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CASSIA SOPHERA IN TREATMENT OF BRONCHIAL ASTHMA

JV'n Dr. Yogita Kumari

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CASSIA SOPHERA IN TREATMENT OF BRONCHIAL ASTHMA

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PREFACE

Community Medicine is an essential knowledge that should be possessed by every medical practitioner. Community Medicine, as a branch of medicine and a major subject of BHMS course, is concerned with health of populations.

It aims to protect and promote the health and well-being of communities and populations through Primary Health Care approach. It deals with analysing and measuring the health needs of populations, their health status and then develops appropriate and technically and practically feasible strategies to prevent and control diseases and improve health of populations through Health Promotion, Health Education and Health Protection.

My attempt in writing this book is to focus attention of pupil and practitioners towards this lesser known drug called Cassia sophera in the cases of Bronchial Asthma, as it is a growing disease due to the environmental factors and pollution levels throughout the globe.

The aim of writing this book is too concise all essential information in an easily comprehensible arrangement that can easily be understood by students without missing any essential information.

JV'n Dr. Yogita Kumari

Author

ABOUT AUTHOR

Dr Yogita Kumari is a Homoeopathic academician and consultant having B.H.M.S. M.D. from Jaipur. Currently, She is working as Assistant Professor in the Department of Community Medicine at Faculty of Homoeopathic Science under Jayoti Vidyapeeth Women's University, Jaipur. She has a vast clinical experience of more than 3 years. She has written and published 12 research papers in various National and International journals.

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First and foremost, I would like to thank my God who have always acted as a continuous source of energy through blessings and supported me and my family throughout our lives and without their encouragement this dissertation would not have been nurtured and matured. No endeavour can start without the blessing of almighty God.

I would like to thank the publisher, who gave me the opportunity to publish this book.

I would like to thank my parents and family for their immense support, without which this work was not possible. I would also like to thank my colleagues who encouraged and supported me in writing this book.

I wish that this book will help students to understand the topic and will also guide them in preparation for the PG examination.

CONTENTS

INTRODUCTION:

Asthma is characterized by chronic airway inflammation and increased airway hyper responsiveness leading to symptoms of wheeze, cough, chest tightness and dyspnea. It is characterized functionally by the presence of airflow obstruction which is variable over short periods of time or is reversible with treatment. It is the major cause of impaired quality of life with impact on work and recreational as well as physical activities and emotions.

Hippocrates (400 BC) was the first to use the term “Asthma” (Greek for “wind” or “to blow”) for respiratory distress. He is considered to be the physician who identified the relationship between the environment and respiratory disease correlating climate and location with illness. Roman doctors described asthma as gasping and the inability to breathe without making noise. Bronchial asthma is one of the major health problems for the developed and developing countries.

Worldwide, it is estimated that 300 million people are affected with bronchial asthma. Asthma prevalence is increasing despite the recent advances in its management including understanding the inflammatory nature of the disease, use of steroids with add on long acting bronchodilators, use of devices to deliver the medications more appropriately. The economic burden associated with this condition is severe.

Homoeopathy is the second most widely used CAM in health care systems according to WHO. Studies have shown that homoeopathic treatment for respiratory diseases is associated with the significant reduction in the use and costs of conventional drugs. Central council for Homoeopathy has conducted several studies to evaluate the therapeutic usefulness of homoeopathy. Homoeopathy, being an individualistic therapy which follows an individualistic approach will boost the immunity and thereby reduce the severity and recurrence of attacks. Since there are many clinical researches on management of bronchial asthma using constitutional as well as specific remedies, one remedy being Cassia sophera ; it acts through the organic nervous system and especially affects the mucous membranes of the respiratory organs.

EPIDEMIOLOGY:

The Global Initiative for Asthma (GINA), 2009 has defined asthma based on its clinical, physiological and pathological characteristics. The predominant clinical feature of asthma is cough, shortness of breath and wheeze, particularly at night and early morning.

The main physiological feature of Asthma is episodic, reversible, airways obstruction (airflow limitation) or expiratory airflow limitation. The predominant pathological feature is airways inflammation, sometimes associated with airways structural changes [1].

Globally, asthma is ranked 16th among the leading causes of years lived with disability and 28th among the leading causes of burden of disease. Asthma kills around 1000 people every day and affects 339 million people and we find the prevalence is slowly rising [2].

