

A Review Article on Asthma

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Abstract—The lungs, alveoli, trachea and other respiratory tract components are the most frequently affected areas by the chronic illness known as asthma. It was discovered that heritable factors other than those connected to the DNA sequence and a changing lifestyle are to blame for the current rise in asthma incidence ecological. Asthma is caused by several inflammatory cells and it is clear that no single The pathophysiology of allergy illnesses can be explained by inflammatory cells, however certain cells are more prevalent in symptoms of asthma, such as mast cells, macrophage's, etc.

Keywords—macrophases, mast cells and asthma.

I. INTRODUCTION

Breathing becomes difficult when you have asthma, a chronic illness. Asthma can be managed, but it cannot be cured. The signature of asthma is the tracheobronchial smooth muscle's hyperresponsiveness to many stimuli, Leading to air tube narrowing, which is frequently accompanied by mucus plugging, mucosal edema and increased secretion. Dyspnea, wheezing, coughing and possibly activity limitation are among the symptoms [1]. Asthma remains the most common chronic respiratory disease in Canada, affecting approximately 10% of the population [2]. Asthma is a heterogeneous disease that affects many individuals. There are approximately 235 million people worldwide who have asthma and in 2015 there were approximately 383,000 asthma-related deaths [3].

There are two types of asthma:

- Extrinsic asthma
- Intrinsic Asthma

Numerous mediators are produced by mast cells and inflammatory cells that are drawn in as part of the initial reaction.

- The release of granule-stored mediators, such as $TNF\alpha$, histamine and protease enzymes.
- The release of phospholipids from the cell membrane is followed by the synthesis of mediators such as LTs, PAFs and PGs [4].

The combination of genetic and environmental variables that contribute to asthma. Allergens and air pollution are included in the exposure to environmental factors. Medications like beta blockers and aspirin may act as potential triggers. To therapy response of spirometer and over times, symptoms of based on the pattern diagnosis is used. Peak expiratory rate flow and volume in one second forced expiratory, the symptoms of frequency, according classified in asthma [5].

Objectives:

- Describe the mechanism of asthma.
- Describe the various asthma treatments' modes of action.
- Identify interprofessional team strategies for improving care coordination and educating the patients regarding the proper use of asthma medication [6].

Classification of Drugs:

1. Bronchodilators

(a). β 2 Sympathomimetics:

- Short acting: -e.g. Salbutamol, Terbutaline,
- Long acting ; - e.g. Salmeterol, formoterol, Bambuterol.

(b) Methylxanthine:-

e.g. Doxophylline, Aminophylline, Theophylline.

(c) Anticholinergics:

e.g. ipratropium bromide, tiotropium bromide.

2. Leukotriene receptor antagonists

e.g. Zileuton, zafirukast, montelukast.

3. Mast Cell Stabilizers

e.g. cromolyn sodium, ketotifen.

4. Corticosteroids

(a) systemic:-

e.g. Beclomethasone, Methyl prednisolone.

(b) Inhalation: -

e.g. flunisolide, Beclomethasonedipropionate, budesonide.

5. Anti-Ige Antibody

e. g. omalizumab. [7].

