPERSENSITIVITY

Immunologic tissue injury in response of allergen exposure.

Classified in to following types are

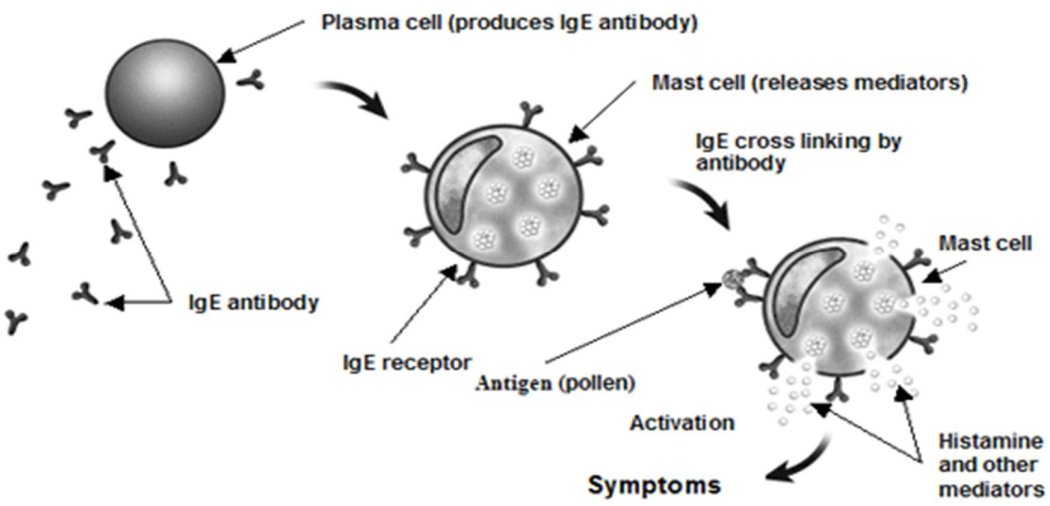
1) Type 1 immediate or reaginic
2) Type 2 cytotoxic
3) Type 3 IgG or IgM mediated
4) Type 4 antibody dependent cell mediated cytotoxicity
5) Type 5 stimulatory/ blocking reactions

Mechanisms[39]

TABLE 19.1 **Types of Hypersensitivity**

Type of Reaction	Time Before Clinical Signs	Characteristics	Examples
Type I (anaphylactic)	<30 min	IgE binds to mast cells or basophils; causes degranulation of mast cell or basophil and release of reactive substances such as histamine	Anaphylactic shock from drug injections and insect venom; common allergic conditions, such as hay fever, asthma
Type II (cytotoxic)	5–12 hours	Antigen causes formation of IgM and IgG antibodies that bind to target cell; when combined with action of complement, destroys target cell	Transfusion reactions, Rh incompatibility
Type III (immune complex)	3–8 hours	Antibodies and antigens form complexes that cause damaging inflammation	Arthus reactions, serum sickness
Type IV (cell-mediated or delayed-type)	24–48 hours	Antigens cause formation of T_C that kill target cells.	Rejection of transplanted tissues; contact dermatitis, such as poison ivy; certain chronic diseases, such as tuberculosis

Type 1 Hypersensitivity pathophysiology

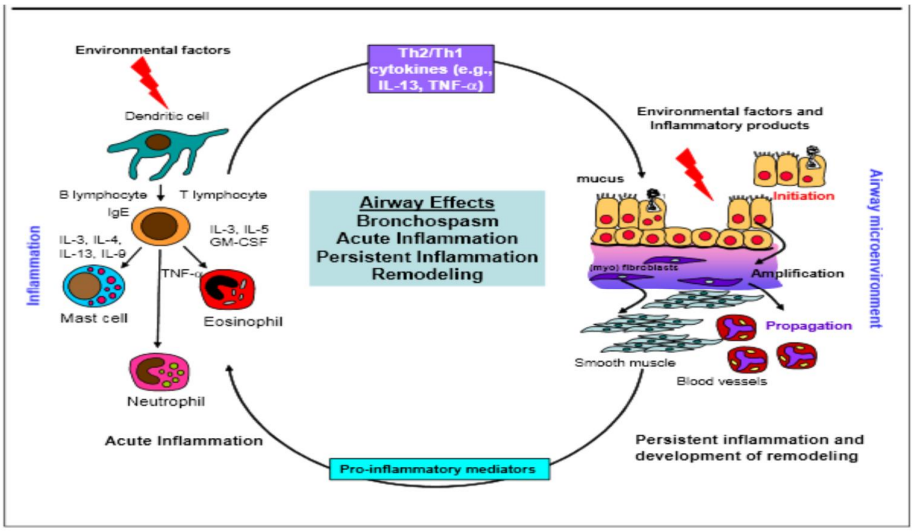


Clinical classification

Four groups based on frequency of symptoms,number of time emergence visit and hospitilization, severity of attack and pulmonary function tests abnormality

1) Intermittent asthma
2) Persistent asthma
3) Acute exacerbation: mild, moderate, severe asthma,
4) Specialvariants: there are 5 types
    a) Cough variant asthma
    b) Exercise induced asthma,
    c) Occupational asthma,
    d) drug-induced asthma (aspirin, other NSAID, beta blocker
    e) Seasonal asthma

Pathophysiological mechanisms at the level of molecule:



Key: GM-CSF, granulocyte-macrophage colony-stimulating factor; IgE, immunoglobulin E; IL-3, interleukin 3 (and similar); TNF-α, tumor necrosis factor-alpha

Inhalation of allergens leads to biphasic response by early and late phases

EARLY	LATE
1.Occurs in 30 min-2 hour 2.Mast cell degranulation and release of preformed mediators 3.Histamine, LTC4, D4, E4, Platelet Activating factor. 4.Broncho constriction	1. Occurs in 6-8 hrs. 2. Release of cytokines & newly generated Mediators. 3.Eosinophilic cationic protein, eosinophilic chemotactic factor. 4. Continued airway hyper responsiveness, Mucus secretion and vasodilatation.

6. Diagnosis of asthma:

CLINICAL EVALUATION OF ASTHMA:

HISTORY:

- The key to the detection and clinical evaluation of asthma is a careful history. Notonly can the physician usually elicit a description of symptoms that are sufficiently characteristic to make a diagnosis, but it may be possible to obtain important information about agents that is responsible for worsening symptoms, so that they can be avoided.
- Physician also establishes trusting relationship with the patient, so that therapeuticprogram will be followed by the positive effect of the physician's optimism and reassurance.

PRESENTING SYMPTOMS:

Characteristically patients with asthma have some combination of dyspnoea, wheeze, chest tightness, cough – symptoms may follow exposure to specific agents such as inhaled (or) ingested antigens (or) viral infection. More often symptoms develop without identifiable cause. One of the most common features is development (or) worsening of symptoms at night. It is known that both normal people and asthmatic patients have circadian rhythms of peak expiratory flow rate (PEFR) – PEFR lowest between 3 and 6 am. So patients wake up in morning with

symptoms mainly wheezing and breathing difficulty, nocturnal cough in asthma patients.

Dyspnoea:

Shortness of breath is extremely common but not invariable complaint of patients with asthma. It is likely related to the scene of effort for ventilation and function of the severity of airway obstruction. Dyspnoea is paroxysmal and most commonly occur late night. The patient may be restless agitated, anxious, sweating, orthopnoic and tachypnoeic, breathing through pursed lips with a prolonged expiration and using accessory muscles of respiration.

Wheeze:

The high velocity of flow through narrowed large airways produces wheeze which is often first audible to the physician. Many patients may not have wheeze during periods of asthma, but its absence should not exclude consideration of diagnosis.

Tightness:

One of the characteristic symptoms of asthma is the sense of chest tightness. Patient commonly volunteer that their symptoms are associated with this sensation of tightness. It is likely that this sensation reflects the

excessive activity of the vagal receptors known to be a fundamental feature of asthma.

Cough:

Cough is usually non-productive, frequently associated with expectoration ofmucoid sputum, at times quite frothy and liquid.

Upper Airways Symptoms:

Although asthma is primarily a disorder of the intrathoracic airways, some patients do indicate that their symptoms originate in the region of larynx. There are evidences that this is narrowing of the extra thoracic upper airways in asthma and that may contribute to the reduction of inspiratory and expiratory flow rate. Although this may not be a frequent cause of flow limitation in patients, inspiratory stridor may be present in asthma or may be the presenting complaint and needs to be evaluated.

ACUTE SEVERE ASTHMA / STATUS ASTHMATICUS:

It is life-threatening emergency that places the patient at risk of develop ingrespiratory failure. Patients are severely dyspnoeic, cyanosed and often moribund. Any patient of asthma may develop acute severe asthma, as a complication. It may be precipitated by infection, allergic factors or psychological stress. Even in mild cases such paroxysms may

supervene without warning. The attacks usher in either as progressive worsening of an existing paroxysm or sudden onset of severe dyspnoea and air hunger in a mild or moderate asthmatic.

Widening of intercostal spaces, decrease in lateral expansion of the chest, tachynoea, tachycardia and fatigue are features of asthma during attack. And decrease in cardiac output, pulses paradoxus, tachycardia, hypoxemia, respiratory acidosis are features of asthma during severe attack.

Physical Examination:

i) Position of patient in which shoulder girdle is fixed.
ii) Accessory respiratory muscles are actively involved in respiration.
iii) The chest is held in near to end inspiratory position.
iv) Percussion note may be unaltered or it may become hyper resonant with Progression of disease.
v) Breath sounds will be obscure with lots of added sounds, rhonchi.
vi) Expiration will be prolonged.
vii) In chronic cases, the chest may be pigeon shaped.

CHRONIC ASTHMATIC:

(i) Chest is barrel shaped

(ii) Shoulders are rounded,

(iii) Usually the patient is of small stature,

(iv) Usually the patient breathes from his mouth,

(v) He/She is a daily wheezer (He coughs more at night with varying amounts of mucous and plugs).

LABORATORY INVESTIGATION:

Blood for Absolute Eosinophil Count:

It one of components of white blood cells is called eosinophils, in the blood, where there is increase in eosinophils in the circulating blood is called eosinophilia.it ranges from 1-6% of eosinophils and 40-450/cumm of absolute eosinophil count[40].

It is calculated by percentage of eosinophils multiplied by the white blood cell count to give the absolute eosinophil count.

Significance:

Eosinophilia is common in bronchial asthma regardless of whether allergic factors can be shown to have an etiologic role. In many asthmatics, the degree of eosinophils may correlate with the severity of asthma.

Other blood investigations finding includes,

Leucocytosis, indicates infection, serum IgE levels will also be high. But these are not specific for asthma, until and unless other infections are ruled out.

Sputum Examination:

A wet preparation of sputum of many asthmatic patients contains spiral casts (Curschmann's spirals), eosinophilia and Charcot Leyden crystals. Presence of sputumand blood eosinophilia is suggestive of the diagnosis of asthma[41].

X-ray chest:

Usually normal x-ray

It may show hyper-inflation. It also reveals complications of severe asthma such As Rib fracture, Pneumothorax and Pneumo mediastinum.

Arterial blood gas Analysis:

Respiratory alkalosis followed by hypoxia with severe respiratory distress, compromises the PCO2 and PH become normal, finally respiratory acidosis develops with hypercapnia and severe hypoxia. Increase in PCO2 level is a bad prognosis.This for to assess the severity for asthma.

Pulmonary Function Test[42]:

It's also lung function test are useful in assessing the functional status of the respiratory system both in physiological and pathological condition.

Types

1) Static lung function tests

Based on volume of air that flow in to or out of lungs.

2) Dynamiclung function test

Based on time ,i.e. the rate at which air flows into or out of lungs.

LUNG VOLUMES:

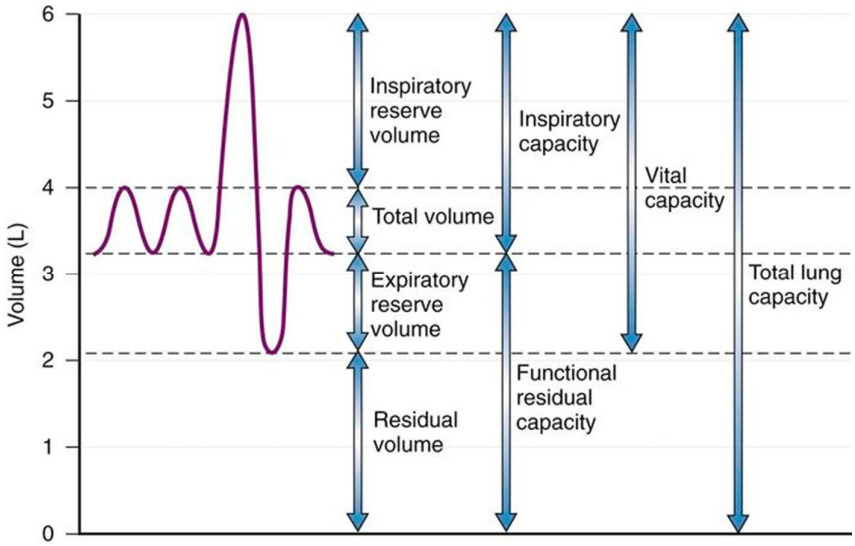
1) Tidal volume
2) Inspiratory reserve volume
3) Expiratory reserve volume
4) Residual volume

LUNG CAPACITIES:

1) Inspiratory capacity
2) Vital capacity
3) Functional residual capacity
4) Total lung capacity

Spirogram :

It is the graphical record of lung volumes and capacities using spirometer. Upward deflection of the spirogram denotes inspiration and the downward curve indicates expiration.