In the Indian study on epidemiology of Asthma, respiratory symptoms in chronic bronchitis (INSEARCH), a survey conducted in two phases across 16 centers in India, the prevalence of asthma in adults were 20.5% with an estimated burden of 17.23million. The highest prevalence (>20%) was generally observed in Latin America, Australia, Europe, North America, and South Africa. The lowest prevalence (<5%) was observed in the Indian subcontinent, Asia-Pacific, the Eastern Mediterranean, and Northern and Eastern Europe.

Differences between countries may be due to factors such as lifestyle, dietary habits, socioeconomic differences, difference of awareness, and environmental factors. Various studies from India have reported a prevalence of Asthma varying from 3.5% to 29.5%. Our own data in 2008 showed that the prevalence in India i.e. approximately 2 crore people are suffering from Asthma. At present 25% of population suffers from allergy in India. And 5% of them are suffering from Asthma in India. And even in developed countries like USA there is 10%

increase in Asthma patients every year. Our own data in 2008 showed the prevalence of asthma in urban school children of Jaipur as 7.59%.

ANATOMY & PHYSIOLOGY OF RESPIRATORY SYSTEM:

The respiratory system comprises nose, pharynx (throat), larynx (voice box), trachea (wind pipe), bronchi, and lungs. The parts may be classified in line with either structure or function. Structurally, the respiratory system consists of two parts:

1. The upper respiratory tract includes the nose, pharynx, and associated structures.
2. The lower respiratory tract includes the larynx, trachea, bronchi, and lungs.

Functionally, the respiratory system also consists of two parts:

1. The conducting zone consists of a series of interconnecting cavities and,

2. Tubes both outside and within the lungs.

These include the nose, pharynx, larynx, trachea, bronchi, bronchioles, and terminal bronchioles, their function is to filter, warm, and moisten air and conduct it into the lungs. The respiratory zone consists of tissues within the lungs where gas exchange occurs[3]. These include the respiratory bronchioles, alveolar ducts, alveolar sacs, and alveoli; they're the central sites of gas exchange between air and blood.

Bronchi: At the superior border of the fifth thoracic vertebra, the trachea divides into a right primary bronchus, which reaches into the right lung, and a left primary Bronchus, which goes into the left lung. The right primary bronchus is more vertical, shorter, and wider than the left. As a result, an aspirated object is more likely to enter and inhabit the right primary bronchus than the left just like

the trachea; the primary bronchi contain incomplete rings of cartilage and are lined by pseudo stratified ciliated columnar epithelium.

At the significant point where the trachea divides into right and left primary bronchi an inside ridge called the carina is created by a posterior and somewhat inferior projection of the last tracheal cartilage. The mucosa of the carina is one among the foremost sensitive areas of the whole larynx and trachea for triggering a cough reflex. Widening and distortion of the carina may be a serious sign because it always indicates a carcinoma of the lymph nodes round the region where the trachea divides. On entering the lungs, the primary bronchi divide into smaller bronchi-the secondary (lobar) bronchi, one for each lobe of the lung. The secondary bronchi continue to branch, forming still smaller bronchi, called tertiary (segmental) bronchi, that divide into bronchioles. Bronchioles successively branch repeatedly, and therefore the smallest ones branch into even smaller tubes called terminal bronchioles[4]. This extensive branching from the trachea resembles an inverted tree and is usually mentioned as the bronchial tree.

As the branching becomes more extensive within the bronchial tree, several structural changes could also be noted.

1. The mucosa within the bronchial tree changes from pseudo-stratified ciliated columnar epithelium within the primary bronchi, secondary bronchi, and tertiary bronchi to ciliated simple columnar epithelium with some goblet cells in larger bronchioles, to mostly ciliated simple cuboidal epithelium with no goblet cells in smaller bronchioles, to mostly nonciliated simple cuboidal epithelium in terminal bronchioles. (In regions where simple nonciliated cuboidal epithelium is present, inhaled particles are removed by macrophages).

2. Plates of cartilage gradually replace the incomplete rings of cartilage in primary bronchi and eventually disappear within the distal bronchioles.