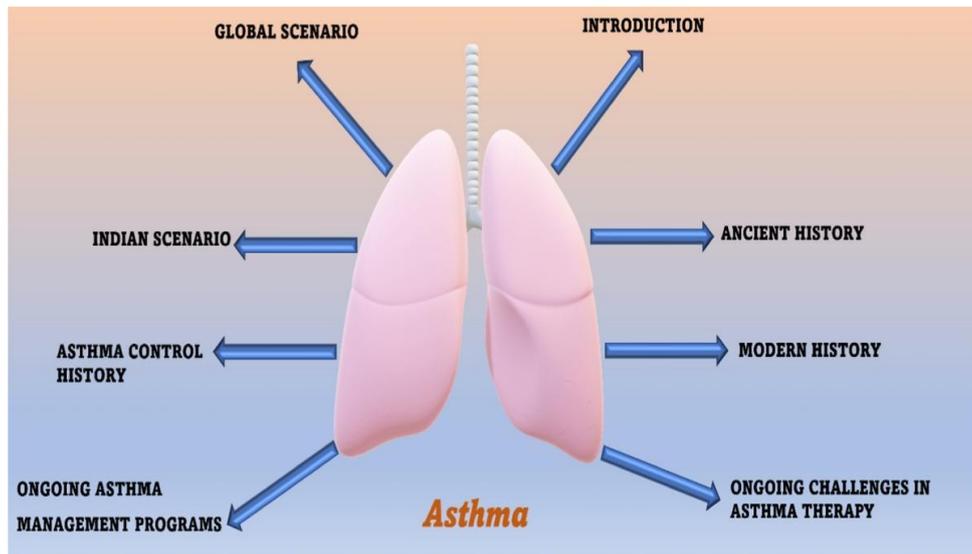


Figure 1: Asthma [8]

Causes of asthma:

- Genetic and environmental factors interact to cause asthma and these factors affect how severe asthma is and how well it responds to therapy.
- It was shown that heritable factors other than those associated with asthma are responsible for the recent rise in asthma incidence.
- Asthma onset beyond the age of 12 is more likely to be caused by inherited factors, whereas blockage before the age of 12 is related to changes in the environment.[9]
- There are numerous elements that interact to cause asthma rather than a single cause. One individual's a person's asthmatic factors may differ greatly from another's.
- Airway hyperresponsiveness or an overreaction of the bronchi to different physiological and environmental stimuli or triggers, is what causes asthma attacks.
- Asthma settings have been linked to a number of environmental elements, including air pollution, allergies and chemicals in.[10]

Symptoms and signs:

- Dehydration
- Symptomatic dyspnea
- Tiredness
- A tachycardia
- Illness of the respiratory systems
- Hypoxemia
- Hypercapnoea
- Having trouble walking

- Having trouble speaking
- Extremely rapid or sluggish respiration
- Pale
- The skin around the neck or ribs is awful.
- Blue or gray around the nail beds or lips.
- Extended expiratory period[11].

Common early warning signs can include:

Sneezing and breathing
 Changes in inhalation, clearing the lungs,
 Itching the throat and feeling fatigued
 Migraine, chest discomfort, sleep issues,
 Chin Itches lower peak flow readings
 Circles of darkness under the eyes: Breathless[12].

Pathophysiology:

Pathophysiology asthma is linked to T helper cell type-2 (Th2) immune responses, which are common in other atopic conditions. A number of allergic (such as dust mites, cockroach residue, furred animals, molds and pollens) and non-allergic (such as infections, tobacco smoke, cold air and exercise) triggers result in a series of immune-mediated events that cause chronic airway inflammation. Increased The cell levels in the airways release particular cytokines, such as interleukin (IL)-4, IL-5, IL-9 and IL-13, which encourage eosinophilic inflammation and mast cell production of immunoglobulin E (IgE)[2].

In asthma, limitation of airflow occurs often and is brought on by a number of airway alterations .These include:

Bronchoconstriction:

allergens that cause asthma episodes, such as smoking, pollen and dust. Breathing becomes challenging as a result of the airways narrowing and producing more mucus. Having asthma is the outcome of the bronchial airways' immunological reaction.

Airway Edema:

As the illness worsens and inflammation increases, more variables restrict airways even more. Edema, inflammation, excessive mucus secretion and the development of caused mucus blockages and structural alterations, such as airway hypertrophy and hyperplasia smooth muscle[13].

Hyperresponsiveness of the airways:

Several mechanisms of action contribute to airway excessive reactivity, such as fever, ineffective, and constructive alterations[14].