Disadvantages :

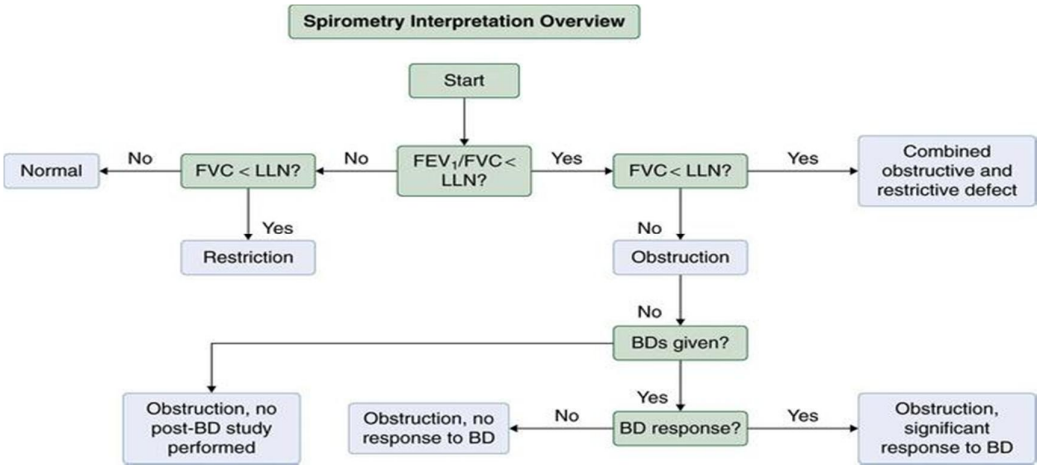
Volume, which cannot be measured by spirometry, is the residual volume.

Plethysmography

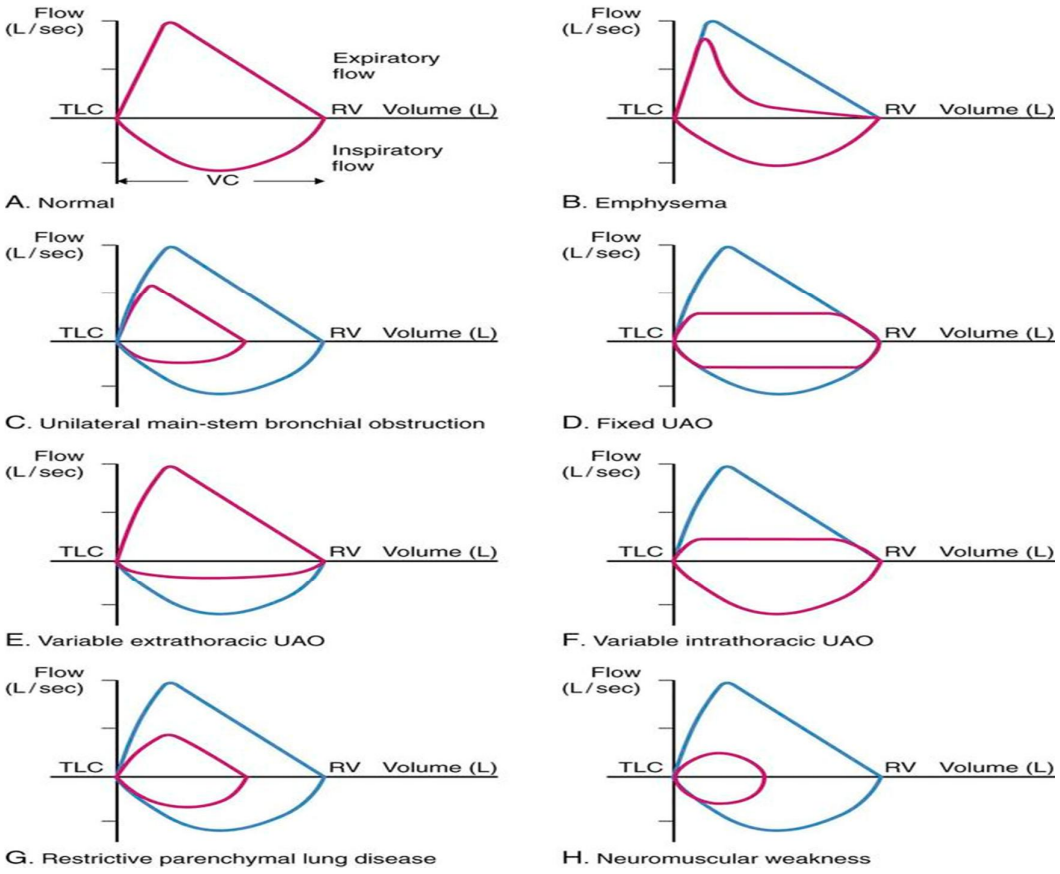
It is a technique used to measure all the lung volumes and capacities.

Measurement of functional residual capacity and residual volume:

1) Helium dilution technique
2) Nitrogen washout method
3) Plethysmography



Tables show to diagnosis restrictive and obstructive lesion.



This diagram shows variable pattern of airway diseases.

During the attack, the lung volume is reduced; with a decrease in the forced vitalcapacity (FVC); the maximal mid expiratory flow rates (MMF) and the forced expiratory volume in one second (FEV1).The FEV1 / FVC ratio is usually less than 75%. The RV/TLC ratio increased owing to air trapping.

1. Skin Sensitivity Testing's:

These tests are done to find out the possible allergens to which he or she is Allergic. A small quantity of allergen is put on skin which is pricked. In positive cases, a wheal is seen at the site after 20-40 minutes of introduction of the allergens in minute doses, in skin by pricking the skin.

2. Radio-Allergosorbent Test (R.A.S.T.):

The importance of these tests comes to play when dermal reactivity tests are Inconclusive. In allergic individuals, the levels of IgE are raised, when exposed to allergens. When skin reactivity tests are inconclusive, one can come to significant Conclusion by measuring IgE specific to individual allergens by RAST.

3. Challenge tests:

a. Allergen / antigen challenge tests:

Challenge or provocation tests are more accurate then the above stated tests.

These tests are done in emergency. The antigens are introduced through inhalation with the help of a nebulizer. Within few minutes (7 to 15 minutes) in positive cases, there will be a fall of 20% or more in FEV1 PEFR indices.

b. Exercise challenge tests:

In positive cases there is a decrease in FEV and PEFR indices after physical Exercises. This decrease is of about 20% of more and is maximal 5-10 minutes, after exercise and recovers to normal over a course of time of 30-60 minutes13.

Though asthma is diagnosed by clinical symptomatology, lung function tests are helpful in supporting the diagnosis in doubtful cases and also for monitoring the control of asthma.

Grading and classification of Asthma:

1. Grading of Severity of Asthma

   Grading can be done based on

   (1) Symptoms of air flow obstruction

   (2) Night symptoms

   (3) Peak expiratory flow rate (personal best and diurnal variation).

Based on the severity, asthma can be graded as:

Grade 1: Mild intermittent asthma

Grade 2: Mild persistent asthma

Grade 3: Moderate persistent asthma

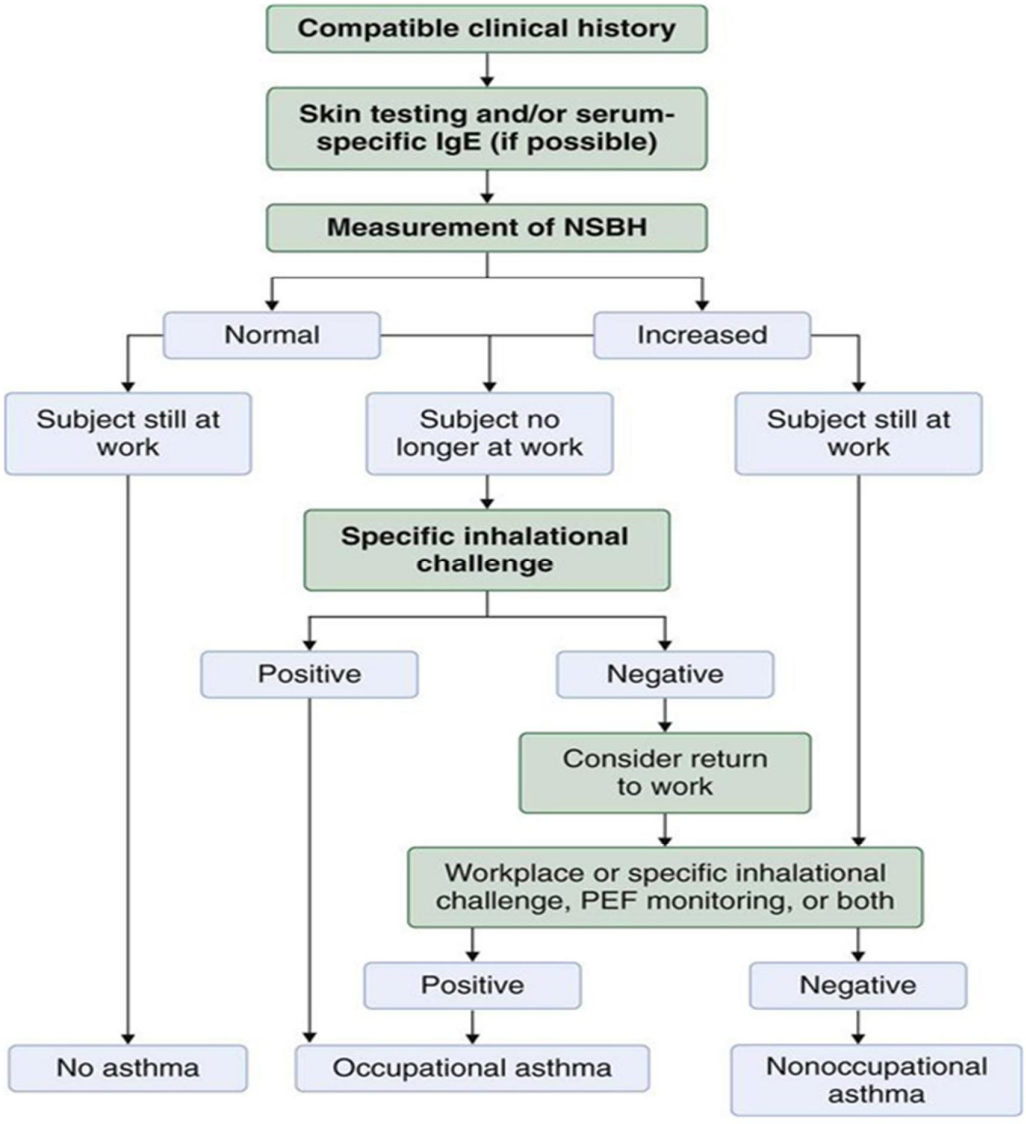
Grade 4: Severe persistent asthma

Classification based on severity[43] GINA

Classification	Days with Symptoms	Nights with symptoms	PEFR	PEFR variability (%)
Mild intermittent	≤2/week	≤2/month	≥80%	<20%
Mild persistent	>2/week but <1/day	>2/month	≥80%	20-30%
Moderate persistent	Daily	>1/week	60-80%	>30%
Severe persistent	Continuous	Frequent	≤60%	>30%

Assessment of control of asthma (GINA guidelines 2012) Asthma control was categorized as uncontrolled, partly controlled and controlled as per guidelines by Global Initiative for Asthma Guidelines.

In particular, the levels asthma control were defined depending on the presence/absence of daytime symptoms, limitation of activities, nocturnal symptoms/awakening, need for reliever/rescue treatment, and FEV_1 /PEFR results in the last four weeks.



Assessment and monitoring

1. Asthma Clinic examination
2. Lung function testing
    - Spirometry
    - Peak expiratory flow monitoring

PEFR is the maximum flow rate generated during a forceful expiration. PEFR primarily reflects large airway flow. FEV over one second is a dynamic measure of flow used in formal spirometry. Although peak flow rate usually correlate well with $FEV_{1,}$ this correlation decreases in patients with asthma as airflow limitation is diminishes.

Daily PEFR monitoring may assist the early changes in asthma that may require therapy, evaluating responsiveness to changes in therapy and giving a quantitative measurement of improvement.

PEFR was pioneered by Martin Wright for assessing lung function.

Peak expiratory flow (PEF) monitoringdevices provide a simple and inexpensive tool used to measure airflow at home. Children aged 5 and above are able to use a peak flow meter. PEFs vary in their ability to detect airflow obstruction. In some patients, PEF declines only when airflow obstruction is severe. So it is advisable to monitor PEF by measuring morning and evening PEFs. Best of three consecutive attempts

should be noted as the final value. Variation in PEF >20% between morning and evening value is consistent with the diagnosis of asthma.