3. Because the amount of cartilage decreases, the quantity of smooth muscle increases.

Smooth muscle encircles the lumen in spiral bands. Because there's no supporting cartilage, however, muscle spasms can close off the airways. This is often what happens during an Asthma attack, which may be a life-threatening situation. During exercise, activity within the sympathetic division of the autonomic nervous system (ANS) increases and therefore the adrenal medulla releases the hormones epinephrine and norepinephrine; both of those events cause relaxation of smooth muscle within the bronchioles, which dilates the airways. Because air reaches the alveoli more quickly, lung ventilation improves. The parasympathetic division of the ANS and mediators of hypersensitive reactions like histamine have the alternative effect, causing contraction of bronchiolar smooth muscle, which ends up in constriction of distal bronchioles.

Lungs: The lungs are paired cone-shaped organs within the chest cavity. They're separated from one another by the heart and other structures within the mediastinum, which divides the chest cavity into two anatomically distinct chambers. As a result, if trauma causes one lung to collapse, the opposite may remain expanded. Each lung is enclosed and guarded by a double-layered serosa called the pleural membrane. The superficial layer, called the pleura, lines the wall of the thoracic cavity; the deep layer, the visceral pleura, covers the lungs themselves. Between the visceral and parietal pleurae may be a small space, the pleural cavity, which contains a little amount of lubricating fluid secreted by the membranes[5]. This pleural fluid reduces friction between the membranes, allowing them to slip easily over each other during breathing. Pleural fluid also causes the 2 membranes to adhere to one another like a film of water causes two glass microscope slides to stick together, a phenomenon called surface tension. Separate pleural cavities surround the left and right lungs. Inflammation of the

pleural membrane, called pleurisy or pleuritis, may in its early stages cause pain because of friction between the parietal and visceral layers of the pleura. If the inflammation persists, excess fluid accumulates within the pleural space, a condition referred to as pleural effusion. The lungs extend from the diaphragm to only slightly superior to the clavicles and lie against the ribs anteriorly and posteriorly. The broad inferior portion of the lung, the base, is concave and fits over the convex area of the diaphragm. The narrow superior portion of the lung is the apex. The surface of the lung lying against the ribs, the costal surface, matches the rounded curvature of the ribs. The mediastinal (medial) surface of each lung contains a part, the hilum, through which bronchi, pulmonary blood vessels, lymphatic vessels, and nerves enter and exit. These structures are held together by the pleura and connective tissue and constitute the basis of the lung. Medially, the left lung also contains a concavity, the cardiac notch, within which the heart lies. Because of the space occupied by the heart, the left lung is about 10% smaller than the right lung. Although the right lung is thicker and broader, it's also somewhat shorter than the left lung because the diaphragm is higher on the right side, accommodating the liver that lies inferior to that. The lungs almost fill the thorax. The apex of the lungs lies superior to the medial third of the clavicles and is that the only area that may be palpated. The anterior, lateral, and posterior surfaces of the lungs lie against the ribs. The base of the lungs extends from the sixth costal cartilage anteriorly to the spinous process of the tenth thoracic vertebra posteriorly. The pleura extend about 5 cm (2 in.) below the base from the sixth costal cartilage anteriorly to the twelfth rib posteriorly. Thus, the lungs don't completely fill the pleural cavity in this area. Removal of excessive fluid within the pleural cavity may be accomplished without injuring lung tissue by inserting a needle anteriorly through the seventh intercostal space, a procedure called thoracocentesis. The needle is passed along the superior border of the lower rib to avoid damage to the intercostal nerves

and blood vessels. Inferior to the seventh intercostal space there's danger of penetrating the diaphragm.

Lobes, Fissures, and Lobules: One or two fissures divide each lung into lobes. Both lungs have an oblique fissure, which extends inferiorly and anteriorly; the right lung also contains a horizontal fissure. The oblique fissure within the left lung separates the superior lobe from the inferior lobe, within the right lung, the superior a part of the oblique fissure separates the superior lobe from the inferior lobe; the inferior part of the oblique fissure separates the inferior lobe from the middle lobe, which is bordered superiorly by the horizontal fissure. Each lobe receives its own secondary (lobar) bronchus. Thus, the right primary bronchus gives rise to 3 secondary (lobar) bronchi called the superior, middle, and inferior secondary (lobar) bronchi, and therefore the left primary bronchus gives rise to superior and inferior secondary (lobar) bronchi. Within the lung, the secondary bronchi produce to the tertiary (segmental) bronchi, which are constant in both origin and distribution; there are 10 tertiary bronchi in each lung. The segment of lung tissue that every tertiary bronchus supplies is termed a Broncho pulmonary segment. Bronchial and pulmonary disorders (such as tumours or abscesses) that are localized in a Broncho pulmonary segment could also be surgically removed without seriously disrupting the encompassing lung tissue. Each Broncho pulmonary segment of the lungs has many small compartments called lobules; each lobule is wrapped in elastic connective tissue and contains a lymph vessel, an arteriole, a venule, and a branch from a terminal bronchiole. Terminal bronchioles subdivide into microscopic branches called respiratory bronchioles. As the respiratory bronchioles penetrate more deep into the lungs, the epithelial lining changes from simple cuboidal to simple squamous. Respiratory bronchioles successively subdivide into several (2–11) alveolar ducts. The respiratory passages from the trachea to the alveolar ducts contain about 25 orders of branching; branching from the trachea into primary