Prevention:

Patients who experience psychologically uncomfortable acute attacks may benefit from some social and familial readjusting; for youngsters, this may include talking to the parents. For extrinsic asthmatics, this method is suitable. Identification of an allergy may be facilitated by the history of the patient (wheezing in reaction to grass, pollen or animal touch), as determined by an intradermal skin prick. Injection of a particular allergen or by detecting particular IgE antibodies in the patient's blood, that is, the radio allegro sorbent test or rast test[15]. It is anticipated that genetic discoveries would result in a more accurate classification of complicated illnesses like asthma and novel medicines will result from genetic findings. Most polymorphisms so far detected do not seem to carry risk that would merit their use for the therapeutic classification of disease, but mixing of genetic polymorphism may be much more informative [16].

- Develop & follow an asthma care strategy.
- Plan measures to decrease the child's contact with triggers.
- Treat symptoms early.
- Be ready for any shifts in your symptoms.
- Recognize when medical assistance is required and seek it out immediately.
- It is not necessary for flare-ups to be a crisis [17].

Treatment of Asthma: During each appointment, the degree of asthma control should be assessed with and treatment tailored to reach control. The majority of asthma patients can achieve control through a combination of avoidance strategies and pharmacological treatments. The primary goal of asthma management is to achieve and sustain disease control to avert exacerbations (a sudden and/or progressive worsening of asthma symptoms that often necessitates immediate medical intervention and the use of oral steroids) and reduce the risk of morbidity and mortality[2]. These medications come in the form of puffers that are used when a person believes an asthma attack is imminent. A controller medication is one that is taken daily by the patient in the form of a pill or puffer to prevent asthma attacks [18].

Controllers of Asthma

- Controls of asthma as following:-
- To prevent problematic and chronic symptom, such as shortness of breath & coughing.
- To lessen your quick- alleviation need for drugs.
- The help of maintained in lung function good.
- Sleeping through the night and maintaining a regular level of activity.
- To avoid asthma episodes that would necessitate an ER visit or hospital stay [19].
- These medications lessen the formation of mucous and inflammation. They have no effect on bronchospasm.
- Either medication is a long-acting bronchodilator for immediate symptom alleviation [20].

Drugs used in Asthma

Drugs for asthma: There are two main groups of asthma drugs.

1. Long-term control drugs:

Examples

The most reliable therapy for long-term management is inhaled corticosteroids.

Theophylline and cromolyn are used as substitute controller medications.

Alternative controller medications include leukotriene modifiers.

Immunomodulators: Omalizumab alters the immunological response to allergies.

2. Beta agonists with short half-lives For instance

Albuterol

Levalbuterol [21].

Additional substance used:

The following medications are used as maintenance therapy to prevent acute attacks:

Salmeterol

Formoterol

Hydroxide of ephedrine

Theophylline

Glucocorticoids [22].

Chronic Obstructive Pulmonary Disease

- COPD is a slowly developing syndrome marked by a progressive decline in lung function
- COP diseases are progressive diseases with in alveolar destruction (emphysema) and bronchiolar fibrosis in variable proportions [2].
- Patients derive < 15% improvement in forced expiratory volume in 1 sec following inhalation of a β Agonist bronchodilator: airway obstruction is largely irreversible.
- A common chronic condition affecting the airways is asthma, which entails a complicated interplay of airflow obstruction, bronchial hyper-responsiveness, and underlying inflammation. This interaction can vary greatly, both among different patients and within the same patient over time [3].

Inflamator cell [23]

Mast cells:

An important component of the early allergic response, which usually begins minutes after being exposed to the right antigen, is the mast cell. Patients with allergic asthma experience immediate symptoms, such as coughing, wheezing and dyspnea, when exposed to a sufficient amount of antigen. After the encounter, these symptoms usually go away within 60 minutes, peaking within 10 to 15 minutes.

Macrophages:

In people with normal and stable asthma, macrophages make up over 90% of the cells recovered by bronchoalveolar lavage, making them the major resident cells in the lower airway.