Procedure for using Peak Flow Meter:

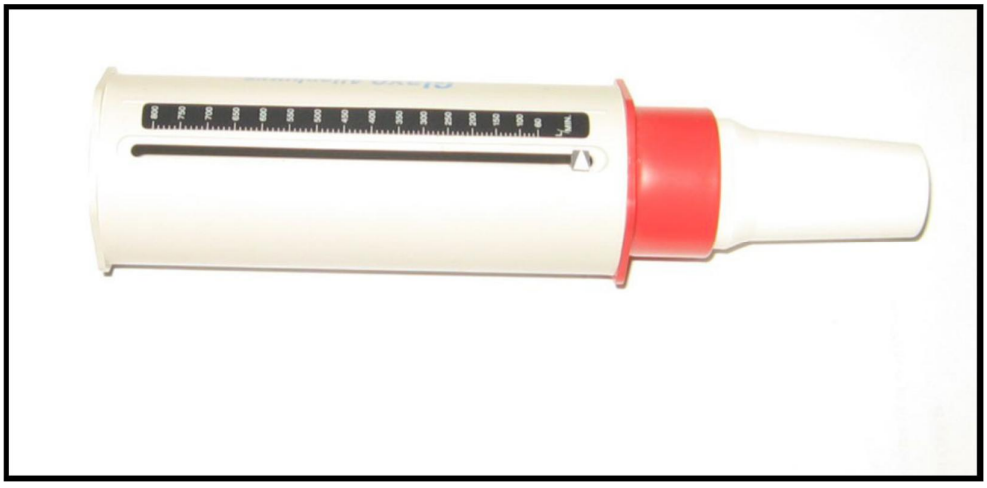
1. Move the indicator to the bottom of the numbered scale.

2. Patient is asked to stand up and take a deep breath,airflow filling the lungs

3. Mouth piece is placed in the mouth and the patient is asked to firmly close his lips around it without putting the tongue inside the hole.

4. Then the patient is asked to blow out as hard and fast as possible in a single blow. The value should be written down. The steps 1 to 4 should be repeated 2 more times to get the personal best peak flow reading

PEAK FLOW METER



Predicted PEFR values according to height in male:

Ht (cm)	PEFR(Lt/min) Boys	Ht (cm)	PEFR(Lt/min) Boys	Ht (cm)	PEFR(Lt/min) Boys
101	120	134	235	167	351
102	123	135	239	168	354
103	127	136	242	169	358
104	130	137	246	170	361
105	134	138	249	171	365
106	137	139	253	172	368
107	141	140	256	173	372
108	144	141	260	174	375
110	148	142	263	175	379
111	151	143	267	176	382
112	155	144	270	177	386
113	158	145	274	178	389
114	162	146	277	179	393
115	165	147	281	180	396
116	169	148	284	181	400
117	172	149	288	182	403
118	176	150	291	183	407
119	179	151	295	184	410
120	183	152	298	185	414
121	186	153	302	186	417
122	190	154	305	187	421
123	193	155	309	188	424
124	197	156	312	189	428
125	200	157	316	190	431
126	204	158	319	191	435
127	207	159	323	192	438
128	211	160	326	193	442
129	214	161	330	194	445
130	218	162	333	195	449
131	221	163	337	196	452
132	225	164	340	197	456
133	228	165	344	198	459
134	232	166	347	199	463

Predicted PEFR values for according to height in female

Ht (cm)	PEFR (lt/min) Girl	Ht (cm)	PEFR (lt/min) Girl	Ht (cm)	PEFR (lt/min) Girl
101	116	134	225	167	334
102	120	135	229	168	337
103	123	136	232	169	341
104	126	137	235	170	344
105	130	138	238	171	347
106	133	139	242	172	351
107	136	140	245	173	354
108	139	141	248	174	357
110	143	142	252	175	361
111	146	143	255	176	364
112	149	144	258	177	367
113	153	145	262	178	370
114	156	146	265	179	374
115	159	147	268	180	377
116	163	148	271	181	380
117	166	149	275	182	384
118	169	150	278	183	387
119	172	151	281	184	390
120	176	152	285	185	394
121	179	153	288	186	397
122	182	154	291	187	400
123	186	155	295	188	403
124	189	156	298	189	407
125	192	157	301	190	410
126	196	158	304	191	413
127	199	159	308	192	417
128	202	160	311	193	420
129	205	161	314	194	423
130	209	162	318	195	427
131	212	163	321	196	430
132	215	164	324	197	433
133	219	165	328	198	436
134	222	166	331	199	440

Asthma were divided into three groups depending on levels of control (GINA guidelines 2012)

Characteristics	**Controlled (All of the following)**	**Partly controlled (Any measure present)**	**Uncontrolled**
Daytime symptoms	None (twice or less/week)	More than twice/week	Three of more features of partly controlled
Limitation of activities	None	Any	
Nocturnal symptoms /awakening	None	Any	
Need for reliever/ rescue treatment	None (twice or less/week)	More than twice/week	
Lung function (PEFR)	Normal	<80%predicted or personal best(if known)	

7. Role of EOSINOPHIL in Asthma

Chronic airway inflammation as evidences by cellular infiltration of airway by activated t lymphocytes, mast cells and macrophages and mainly eosinophils and other mediators. Eosinophils are white blood cells and immune system components responsible and also control mechanisms associated with allergy and asthma.

The eosinophil is a specialized cell of the immune system first recognized by “Paul Ehrlich” approximately 130 years ago. The eosinophil a nucleus with two lobes, and cytoplasm filled with

approximately more than 200 large granules containing enzymes and proteins with variable functions.

Eosinophils are one of the components of white blood cell, where there is increase in eosinophils in the circulating blood called eosinophilia.it ranges from 1-6% of eosinophils and 40-450/cu mm of absolute eosinophil count.

It is calculated by percentage of eosinophils multiplied by the white blood cell count to give the absolute eosinophil count.

Eosinophils increased in parasite infection, allergy, asthma, etc. Its are response for disintegration, detoxification, removal of foreign proteins and other inflammatory responses.

Mechanism of action of eosinophils

Eosinophils are neither markedly motile nor phagocytic like the neutrophils. Still eosinophil attack them by some special type of cytotoxic substances present in their granules. The lethal and released at the time of exposure to parasite or foreign protein are

1) eosinophil peroxidase: this enzyme is capable of destroying helminths, bacteria and tumour cells.

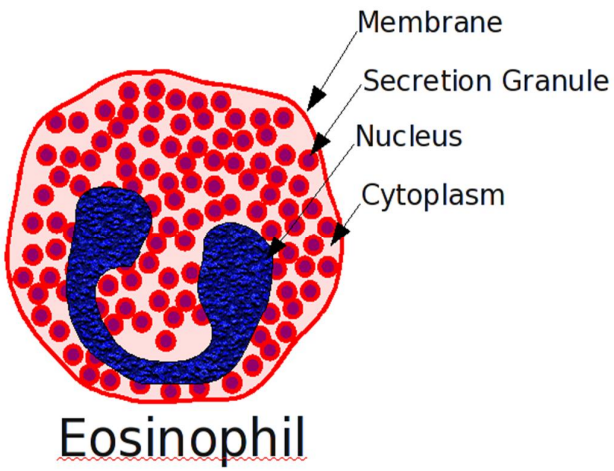
2) Major basic protein (MBP): Very active against helminths.its causing distension (ballooning) and detachment of tegumental sheath (skin like covering) of these organisms.

3) Eosinophil cationic protein (ECP): This substance is major destroyer of helminths and 10 times more toxic than MBP. It's also neurotoxin.

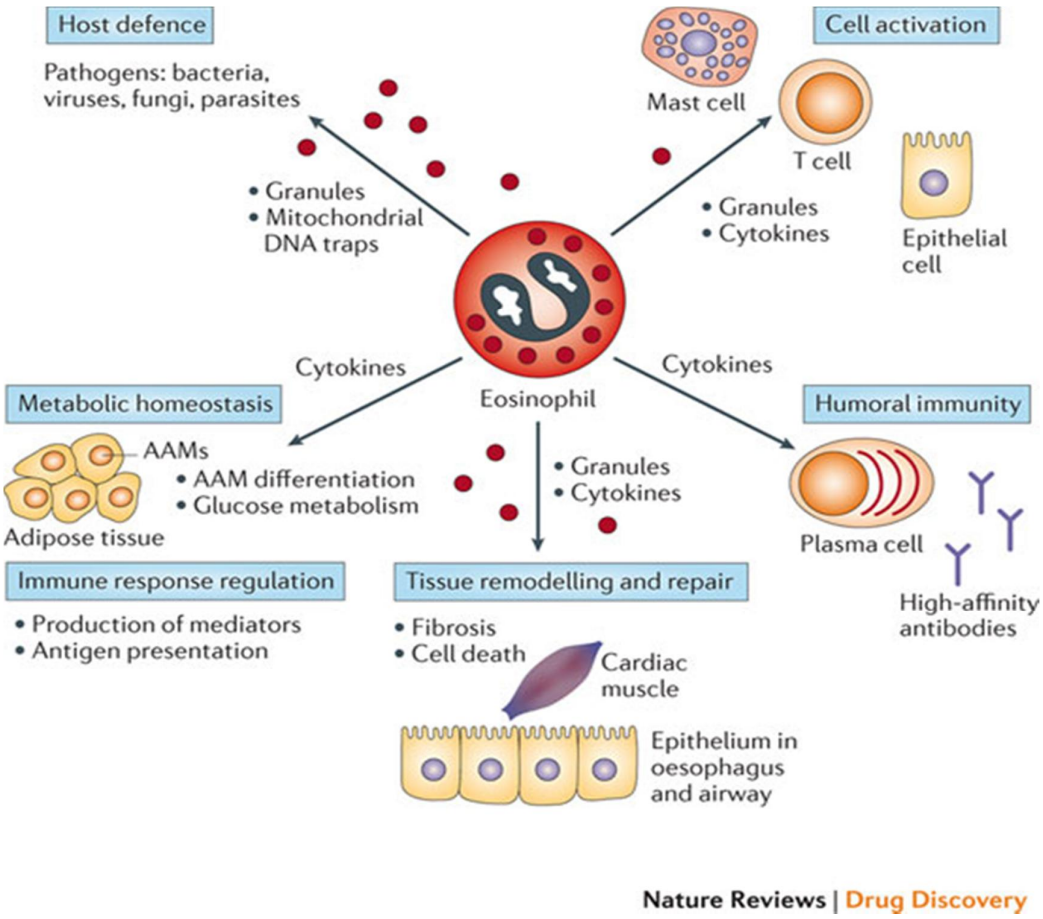
4) Eosinophil-derived neurotoxin: its destroys the nerve fibre mainly myelinated nerve fibers.

5) Cytokine: like IL-4,IL-5 accelerate inflammatory responses.

Diagram of eosinophil



Mechanism of eosinophil:



8. Management of asthma:

Goals of therapy:

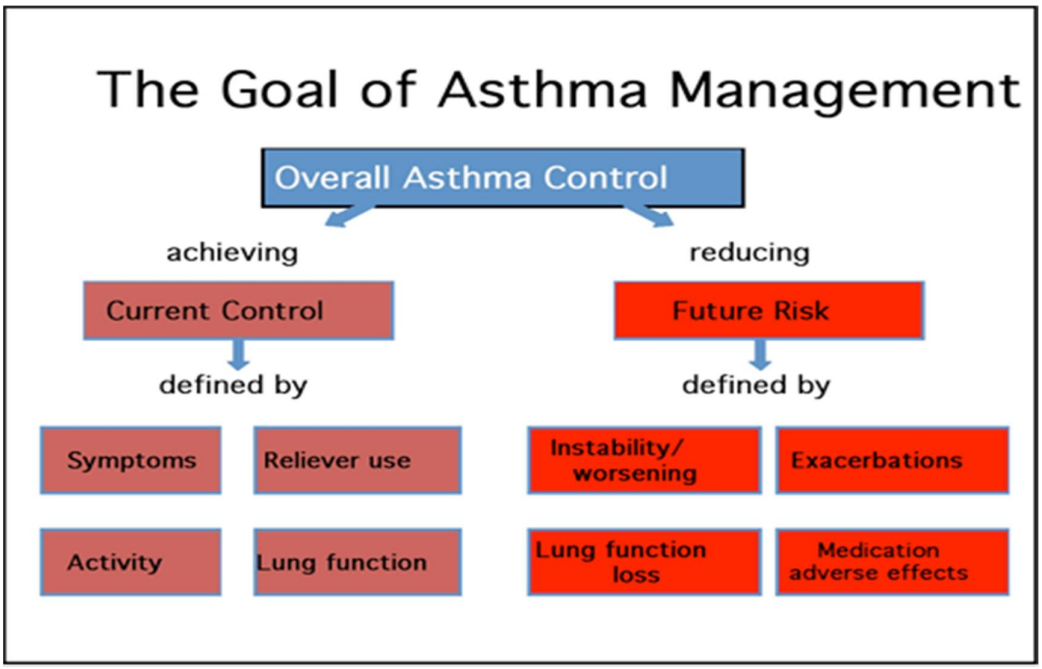
Themain goals for the prevention and management of asthma are as follows:

1. Preventing chronic and troublesome symptoms, such as coughing or breathlessness at night, in the morning or after exertion.

2. Maintaining nearly normal pulmonary function.

3. Maintaining normal activity levels (including exercise).

4. Preventing recurrent exacerbation and minimizing the need for emergency care and hospitalization.



Successful management of asthma patient depends on various factors

1. Asthma exhibits considerable heterogeneity with respect to ethology, clinical presentation, severity, and response to therapy. So it is very difficult standardize the management protocol. Each patient requires tailor made management strategies, depending on the symptoms.

2. Asthmatic have varying degrees of symptomatology at different points of time hence a close monitoring is required for appropriate adjustment of therapy.

Management of asthma practically divided into two parts

A. Non pharmacological therapy

B. Pharmacological therapy

A. Non pharmacological therapy

Recent studies suggest that educating the patient /parent about the disease along with measures to implement environmental control programs are very effective in reducing asthma morbidity and mortality.

Education:

Counselling the patients about nature of the disease and the importance of compliance for its successful management has rendered fruitful results.

Environmental control:

In adults who remain symptomatic despite intense therapy, measures to control environmental triggers like avoidance of aero allergens, air pollution, some drugs have helped in long term remission.

Vaccination:

Inactivated influenza vaccine may safely be given to asthmatic , in order to reduce asthma exacerbations.

Immunotherapy:

Allergen immunotherapy also appears to be of benefit in some patients with defined allergic triggers.

Pharmacological therapy

Medications

1. Corticosteroids

Inhaled corticosteroids are the mainstay of treatment for long term control of asthma. Glucocorticoids are not bronchodilators but reducing bronchial hyperreactivity, inflammatory effect like antigen: antibody reaction, mucosal edema, other trigger stimuli

Inhaled corticosteroids (ICS):

Beclamethasonedipropionate, Triamcinolone acetonide,

Budesonide, Flunisonide, Fluticasone,Ciclesonide .

Systemiccorticosteroids:

hydrocortisone, prednisolone, dexamethasone,etc

it's useful in severe chronic asthma, statusasthmaticus/acute severe asthma

2. Leukotriene antagonists.

It is usually given as an add on therapy with low dose ICS in insufficiently controlled patients. Drugs are Montelukast, Zafirlukast,

3. Short acting and Long acting inhaled beta 2 agonists

In asthmatic who are not controlled adequately on medium dose ICS, this drug can be used as an add on therapy. But mono therapy on long term basis is best avoided. Long acting beta agonists preferably used inhalation form.

- **Short durations** are epinephrine, isoetharine, isoproterenol
- **Intermediate duration** are salbutamol, terbutaline, metaproterenolalbuterol, pirbuterol, bitolterol, fenoterol, etc.
- **long duration** are salmeterol, formoterol, etc.

4. AntiIgE (omalizumab)

This recently introduced drug is shown to be effective in moderate and severe persistent asthma .dose 30-700 iu/ml based on IgE level

5. methylxanthines

a. oral theophylline

b. IV Aminophylline

6. Anticholinergics:

Ipratropium bromide by MDI,mainly useful in drug induced asthma e.g. Beta blockers

7. Lipoxygenase inhibitor:

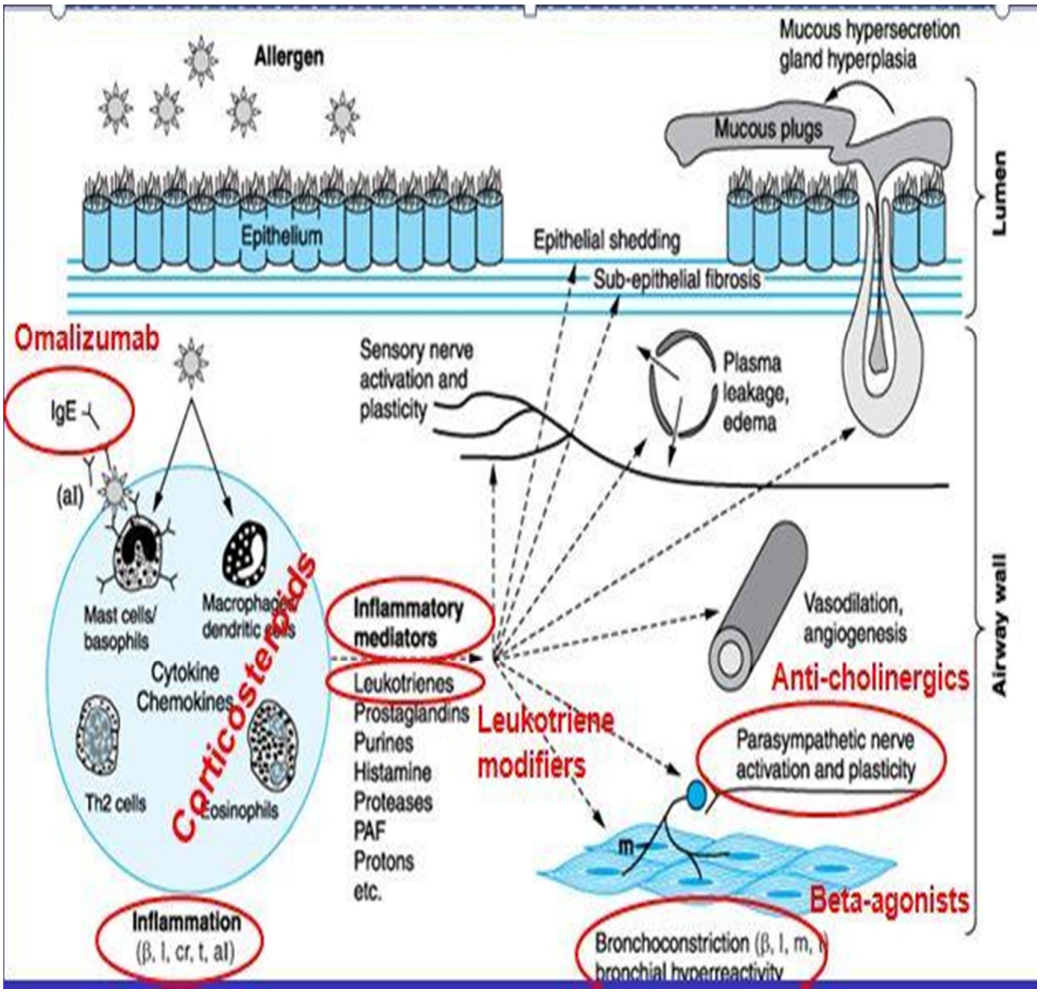
Zileuton 600mg qid reserved for severe asthma.

8. Alternative medication:

Methotrexate, cyclosporine, tacrolimus, mycophenolate mofetil,

Reliever medications:

Rapidly acting inhaled beta 2 agonists and oral short acting beta 2 agonists are used in acute episodes. Rapidly acting inhaled beta 2 agonists are most effective bronchodilators for the relief of acute symptoms.



Pregnant Women

Pregnant women with asthma must control the disease and ensure a good supply of oxygen to their babies. Poor asthma control also the risk of preeclampsia, and also develops high blood pressure and proteinuria. Poor asthma control increases the risk of low birth weight.

Differential diagnosis for asthma:

1) Its may be Airway Foreign Body, but localized sign and symptoms
2) Its may be Allergic reaction
3) Its may be Alpha1-Antitrypsin Deficiency, its main associated with COPD
4) Aspergillosis,fungal infection with high eosinophil levels
5) Bronchiectasis, Its associated with productive sputum with postural Chances and signs
6) its can be Bronchiolitis
7) Chronic Obstructive Pulmonary Disease, similar to asthma.
8) its may be Churg-Strauss Syndrome
9) its can be Cystic Fibrosis
10) Gastrooesophageal Reflux Disease, main associate with peptic ulcer diseases
11) Heart Failure its associate with sign and symptoms
12) Pulmonary Embolism its associate with cardiac or renal or other precipitating factors
13) its may be Pulmonary Eosinophilia associated with increased eosinophils count, sputum eosinophilia, worm infection
14) it's may be Sarcoidosi, Its can be Sinusitis, Upper Respiratory Tract Infection, etc,

This tables show Different between asthma and COPD

	ASTHMA	COPD
Age of onset	Any (usually under 35)	Usually after 40
First symptom	Usually shortness of breath and wheeze	Usually chronic cough and expectoration
Attack triggers	Common and marked	Less marked
History of smoking	Any	Common
Freedom from symptoms between attacks	Usually yes	Usually not
Hyperinflation	Rare	Common
Cor pulmonale	No	May be present
Chronic hypercapnea	No	May be present
Peripheral eosinophilia Raised IgE	Common	No
DLCO	Normal or high	Usually low
Chronic hypoxemia or hypercapnea	No	Common in advanced cases
Response to inhaled corticosteroids	Usually yes	No

AIM OF THE STUDY

Aim:

To study the correlation of absolute eosinophil count with asthma control in adult

MATERIALS AND METHODOLOGY

a) Study design:

Cross sectional study

b) Place:

Asthma Clinic,
Govt Royapettah Hospital,
Kilpauk Medical College, Chennai

c) Period of study:

April 2014 to September 2014

d) Study population:

Aged 18 to 75 years attending asthma clinic.

e) Sample size: 40 patients

f) Inclusion criteria:

All patients with asthma, aged 18 to 75 years.

g) Exclusion criteria:

1. pulmonarytuberculosis, drug allergy patients,

2. acute infections, chronic inflammatory disorders, chronic systemic diseases and malignancies

3. Immunocompromide state,

4. cardiac failure, pulmonary edema, pulmonary embolism, COPD,

5. Systemic Fungal infection, worm infestation, etc

h) Methodology:

1. All asthma patients aged between 18 to 75 years .

2. Detailed history and clinical examination was done.

3. Peak expiratory flow rate (PEFR) was measured by using peak flow meter.

4. Asthma control was assessed based on the parameters of GINA guidelines.

5. Informed consent was obtained and blood samples were taken for measuring serum absolute eosinophil count.

6. Under strict aseptic precautions, 5 ml of venous blood was drawn.

7. Absolute eosinophils count in peripheral blood.

8. Data entry form was filled, data was analyzed and statistical significance of the result was determined.

i) Statistical analysis

1. One way Anova followed by TUKEYHSD method for quantitative variables

2. Chi square test for qualitative variable

3. Correlation was done.

4. Based on this data statistical significance was determine

PROFORMA

1. SR. No.

2. Name:

3. Age:

4. Sex:

5. Address:

6. Occupation

7. Chief Complaints:

   i. Cough /wheeze
   ii. Nighttime symptoms and awakening
   iii. Daytime symptoms
   iv. Limitation of activities
   v. Need for reliever medications

8. Similar complaints in the past:

9. Hospitalisation: ----times

10. Emergency visit: ----times

11. Seasonal variation: Yes/No

12. Sleep disturbed due to cough, breathlessness: Yes/No

13. Nature of attack: After exercise/ Cough/ Cold/ Associated with fever.

14. Associated conditions: Rhinosinusitis/ GERD/ obesity

15. Socio economic condition

16. Precipitating factors

    Allergen

    Indoor : Domestic mites, cockroach,

    Furredanimals (dogs, cats, mice)

    Outdoor: Pollens, fungi, molds, yeasts

    Infections (predominantly viral)

    Occupational

    Diet

    Tobacco smoke (active and passive)

    Indoor/outdoor air pollution

    Strong odours(perfumes), Smoke, mosquito repellents

17. **EXAMINATION**

GENERAL EXAMINATION

- Built & nourishment:
- Height: weight: BMI:
- P/I/CY/CL/LN/PE
- VITALS- PR.BP,RR,TEMPERATURE

SYSTEMIC EXAMINATION

- ❖ RESPIRATORY SYSTEM
- Inspection :
- Palpation :
- Percussion :
- Auscultation:
- ❖ Cardiovascular system
- ❖ Central nervous system
- ❖ Abdominal system

18. Investigations:

- **X – Ray chest**
- **Spirometry /PEFR**
- **ECHO**
- **Sputum microscopic exam**
- **Absolute eosinophil count**

19. Asthma medications

20. CONTROLLED/PARTLY CONTROLLED/UNCONTROLLED

RESULTS

Chart : 1 - Sex distribution with level of asthma control

			Levels of Asthma Control			Total
			Uncontrolled	**Partly controlled**	**Controlled**	
Sex	**Male**	**Count**	**6**	**5**	**10**	**21**
		% within Sex	28.6%	23.8%	47.6%	100.0%
		% within Levels of Asthma Control	50.0%	41.7%	62.5%	52.5%
	Female	Count	6	7	6	19
		% within Sex	31.6%	36.8%	31.6%	100.0%
		%with in levels of Asthma control	50.0%	58.3%	37.5%	47.5%
Total	Count		12	12	16	40
	% within Sex		30.0%	30.0%	40.0%	100.0%
	% within Levels of Asthma Control		100.0%	100.0%	100.0%	100.0%

Totally 40 patients, male 21, female 19 uncontrolled asthma 6 for male 6 for female. Partly controlled male 5, female 7.