Neutrophils:

Over the last 20 years, research involving asthmatic patients and bronchoscopy methods has primarily concentrated on how eosinophils contribute to asthma development; however, recent studies that have evaluated acute asthma exacerbations

T- Lymphocytes:

Insbesondere T-Lymphozyten sind für die Entstehung von Asthma maßgeblich verantwortlich. There has been special emphasis on a T cell subset, the 2 type, That secrete cytokines such as IL-4, IL-5, IL-9, and IL-13 [24].

Basophil:

The basophil roles of asthma are uncertain in the cell, and their detection through immunocytochemistry has been challenging. A small rise in basophils has been recorded in the airways of asthmatic patients using a basophil-specific marker, with their numbers increasing following allergen challenge.

Platelets:

Pathophysiology of allergic disease, since platelet activation may be observed and there is evidence for platelets in bronchial biopsies of asthmatic patient. After allergen challenge there is a significant fall in circulating platelets and release the chemokine [25].

The influence of genetic factors on asthma therapy:

It's unclear if these variations in reaction indicate failure of β -agonists of the arginine-16 gene type, who are sufficient on their own when their dosage is increased [26]. About one-third of asthma's genetic predisposition has been revealed to date. Existing research programs carried out in several countries are likely to identify the remaining important genetic effects within the next five years. The stage of genetic knowledge will need to be followed by a number of important studies [27].

Adverse Effects of Anti-asthmatic drugs:[28,29]

Diarrhea, nausea and plasma concentration

An irregular heartbeat

Convulsions

Tension

Fatigue

A rapid heartbeat

Hyperglycemia

High potassium levels

A low magnesium level

The tremors

Throwing up

Sleeplessness
Anxiety
Stress

II. CONCLUSION

The lungs are impacted by asthma, a respiratory illness. It has an effect on other body parts as well. There are two categories of asthma: extrinsic and intrinsic. Allergens and air pollution are among the environmental factors that contribute to asthma. A variety of medications are used for asthma. They are referred to as leukotriene receptor agonists and asthma medications (the medication, This substance). (Zileuton, Montelukast), corticosteroids such as beclomethasone, and mast cell stabilizers like ketotifen. Inhalation therapy consists of several widely used drugs, such as flunisolide, beclomethasone dipropionate, and budesonide. The signs of asthma can differ greatly from one person to another.