Impression: Absolute eosinophil count more than 450 cells per cumm seen in 2/3 of patients. (uncontrolled and partially controlled).

Sex wise difference: male-sum of uncontrolled and partly controlled(11) equal to controlled group(10).Female- sum of uncontrolled and partly controlled (68%)(13) 2 times greater than controlled group(6) (32%).

Chart 2 : Chi-Square Tests

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	1.236(a)	2	.539
Likelihood Ratio	1.246	2	.536
Linear-by-Linear Association	.511	1	.475
N of Valid Cases	40		

A 0 cells (.0%) have expected count less than 5. The minimum expected count is 5.70.

Chart 3 : This show male ,female sex with levels of asthma control

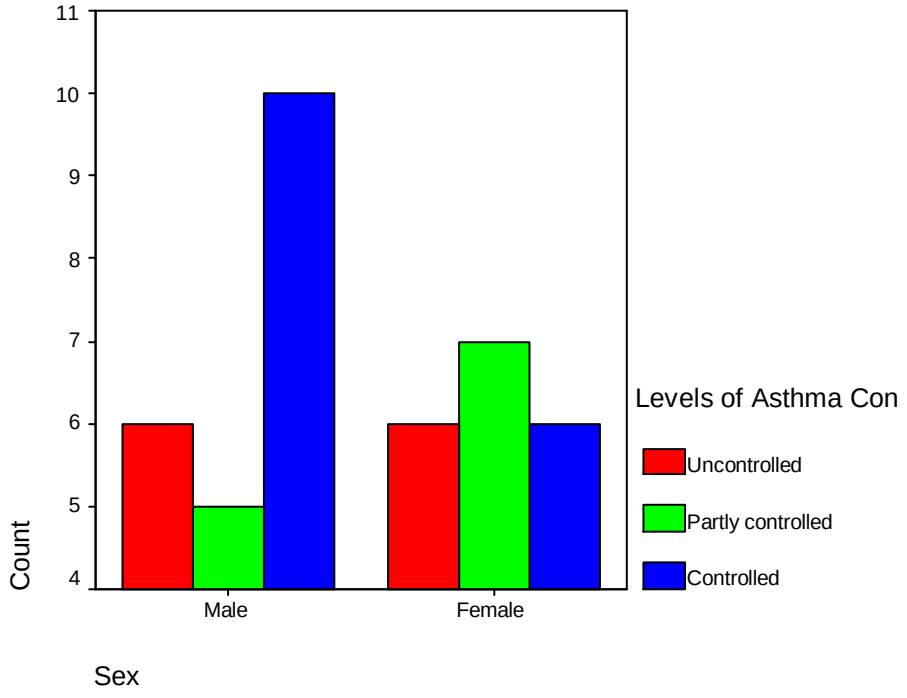


Chart - 4 : This show socio economic status with levels of asthma control Equal in both social economic status.

			Levels of Asthma Control			
			Uncontrolled	Partly controlled	Controlled	Total
Socio economic status	Low	Count	8	5	7	20
		% within Socio economic status	40.0%	25.0%	35.0%	100.0%
		% within Levels of Asthma Control	66.7%	41.7%	43.8%	50.0%
	High	Count	4	7	9	20
		% within Socio economic status	20.0%	35.0%	45.0%	100.0%
		%within levels of asthma control	33.3%	58.3%	56.3%	50.0%
Total		Count	12	12	16	40
		% within Socio economic status	30.0%	30.0%	40.0%	100.0%
		% within Levels of Asthma Control	100.0%	100.0%	100.0 %	100.0%

Chart 5 : Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	1.917(a)	2	.384
Likelihood Ratio	1.945	2	.378
Linear-by-Linear Association	1.272	1	.259
N of Valid Cases	40		

A 0 cells (.0%) have expected count less than 5. The minimum expected count is 6

Chart 6 : This show level of asthma control with low and high social economic status. High social economic and low social economic are equal disturbance with level of asthma control

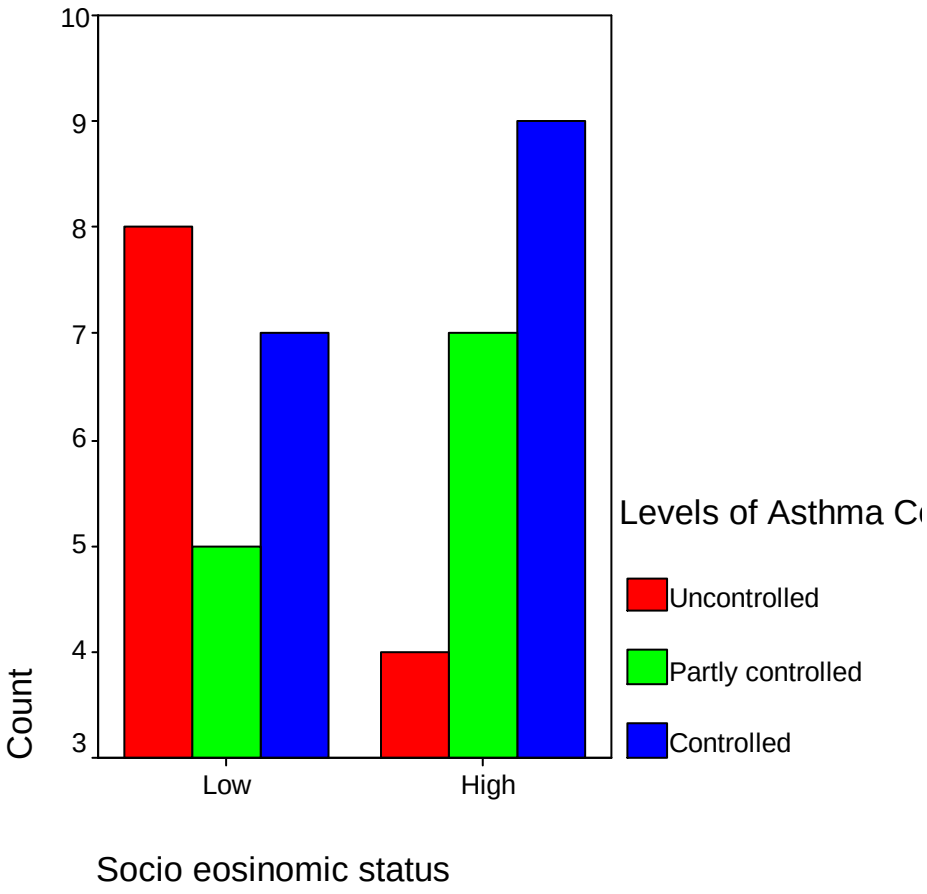


Chart 7 : This tables show relation between Precipitating factor with Levels of Asthma Control

			Levels of Asthma Control			Total
			Uncontrolled	Partly controlled	Controlled	
	Nil	**Count**	**0**	**5**	**8**	**13**
		% within Precipitating factor	.0%	38.5%	61.5%	100.0%
		% within Levels of Asthma Control	.0%	41.7%	50.0%	32.5%
	Pet animals	Count	3	3	4	10
		% within Precipitating factor	30.0%	30.0%	40.0%	100.0%
		% within Levels of Asthma Control	25.0%	25.0%	25.0%	25.0%
	Viral infections	Count	2	1	1	4
		% within Precipitating factor	50.0%	25.0%	25.0%	100.0%
		% within Levels of Asthma Control	16.7%	8.3%	6.3%	10.0%
	Mosquito repellents	Count	3	1	2	6
		% within Precipitating factor	50.0%	16.7%	33.3%	100.0%
		% within Levels of Asthma Control	25.0%	8.3%	12.5%	15.0%
		Count	3	2	1	6

	Pollens	% within Precipitating factor	50.0%	33.3%	16.7%	100.0%
		% within Levels of Asthma Control	25.0%	16.7%	6.3%	15.0%
	Smoke	Count	1	0	0	1
		% within Precipitating factor	100.0%	.0%	.0%	100.0%
		%with in levels of asthma control	8.3%	0%	0%	2.5%
Total		Count	12	12	16	40
		% within Precipitating factor	30.0%	30.0%	40.0%	100.0%
		% within Levels of Asthma Control	100.0%	100.0%	100.0%	100.0%

This chart show precipitating factor with asthma control. Precipitating factors are pet animals, mosquito repellants, viral infections, pollens, smoke.

1) pet animals - 10(3*3*4)(25%)

2) mosquito repellents - 6(3*1*2)(15%)

3) viral infections - 4(2*1*1)(10%)

4) pollens - 6(3*2*1)(15%)

5) smoke - 1(1*0*0)(2.5%)

Out of 40 patients,

1) 27 had precipitating factors (67.5%),

2) 13 had no precipitating factors (32.5%)

CHART 8 : Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	11.704(a)	10	.305
Likelihood Ratio	15.419	10	.118
Linear-by-Linear Association	8.182	1	.004
N of Valid Cases	40		

A 17 cells (94.4%) have expected count less than 5. The minimum expected count is .30.

CHART 10 : This show allergens 67.5%, nil precipitating factor 12.5%, Allergen mainly pet animals in city cultures compares with rurals (25%)

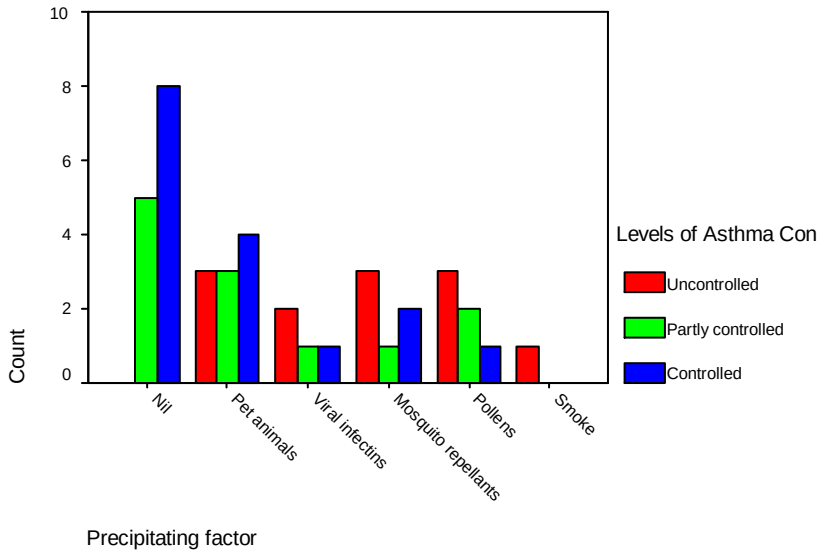


CHART 11 : This table show X ray chest with Levels of Asthma Control.

			Levels of Asthma Control			Total
			Uncontrolled	Partly controlled	Controlled	
Chest x ray	Normal	Count	4	12	16	32
		% within X ray chest	12.5%	37.5%	50.0%	100.0%
		% within Levels of Asthma Control	33.3%	100.0%	100.0%	80.0%
	Hyperinflation	Count	8	0	0	8
		% within X ray chest	100.0%	.0%	.0%	100.0%
		% with in levels of asthma control	66.7%	.0%	.0%	20.0%
Total		Count	12	12	16	40
		% within X ray chest	30.0%	30.0%	40.0%	100.0%
		% within Levels of Asthma Control	100.0%	100.0%	100.0%	100.0%

Chart 12 : Chi-Square Tests

	Value	Df	Asymp. Sig. (2-sided)
Pearson Chi-Square	23.333(a)	2	.000
Likelihood Ratio	24.756	2	.000
Linear-by-Linear Association	17.098	1	.000
N of Valid Cases	40		

A 3 cells (50.0%) have expected count less than 5. The minimum is 2.40.

CHART 13 : This show relation between x ray chest and level of asthma control.

Normal x ray chest - 32(80%)

Hyperinflation chest x ray- 8(20%) mainly uncontrolled patients

Normal chest x ray had 32 mainly controlled group-16(40%),partly controlled -12(30%)uncontrolled -4 (10%)

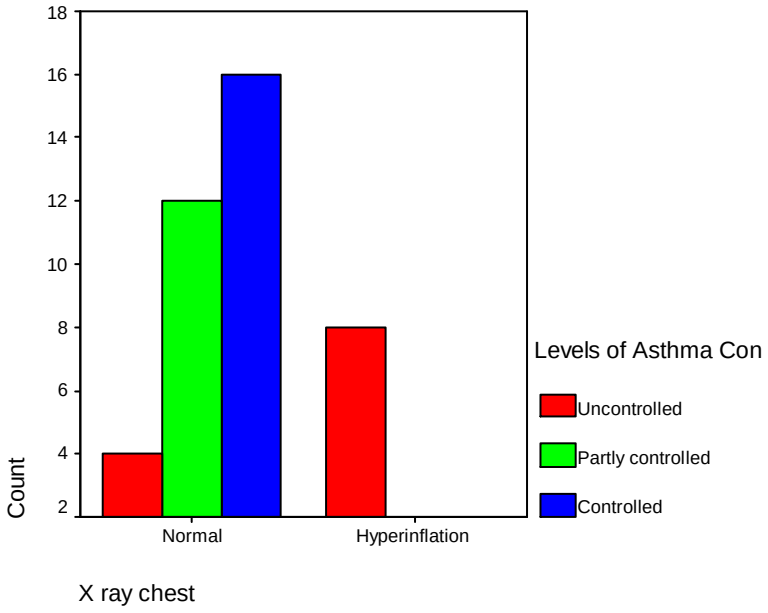


CHART14 : Sputum microscopic examination with Levels of Asthma Control

			Levels of Asthma Control			
			Uncontrolled	Partly controlled	Controlled	Total
Sputum examination	Normal	**Count**	**1**	**0**	**11**	**12**
		% within Sputum microscopic	8.3%	.0%	91.7%	100.0%
		% within Levels of Asthma Control	8.3%	.0%	68.8%	30.0%
	Eosnophils	Count	11	12	5	28
		% within Sputum microscopic	39.3%	42.9%	17.9%	100.0%
		% with in level of asthma control	91.7%	100.0%	31.3%	70.0%
Total		Count	12	12	16	40
		% within Sputum microscopic	30.0%	30.0%	40.0%	100.0%
		% within Levels of Asthma Control	100.0%	100.0%	100.0%	100.0%

Chart - 15 : Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	19.266(a)	2	.000
Likelihood Ratio	22.110	2	.000
Linear-by-Linear Association	13.027	1	.000
N of Valid Cases	40		

3 cells (50.0%) have expected count less than 5. The minimum expected count is 3.60.

CHART 16 : This show relation between sputum microscopic examinations with level of asthma control

1) normal sputum examination -12 (30%) mainly controlled group(27.5%)

2) Sputum eosinophilia - 28(70%)

   A) Controlled group - 5 (12.5%)

   B) Partly controlled group - 12 (30%)

   C) Uncontrolled group - 11 (27.5%)

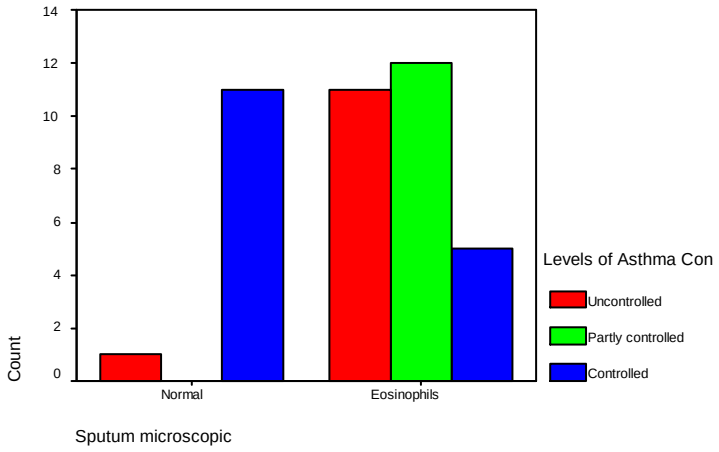


CHART 17 : This table show ECHO with Levels of Asthma Control

			Levels of Asthma Control			Total
			Uncontrolled	Partly controlled	Controlled	
ECHO	Normal	Count	6	12	16	34
		% within ECHO	17.6%	35.3%	47.1%	100.0%
		% within Levels of Asthma Control	50.0%	100.0%	100.0%	85.0%
	Mild pulmonary hypertension	Count	6	0	0	6
		% within ECHO	100.0%	.0%	.0%	100.0%
		% within levels of asthma control	50.0%	.0%	.0%	15.0%
Total		Count	12	12	16	40
		% within ECHO	30.0%	30.0%	40.0%	100.0%
		% within Levels of Asthma Control	100.0%	100.0%	100.0%	100.0%

This tables shows normal echo study in 34 patients (85%), mild pulmonary hypertension in 6 (15%).

Table 17 : Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	16.471(a)	2	.000
Likelihood Ratio	17.181	2	.000
Linear-by-Linear Association	12.069	1	.001
N of Valid Cases	40		

3 cells (50.0%) have expected count less than 5. The minimum expected count is 1.80.

CHART - 18

ECHO 1) Normal study - 34 (85%)

2) mild pulmonary HT - 6 (15%) mainly uncontrolled group

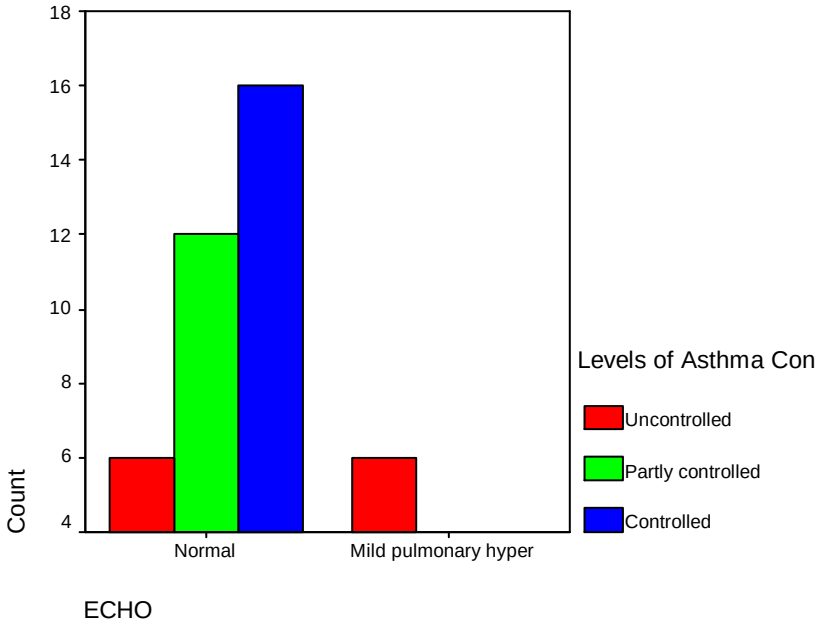


CHART 19 : Absolute eosinophil count with Levels of Asthma Control

			Levels of Asthma Control			Total
			Uncontrolled	Partly controlled	Controlled	
ABSOLUTE EOSINOPHIL COUNT	< 450	Count	0	1	16	17
		% within Absolute eosinophil count	.0%	5.9%	94.1%	100.0%
		% within Levels of Asthma Control	.0%	8.3%	100.0%	42.5%
	> 450	Count	12	11	0	23
		% within Absolute eosinophil count	52.2%	47.8%	.0%	100.0%
		%within levels of asthma control	100.0%	91.7%%	0%	57.5%
Total		Count	12	12	16	40
		% within Absolute eosinophil count	30.0%	30.0%	40.0%	100.0%
		% within Levels of Asthma Control	100.0%	100.0%	100.0%	100.0%

Table 20 : Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	36.249(a)	2	.000
Likelihood Ratio	47.664	2	.000
Linear-by-Linear Association	29.560	1	.000
N of Valid Cases	40		

0 cells (.0%) have expected count less than 5. The minimum expected count is 5.10.

CHART - 21

These show 40 patients had

1) Absolute eosinophil count less than 450 -17 (42.5%)

2) Absolute eosinophil count more than 450 -23 (57.5%)

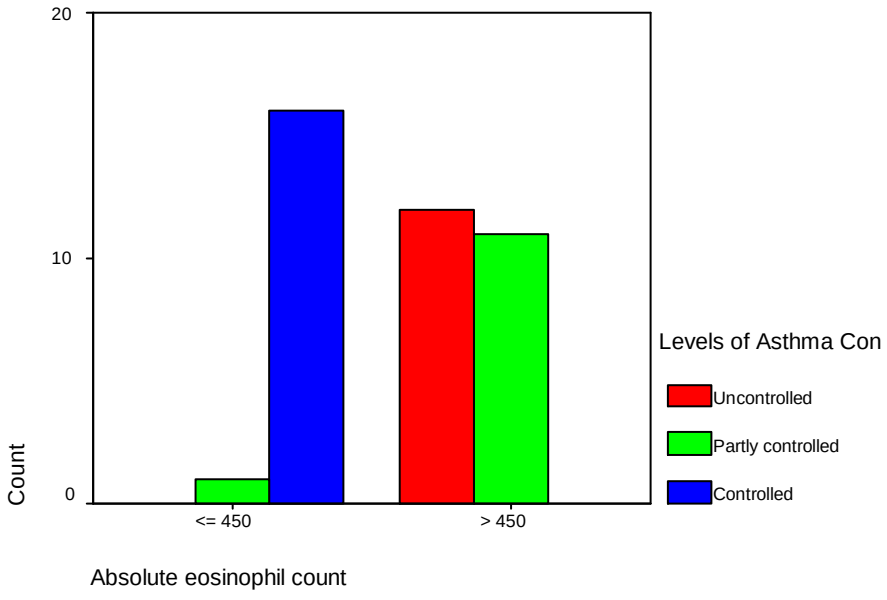


CHART 18

One way Descriptive

Age in years

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
Uncontrolled	12	34.33	8.574	2.475	28.89	39.78	23	52
Partly controlled	12	33.42	5.418	1.564	29.97	36.86	22	42
Controlled	16	37.44	10.302	2.575	31.95	42.93	20	54
Total	40	35.30	8.549	1.352	32.57	38.03	20	54

ANOVA

CHART 19

Age in years

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	126.879	2	63.440	.862	.431
Within Groups	2723.521	37	73.609		
Total	2850.400	39			

CHART - 20

One way -Descriptives

Weight in kg

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
Uncontrolled	12	63.50	12.072	3.485	55.83	71.17	46	86
Partly controlled	12	60.67	9.039	2.609	54.92	66.41	44	76
Controlled	16	67.06	10.529	2.632	61.45	72.67	45	80
Total	40	64.08	10.683	1.689	60.66	67.49	44	86

CHART 21

ANOVA

Weight in kg

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	286.171	2	143.085	1.271	.292
Within Groups	4164.604	37	112.557		
Total	4450.775	39			

CHART 22

Oneway

Descriptives : Height

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
uncontrolled	12	165.75	5.643	1.629	162.16	169.34	156	174
Partly controlled	12	160.50	7.728	2.231	155.59	165.41	146	170
Controlled	16	166.56	4.546	1.136	164.14	168.98	160	174
Total	40	164.50	6.397	1.011	162.45	166.55	146	174

CHART – 23

ANOVA

Height

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	278.813	2	139.406	3.916	.029
Within Groups	1317.188	37	35.600		
Total	1596.000	39			

CHART - 24 - Post Hoc Tests:Multiple Comparisons -Dependent Variable: Height -Tukey HSD

(I) Levels of Asthma Control	(J) Levels of Asthma Control	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Uncontrolled	Partly controlled	5.25	2.436	.093	-.70	11.20
	Controlled	-.81	2.279	.932	-6.38	4.75
Partly controlled	Uncontrolled	-5.25	2.436	.093	-11.20	.70
	controlled	-6.06(*)	2.279	.030	-11.63	-50
Controlled	Uncontrolled	.81	2.279	.932	-4.75	6.38
	Partly controlled	6.06(*)	2.279	.030	.50	11.63

- The mean difference is significant at the .05 level.

CHART 24 : Homogeneous Subsets Height-Tukey HSD

Levels of Asthma Control	N	Subset for alpha = .05	
		1	2
Partly controlled	12	160.50	
Uncontrolled	12	165.75	165.75
Controlled	16		166.56
Sig.		.076	.935

Means for groups in homogeneous subsets are displayed.