REFERENCE

- [1] Tripathi, K.D., Essentials of medical pharmacology, Jay pee publishers ptd ltd, New delhi. Volume 2008, sixth edition, Page no 216-217.
- [2] Kim.H,Mazza J.Asthma.Allergy asthma clinical immunology. 2018 Sep 12;14(2),50.
- [3] Jennifer Y. SO, Albert J,Shenoy K.Asthma: Diagnosis and treatment.European medical journal.2018,3[4]:111-121.<https://doi.org/10.33590/emj/10313763>.
- [4] Riedler. J, Nowak, D. and von Mutius, E. 2001, Exposure to farming in early life and development of asthma and allergy: a cross-sectional survey. Lancet, page no 299, 1229-1133.
- [5] Hu, F. Persky, V., Flay, B. and Richardson, J. volume 1997, An epidemiological study of asthma prevalence and related factors among young adults. J. Asthma, page no 34, 67- 76.
- [6] Sharma S, Muhammad F, Chakraborty R.Asthma medications.Statpearls,2023.
- [7] Salil K Bhattacharya, ParantapaSen, Arunabha Ray and Prasun K Das, Pharmacology,second edition, A division of reed elsevier india pvt, ltd. Page no 312-315.
- [8] Kapri A,Pant S,Gupta N,Paliwal S,Nain S.Asthma history, current s situation an overview of its control history, challenges, and ongoing management programs: An updated review.proc natl acad sci india sect B biol Sci.2022 .1-13.
- [9] Busse WW, Lemanske RF. Asthma. N Engl J Med, volume 2001, page no 350- 362.
- [10] Cookson WO, Moffatt M F. Genetics of asthma and allergic disease. Hum mol 2000; Page no 10-11.
- [11] Prescott &Dunn's, Pharmacology, 4th Edition, CBS publishers and distributors,pvt,Ltd Delhi page no 80-82.
- [12] Ying S, Humbert M, Meng Q, et al. Local expression of epsilon germline gene transcripts and RNA for the Epsilon heavy chain of IgE in the bronchial mucosa in atopic and nonatopic asthma. J Allergy ClinImmunol 2001; Page no 686-692.
- [13] Brightling CE, Bradding P, and Symon FA, Pavord ID. Mast- cell infiltration of airway smooth muscle in Asthma, N Engl J Med. Volume 2002, page no 1699- 1705.
- [14] Milgrom H, Fick RB Jr, Su JQ, et al, Anti -IgE therapy in asthma: rationale and therapeutic potential, Int Arch Allergy immunol , volume 2000, page no 196- 200.
- [15] Vincent S D, Toelle BG &Aroni RA, "Exasperation" of Asthma a qualitative study of patient language about Worsening asthma. Med J Aust 2006 'page no. 451- 455.
- [16] Bennett P.N. & Brown M.J, Clinical Pharmacology, 10th Edition, Churchill Livingstone Elsevier' page no 502-503.
- [17] Fahy JV, Kim KW & Liu J, Guideline for the diagnosis and management of asthma ,"NIH publication & Department of health and human services .National Asthma education and prevention program 1991, page no.816-822.
- [18] Chand S, Barar F.S.K, Essentials of Pharmacotherapeutics, 1st Edition, published by S. Chand & Company LTD. , Ram Nagar New Delhi , Page No. 112.
- [19] Dworski R, Barry KA, and Barnes CN, Sputum cysteinylleukotrienes increase after allergen inhalation in atopic Asthma, Am JR Care Med, volume 2000; page 55- 58.
- [20] McKenzie. R, Kelly. MM, and Leigh.JK, Induced sputum examination;diagnosis of pulmonary involvement in Fairy's disease, volume 2004, page no 20- 21.
- [21] Haahtela T, and Laitinen A, A comparative study of the effects of an inhaled corticosteroid, budesonide, and a Beta 2-agonist, terbutaline, on

- airway inflammation in newly diagnosed asthma; controlled trial, *J Allergy and Clin Immunol*, volume 1994, page no 42- 44.
- [22] Satoskar R.S, Bhandarkar S.D &Nirmala N. Rege, *Pharmacology &pharmacotherapeutics*, Twenty first Edition, Popular prakashan. Page no 358-361.
- [23] Bateman ED, Boushey HA & Pedersen SE, Goal investigators group. Can guideline defined Asthma control be Achieved, The gaining optimal asthma control study. *Am J RespirCrit care Med* 2004, page no. 836- 844.
- [24] Lambrecht BN, The dendritic cell in allergic airway disease, A new player to the Clin Experiment Allergy , Volume 2001, page no 206-218.
- [25] Saltos N Borgas T., Gibson PG, Airway mast cells and eosinophil's correlate with clinical severity and airway Hyper responsiveness in corticosteroid- treated asthma, *Jc Allergy Clin Immunology*, volume 2000, page no 751–755.
- [26] Lim S, Barnes PJ, and Jatakanon A, Changes in sputum eosinophils predict loss of asthma control, *Am J RespirCrit, Care Med*, volume 2000, page no 161- 165.
- [27] Barnes P J. Cytokine modulators as novel therapies for asthma. *Annu Rev PharmacolToxicol* 2002; page no 42,81–98.
- [28] Kharitonov. SA, Durham. SR, and Campbell. D, Increase in exhaled nitric oxide levels in patients with difficult Asthma, and correlation with symptoms and disease severity despite treatment with oral and inhaled corticosteroids, *Asthma and Allergy Group*, volume 2001, page no 101-104.
- [29] Richand A. Harvey, Pamela C. Champe, Luigi X. Cubeddu, *Pharmacology*, 4th Edition, Distribution in India. Page no 321-322.