CHART 26 : Hospitalisation * Levels of Asthma Control

			Levels of Asthma Control			
			Uncontrolled	**Partly controlled**	**Controlled**	**Total**
HOSPITALIZATION	0	**Count**	**0**	**3**	**8**	**11**
		% within Hospitalisation	.0%	27.3%	72.7%	100.0%
		% within Levels of Asthma Control	.0%	25.0%	50.0%	27.5%
	1	Count	2	7	8	17
		% within Hospitalisation	11.8%	41.2%	47.1%	100.0%
		% within Levels of Asthma Control	16.7%	58.3%	50.0%	42.5%
	2	Count	3	2	0	5
		% within Hospitalisation	60.0%	40.0%	.0%	100.0%
		% within Levels of Asthma Control	25.0%	16.7%	.0%	12.5%
	3	Count	7	0	0	7
		% within Hospitalisation	100.0%	.0%	.0%	100.0%
		%within level of asthma control	58.3%	.0%	0%	17.5%
Total		Count	12	12	16	40
		% within Hospitalisation	30.0%	30.0%	40.0%	100.0%
		% within Levels of Asthma	100.0%	100.0%	100.0%	100.0%

CHART 27 : Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	29.077(a)	6	.000
Likelihood Ratio	34.448	6	.000
Linear-by-Linear Association	22.183	1	.000
N of Valid Cases	40		

CHART 28

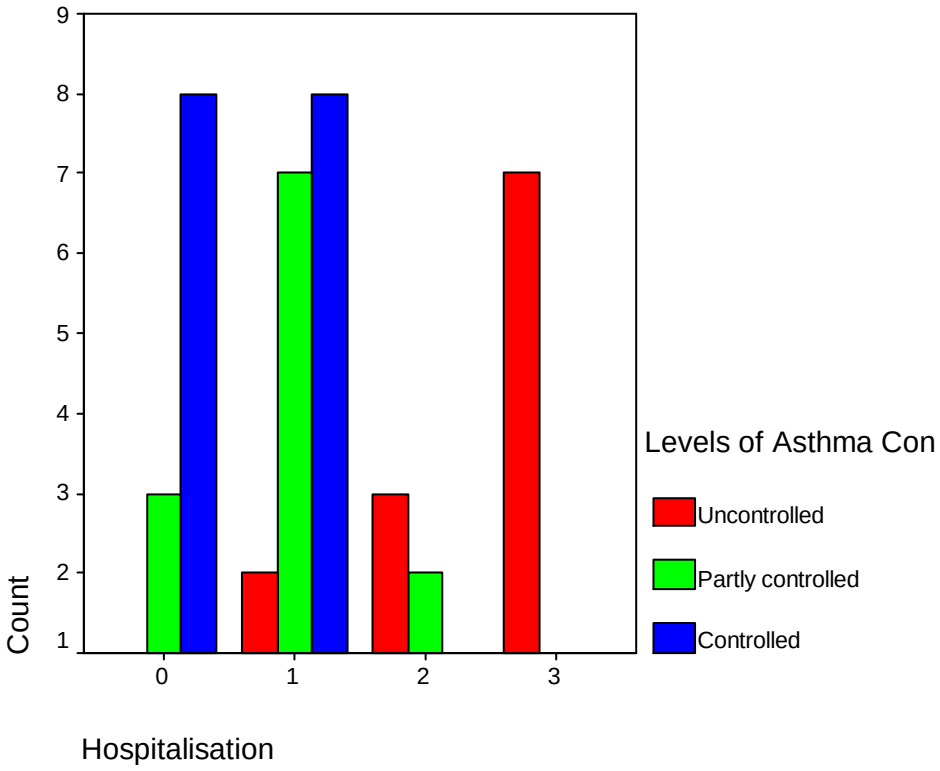


CHART - 29 : Emergency visit * Levels of Asthma Control

			Levels of Asthma Control			
			Uncontrolled	Partly controlled	Controlled	Total
EMERGENCY VISIT	0	Count	0	2	14	16
		% within Emergency visit	.0%	12.5%	87.5%	100.0%
		% within Levels of Asthma Control	.0%	16.7%	87.5%	40.0%
	1	Count	2	8	2	12
		% within Emergency visit	16.7%	66.7%	16.7%	100.0%
		% within Levels of Asthma Control	16.7%	66.7%	12.5%	30.0%
	2	Count	8	2	0	10
		% within Emergency visit	80.0%	20.0%	.0%	100.0%
		% within Levels of Asthma Control	66.7%	16.7%	.0%	25.0%
	3	Count	2	0	0	2
		% within Emergency visit	100.0%	.0%	.0%	100.0%
		% within levels of asthma control	16.7%	.0%	.0%	5.0%
Total		Count	12	12	16	40
		% within Emergency visit	30.0%	30.0%	40.0%	100.0%
		% within Levels of Asthma	100.0%	100.0%	100.0%	100.0%

CHART 30 : Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	40.514(a)	6	.000
Likelihood Ratio	44.226	6	.000
Linear-by-Linear Association	27.746	1	.000
N of Valid Cases	40		

11 cells (91.7%) have expected count less than 5. The minimum expected count is .60.

CHART 31

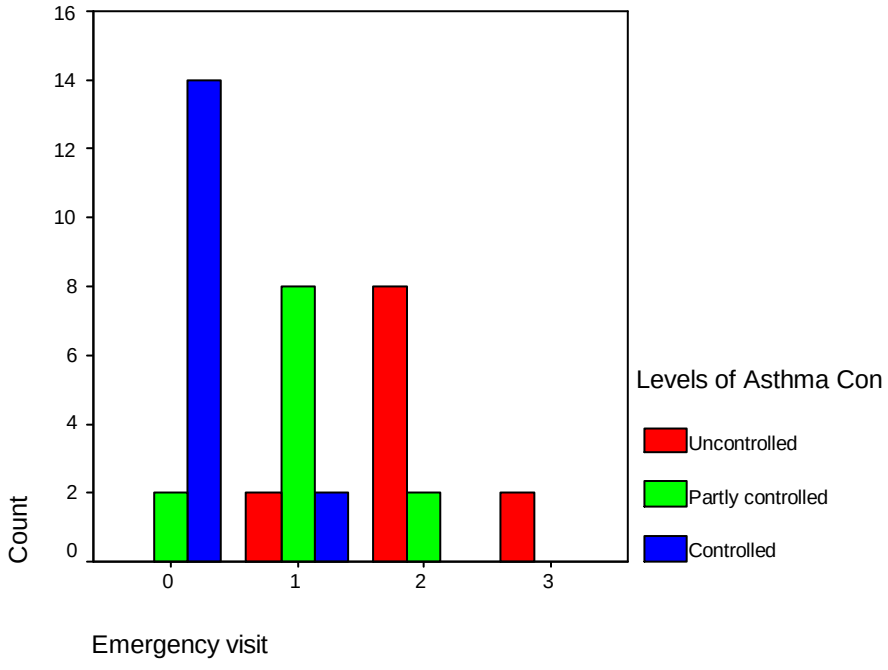


CHART 32 : Oneway : Descriptives

PEFR

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
uncontrolled	12	184.25	15.088	4.356	174.66	193.84	160	200
Partly controlled	12	260.33	29.137	8.411	241.82	278.85	200	290
Controlled	16	346.56	21.062	5.265	335.34	357.79	310	378
Total	40	272.00	71.877	11.365	249.01	294.99	160	378

CHART 33

ANOVA

PEFR

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	182987.146	2	91493.573	183.018	.000
Within Groups	18496.854	37	499.915		
Total	201484.000	39			

CHART - 34 Post Hoc Tests:Multiple Comparisons

Dependent Variable: PEFR

Tukey HSD

(I) Levels of Asthma Control	(J) Levels of Asthma Control	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Uncontrolled	Partly controlled	-76.08(*)	9.128	.000	-98.37	-53.80
	Controlled	-162.31(*)	8.538	.000	-183.16	-141.47
Partly controlled	Uncontrolled	76.08(*)	9.128	.000	53.80	98.37
	Controlled	-86.23(*)	8.538	.000	-107.08	-65.38
Controlled	Uncontrolled	162.31(*)	8.538	.000	141.47	183.16
	Partly controlled	86.23(*)	.8.538	.000	65.38	107.08

* The mean difference is significant at the .05 level.

CHART 35 : Homogeneous Subsets:PEFR

Tukey HSD

Levels of Asthma Control	N	Subset for alpha = .05		
		1	2	3
Uncontrolled	12	184.25		
Partly controlled	12		260.33	
Controlled	16			346.56
Sig.		1.000	1.000	1.000

Means for groups in homogeneous subsets are displayed.

a Uses Harmonic Mean Sample Size = 13.091.

b The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

CHART 36 : Oneway

Descriptives : Normal PEFR

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
Uncontrolled	12	338.58	26.092	7.532	322.01	355.16	298	375
Partly controlled	12	319.58	32.523	9.388	298.92	340.25	265	361
Controlled	16	343.25	21.306	5.326	331.90	354.60	311	375
Total	40	334.75	27.747	4.387	325.88	343.62	265	375

CHART 37 : ANOVA:Normal PEFR

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	4092.667	2	2046.333	2.920	.066
Within Groups	25932.833	37	700.887		
Total	30025.500	39			

CHART 38 :Post Hoc Tests:Multiple Comparisons

Dependent Variable: Normal PEFR :Tukey HSD

(I) Levels of Asthma Control	(J) Levels of Asthma Control	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Uncontrolled	Partly controlled	19.00	10.808	.198	-7.39	45.39
	Controlled	-4.67	10.110	.890	-29.35	20.02
Partly controlled	Uncontrolled	-19.00	10.808	.198	-45.39	7.39
	controlled	-23.67	10.110	.062	-48.35	1.02
Controlled	Uncontrolled	4.67	10.110	.890	-20.02	29.35
	Partly controlled	23.67	10.110	.062	-1.02	48.35

CHART 39

Homogeneous Subsets : Normal PEFR : Tukey HSD

Levels of Asthma Control	N	Subset for alpha = .05
		1
Partly controlled	12	319.58
Uncontrolled	12	338.58
Controlled	16	343.25
Sig.		.070

Means for groups in homogeneous subsets are displayed.

a Uses Harmonic Mean Sample Size = 13.091.

b The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

CHART 39 : Oneway : Descriptives : PEFR Percentage

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
Uncontrolled	12	54.5487	4.31305	1.24507	51.8083	57.2891	47.48	64.31
Partly controlled	12	81.4276	4.03905	1.16597	78.8613	83.9939	75.47	87.58
Controlled	16	100.9829	1.59778	.39945	100.1315	101.8343	98.16	105.92
Total	40	81.1860	19.74730	3.12232	74.8706	87.5015	47.48	105.92

CHART40

ANOVA : PEFR Percentage

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	14785.908	2	7392.954	647.624	.000
Within Groups	422.374	37	11.416		
Total	15208.282	39			

CHART 41 : Post Hoc Tests:Multiple Comparisons

Dependent Variable: PEFR Percentage :Tukey HSD

(I) Levels of Asthma Control	(J) Levels of Asthma Control	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Uncontrolled	Partly controlled	-26.8789(*)	1.37934	.000	-30.2465	-23.5112
	Controlled	-46.4342(*)	1.29026	.000	-49.5843	-43.2840
Partly controlled	Uncontrolled	26.8789(*)	1.37934	.000	23.5112	30.2465
	Controlled	-19.5553(*)	1.29026	.000	-22.7054	-16.4051
Controlled	Uncontrolled	46.4342(*)	1.29026	.000	43.2840	49.5843
	Partly controlled	19.5553(*)	1.29026	.000	16.4051	22.7054

* The mean difference is significant at the .05 level.

CHART 42 : Homogeneous Subsets: PEFR Percentage Tukey HSD

Levels of Asthma Control	N	Subset for alpha = .05		
		1	2	3
Uncontrolled	12	54.5487		
Partly controlled	12		81.4276	
Controlled	16			100.9829
Sig.		1.000	1.000	1.000

Means for groups in homogeneous subsets are displayed.

A Uses Harmonic Mean Sample Size = 13.091.

B The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

CHART 43 : Oneway:Descriptives

Absolute eosinophil count

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
uncontrolled	12	1070.00	130.384	37.639	987.16	1152.84	900	1260
Partly controlled	12	489.17	48.092	13.883	458.61	519.72	380	560
Controlled	16	218.25	54.243	13.561	189.35	247.15	120	300
Total	40	555.05	368.841	58.319	437.09	673.01	120	1260

CHART 44 : ANOVA

Absolute eosinophil count

	Sum of Squares	df	Mean Square	F	Sig.
Between Groups	5049117.233	2	2524558.617	364.058	.000
Within Groups	256576.667	37	6934.505		
Total	5305693.900	39			

CHART 45 : Post Hoc Tests:Multiple Comparisons :Dependent Variable: Absolute eosinophil count ;Tukey HSD

(I) Levels of Asthma Control	(J) Levels of Asthma Control	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
					Lower Bound	Upper Bound
Uncontrolled	Partly controlled	580.83(*)	33.996	.000	497.83	663.83
	Controlled	851.75(*)	31.801	.000	774.11	929.39
Partly controlled	Uncontrolled	-580.83(*)	33.996	.000	-663.83	-497.83
	Controlled	270.92(*)	31.801	.000	193.28	348.56
Controlled	Uncontrolled	-851.75(*)	31.801	.000	-929.39	-774.11
	Partly controlled	-270.92(*)	31.801	.000	-348.56	-193.28

* The mean difference is significant at the .05 level.

CHART 46 : HomogeneousSubsets:Absolute eosinophil count:Tukey HSD

Levels of Asthma Control	N	Subset for alpha = .05		
		1	2	3
Controlled	16	218.25		
Partly controlled	12		489.17	
Uncontrolled	12			1070.00
Sig.		1.000	1.000	1.000

A Uses Harmonic Mean Sample Size = 13.091.b The group sizes are unequal. The harmonic mean of the group sizes is used. Type I error levels are not guaranteed.

DISCUSSION

Bronchial Asthma is one of the most common disorders of inflammatory reactions of the respiratory tract, characterized by breathlessness, wheezing, minimal cough and expectoration. It is true that allopathic treatment can arrest the disease, but cannot cure completely.

This study was conducted on the patient who attended the Asthma op in Govt Royapettah Hospital, Kilpauk Medical College, Chennai. The patients of 18-75 years were selected for the study. Both the sexes were included and who belong to different socio-economic groups was taken as per inclusion criteria. A total of 40 cases were selected minimum of study was 6months. The statistical analysis made here is based on the data obtained from 40 cases.

In our study the total number of patients, recruited were 40. There was almost equal distribution of cases in the three groups with 12(30%) in uncontrolled, 16(40%) in controlled, and 12(30%) in partly controlled group as per GINA guidelines.

Sex distribution was almost equal among the subjects with 51% males and 49% females. Fifty percent of boys were found to be in the controlled group while the majority of girls (48.6%) were in the uncontrolled group. Totally 40 patient, male 21, female 19, uncontrolled asthma 6 for male 6 for female. Partly controlled male 5 ,female7.

Impression : Absolute eosinophil count more than 450 cells per cumm seen in 2/3 of patients.(uncontrolled and partially controlled).

Sex wise difference : male - sum of uncontrolled and partly controlled(11) equal to controlled group(10).Female- sum of uncontrolled and partly controlled (13) (68.4%)2 times greater than controlled group(6)(31.6%)

Among the overall sum of uncontrolled and partly controlled group, females constituted 68.4%, even though the initial sample sizes in both sexes were comparable. Males had sum of uncontrolled and partly controlled constituted 50%. The increased absolute eosinophil count seen in girls noted in our study needs to be validated by a larger study.

High social economic and low social economic are equal disturbance with level of asthma control.

An attempt was made in our study to find the correlation of height, weight and BMI with absolute eosinophil count.

Our study also find out relation between precipitating factor and asthma control. Asthma precipitating factors are pet animals, mosquitorepellants, viralinfections, pollens, smoke.

1) pet animals - 10(3*3*4)(25%)

2) mosquito repellants - 6(3*1*2)(15%)

3) viral infections - 4(2*1*1)(10%)

4) pollens - 6(3*2*1)(15%)

5) smoke - 1(1*0*0)(2.5%)

Out of 40 patients,

1) 27 had precipitating factors(67.5%),

2) 13 had no precipitating factors(32.5%)

An attempt was made in our study to find the correlation of hospitalization, emergency visit, x ray chest, ECHO with absolute eosinophil count.

Fifty seven and half percent (57.5%%) of the asthmatic had increased eosinophil count(more than 450 cell per cumm) and forty two and half percent (42.5%) had normal absolute eosinophil count and p value 0.000.. Among the 40 asthmatic patients 27(67.5%) had precipitating factors. So may be allergic asthma patient had increased absolute eosinophil count compares with non precipitating(non atopic) factors.

Our study shows that serum absolute eosinophil count have a significant impact on asthma control, higher levels correlating with poor

control so increased dose or frequent steroid inhalation and dose adjustment, lower absolute eosinophil count well control of asthma.

Lung function as measured by PEFR in our study, showed a positive correlation with absolute eosinophil count. Patients with high absolute eosinophil count(more than 450) had a much lower PEFR %, thus indicating an impaired lung function. High absolute eosinophil count is generally associated with inflammatory and infectious diseases thus impairing pulmonary function and contributing to poor control.

Respiratory tract infections are the commonest trigger for asthma exacerbation. High absolute eosinophil count is frequently associated with increased incidence of respiratory tract infections and inflammation. Thus it is worthwhile to assess the absolute eosinophil count status in all adults with asthma especially so in the uncontrolled and partly controlled group. Supplementation of systemic or inhaler steroids decreases the incidence of respiratory inflammation, thus diminishing the asthma exacerbations. It is difficult to ascertain that high absolute eosinophil count alone is responsible for poor asthma control from observational study like ours. Interventional studies with larger sample size would be required to prove the cause-effect relationship.

Persistent inflammation is the prime contributory factor for the symptomatology in uncontrolled asthma. Measurement of absolute eosinophil count can be useful in assessing the severity of such

inflammation. So the evaluation of absolute eosinophil count is a useful baseline investigation and its serial measurements can be taken as a marker for adjusting the dose of inhaled steroids.

Thus we infer from our study that there is a positive correlation between absolute eosinophil count and asthma control.

STUDY LIMITATION

1. It is a hospital based study with a small catchment area. There has to be multi-centric randomized control study for validation.
2. Even though there is a strong correlation between the degree of asthma control and absolute eosinophil count levels, the design of the study does not allow conclusions about the actual cause- effect relationship.
3. The adults included in this study are from a large referral centre and are thus not the actual representation of general population.
4. In our study we did not include the detail history regarding to find out the exact factors responsible for increased absolute eosinophil count
5. Since it was not an interventional study, we did not reassess the control in our patients after inhalation steroids and systemic steroids.
6. Very small member of patient involved in our studySince this is a time bound study, therefore couldn't follow the cases for a longer period. Some good cases couldn't be considered in this study because of discontinued treatment in between the study period and no proper history.

CONCLUSION

There were a total number of forty cases taken up at random for the study. Conclusions were arrived after statistical analysis of patients with an bronchial asthma.

The following conclusions are drawn from the study.

1. The prevalence of Allergic bronchial asthma is more in females 19 (47.5%) than in males 21 (52.5%).

2. Bronchial asthma is equal on both social economic status.

3. Absolute eosinophil count in majority of the cases 23 (57.5%) had increase absolute eosinophil count more than 450 cells/mm3

4. Our study shows that serum absolute eosinophil count have a significant impact(p value 0.000) on asthma control, higher levels correlating with poor control ,so increased dose or frequent steroid inhalation and dose adjustment, lower absolute eosinophil count well control of asthma.

5. Our study show fifty seven and half percent (57.5%) of the asthmatic had increased eosinophil count and forty two and half percent (42.5%) had normal absolute eosinophil count.. Among the 40 asthmatic patients 27(67.5%) had precipitating factors.so

may be allergic asthma patient had increased absolute eosinophil count compares with non precipitating(non atopic) factors.

6. It is the assessment of susceptibility and sensitivity, which determines potency selection and its repetition.

7. The disease intensity of scores used for the assessment of effect of treatment showed significant improvement after the treatment.

8. A detailed case taking and evaluation is necessary for the management in these cases.

9. Creating awareness and promoting knowledge education, among the patients and their families is also vital for the proper implementation of the treatment.

10. Measurement of absolute eosinophil count can be useful in assessing the severity of such inflammation. So the evaluation of absolute eosinophil count is a useful baseline investigation and its serial measurements can be taken as a marker for adjusting the dose of inhaled steroids.

RECOMMENDATIONS

1. It is worthwhile to assess precipitating factor and eosinophil count in all asthmatic and provide supplementation oral and inhaler steroids, wherever necessary, in order to improve the control

2. Evaluation of absolute eosinophil count and its serial monitoring can be used as a reliable marker in assessing asthma control and adjusting the dosage of inhaled steroids.

3. Bigger sample with extended time of research would provide better results.

4. It will be always scientific if control (placebo) group would have been kept simultaneously to verify the effectiveness of treatment.

5. Peak flow meters, spirometry, and Immunoglobin assay, and skin prick test studies if done simultaneously during the course of study would provide valuable data regarding the changes taking place in the drugs.

6. Universal standardized scale should be used, so that evaluation of outcome of the study would become precise.

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Annexure I

Proforma for asthma control

Name :

Age and sex:

Height: weight:

Serial no: PEFR:

Classification

Pre requisite; (asthma patients age 18 -75 yrs)

CHARACTERISTICS	CONTROLLED (All of the following)	PARTLY CONTROLLED (Any measure present)	UNCONTROLLED
Daytime symptoms	None (twice or less/week)	More than twice/week	Three or more features of partly controlled asthma
Limitation of activities	None	Any	
Nocturnal symtoms /awakenings	None	Any	
Need for reliever/rescue treatment	None (twice or less/week)	More than twice/week	
Lung function (PEF or FEV1)	Normal	<80%predicted or personal best	

Type of control:

Absolute eosinophil count:

S.No	Name	Age	Sex	Weight in kg	height	socio eosinomic status	hospitalisation	emergency visit	precipitating factor	x ray chest	sputum microscopic	ECHO	PEFR	Normal PEFR	PEFR Percent	Levels of Asthma Control	absolute eosinophil count
1	gokulnath	22	m	45	165cm	high	0	0	pet animals	normal	normal	normal	350	344	101.744	controlled	250
2	chithra	32	f	44	146cm	high	0	0	pet animals	normal	eosinophils	normal	200	265	75.4717	partly controlle	550
3	prashanth	24	m	54	160cm	low	0	0	pollens	normal	normal	normal	320	326	98.1595	controlled	275
4	saraswathi	42	f	60	147cm	low	0	1	squito repella	normal	eosinophils	normal	203	268	75.7463	partly controlle	475
5	vanitha	36	f	55	161cm	low	3	2	pollens	yperinflation	hils,charcot-leyden	monary hype	166	314	52.8662	uncontrolled	1000
6	vignesh	27	m	56	166cm	high	1	1	pet animals	normal	normal	normal	350	347	100.865	controlled	300
7	rajendren	54	m	80	173cm	low	0	0	nil	normal	normal	normal	372	372	100	controlled	230
8	kavitha	44	f	74	160cm	low	0	0	nil	normal	normal	normal	310	311	99.6785	controlled	120
9	bharath	30	m	78	174cm	high	1	0	pet animals	normal	normal	normal	378	375	100.8	controlled	235
10	malarkodi	29	f	46	163cm	low	3	3	pollens	normal	hils,charcot-leyden	normal	170	321	52.9595	uncontrolled	1100
11	harini	22	f	53	156cm	high	1	1	pet animals	normal	osinophil	normal	256	298	85.906	partly controlle	520
12	yuvaraj	34	m	60	165cm	high	1	0	viral infectins	normal	normal	normal	350	344	101.744	controlled	150
13	balaji	35	m	67	168cm	low	1	1	viral infectins	normal	eosinophil	normal	275	354	77.6836	partly controlle	470
14	ramesh	47	m	78	172cm	low	0	0	nil	normal	normal	normal	370	368	100.543	controlled	200
15	saranya	45	f	56	163cm	low	0	0	nil	normal	normal	normal	340	321	105.919	controlled	140
16	vijayabharathi	42	f	65	165cm	high	1	0	nil	normal	eosinophil	normal	330	328	100.61	controlled	180
17	prakash	40	m	86	174cm	low	3	2	squito repella	yperinflation	-leyden crystals,cu	monary hype	200	375	53.3333	uncontrolled	900
18	asha	30	f	56	160cm	low	1	0	nil	normal	eosinophil	normal	265	311	85.209	partly controlle	480
19	evangelene	23	f	50	168cm	high	3	2	pet animals	normal	t-leyden crystal,cu	normal	160	337	47.4777	uncontrolled	1020
20	banumathi	33	f	52	160cm	high	1	1	nil	normal	eosinophils	normal	260	311	83.6013	partly controlle	475
21	sangeetha	31	f	53	163cm	low	2	1	viral infectins	yperinflation	phils,curschmann's	monary hype	180	321	56.0748	uncontrolled	1200
22	saravanan	46	m	70	173cm	low	1	2	squito repella	yperinflation	hils,charcot-leyden	monary hype	200	372	53.7634	uncontrolled	980
23	begam	45	f	65	161cm	low	0	0	nil	normal	eosinophil	normal	320	314	101.911	controlled	267
24	karthick	34	m	78	170cm	high	1	0	nil	normal	normal	normal	365	361	101.108	controlled	245
25	saran	20	m	76	172cm	high	0	0	pet animals	normal	normal	normal	370	368	100.543	controlled	280
26	peterjohn	45	m	73	168cm	high	1	0	nil	normal	eosinophil	normal	355	354	100.282	controlled	200
27	kesavan	35	m	78	173cm	high	2	2	squito repella	yperinflation	hils,charcot-leyden	normal	200	372	53.7634	uncontrolled	1250
28	laxmi	40	f	65	166cm	high	1	0	squito repella	normal	hils,charcot-leyden	normal	335	331	101.208	controlled	250
29	gunasundari	34	f	62	160cm	low	3	2	viral infectins	yperinflation	hils,charcot-leyden	monary hype	200	311	64.3087	uncontrolled	1100
30	karthikeyan	36	m	67	165cm	low	0	1	nil	normal	eosinophils	normal	275	344	79.9419	partly controlle	560
31	balamurugan	29	m	70	168cm	low	1	1	pet animals	normal	eosinophils	normal	180	354	50.8475	uncontrolled	900
32	yuvaraj	34	m	72	165cm	high	2	2	smoke	yperinflation	hils,charcot-leyden	normal	200	344	58.1395	uncontrolled	950
33	subalaxmi	46	f	70	165cm	low	1	1	squito repella	normal	eosinophils	normal	330	328	100.61	controlled	170
34	jeevitha	29	f	56	161cm	high	2	1	pollens	normal	eosinophils	normal	275	314	87.5796	partly controlle	470
35	padma	33	f	60	160cm	high	1	1	pollens	normal	eosinophils	normal	265	311	85.209	partly controlle	460
36	nagaraj	52	m	64	165cm	low	3	2	pollens	yperinflation	hils,charcot-leyden	monary hype	180	344	52.3256	uncontrolled	1260
37	sakthivel	41	m	76	170cm	high	1	2	nil	normal	eosinophils	normal	290	361	80.3324	partly controlle	530
38	swathy	23	f	56	156cm	high	3	3	pet animals	normal	hils,charcot-leyden	normal	175	298	58.7248	uncontrolled	1180
39	tamilselvan	37	m	70	168cm	low	1	2	nil	normal	eosinophils	normal	285	354	80.5085	partly controlle	500
40	praveen	31	m	67	165cm	high	2	1	pet animals	normal	hils,charcot-leyden	normal	275	344	79.9419	partly controlle	380