bronchi is termed first-order branching, from primary bronchi into secondary bronchi is termed second-order branching, and so on all the way down to the alveolar ducts.

Alveoli: Around the circumference of the alveolar ducts are numerous alveoli and alveolar sacs. An alveolus is a cup-shaped out pouching lined by simple squamous epithelium and supported by a thin elastic basement membrane; an alveolar sac consists of two or more alveoli that share a common opening. The walls of alveoli comprise of two forms of alveolar epithelial cells. The more numerous type I alveolar cells are simple squamous epithelial cells that form a nearly continuous lining of the alveolar wall. Type II alveolar cells, also called septal cells, are fewer in number and are found between type I alveolar cells. The thin type I alveolar cells are the main sites of gas exchange. Type II alveolar cells, rounded or cuboidal epithelial cells with free surfaces containing microvilli, secrete alveolar fluid, which keeps the surface between the cells and therefore the air moist. Included within the alveolar fluid is surfactant, a complex mixture of phospholipids and lipoproteins. Surfactant lowers the surface tension of alveolar fluid, which reduces the tendency of alveoli to collapse. Associated with the alveolar wall are alveolar macrophages (dust cells), phagocytes that remove fine dust particles and other debris from the alveolar spaces. Also present are fibroblasts that produce reticular and elastic fibres. Underlying the layer of type I alveolar cells is an elastic basement membrane. On the outer surface of the alveoli, the lobule's arteriole and venule disperse into a network of blood capillaries that comprises a single layer of endothelial cells and basement membrane. The exchange of O2 and CO2 between the air spaces within the lungs and therefore the blood takes place by diffusion across the alveolar and capillary walls, which together form the respiratory membrane.

Extending from the alveolar air space to blood plasma, the respiratory membrane consists of 4 layers:

1. A layer of type I and type II alveolar cells and associated alveolar macrophages that constitutes the alveolar wall

2. An epithelial basement membrane underlying the alveolar wall

3. A capillary basement membrane that's often fused to the epithelial basement membrane

4. The capillary endothelium instead of having several layers, the respiratory membrane is extremely thin only 0.5 m thick, about one-sixteenth the diameter of a red blood corpuscle to allow rapid diffusion of gases.

It's been estimated that the lungs contain 300 million alveoli, providing an immense surface area of 70 m2 (750 ft2) about the dimensions of a racquet ball court for gas exchange.

ARTERIAL SUPPLY OF THE LUNGS: The bronchial arteries supply nutrition to the bronchial tree and to the pulmonary tissue. These are small arteries that modify in number, size and origin, but usually they're as follows.

(1) On the right side there's one bronchial artery which arises either from the third posterior intercostal artery or from the upper left bronchial artery.

(2) On the left side there are two bronchial arteries both of which arise from the descending thoracic aorta, the upper opposite fifth thoracic vertebra and therefore the lower just below the left bronchus.

Deoxygenated blood is brought to the lungs by the pulmonary arteries and oxygenated blood is returned to the guts by the pulmonary veins. There are precapillary anastomoses between bronchial and pulmonary arteries[6]. These connections enlarge when anyone of them is obstructed in disease.

VENOUS DRAINAGE OF THE LUNGS: The venous blood from the first one or two divisions of the bronchi is carried by bronchial veins. Usually there are two bronchial veins on both sides. The right bronchial veins drain into the azygous vein. The left bronchial veins drain either into the left superior intercostal vein or into the hemiazygos vein. The greater part of the venous blood from the lungs is drained by the pulmonary veins.

LYMPHATIC DRAINAGE OF THE LUNGS: There are two sets of lymphatics, both of which drain into the bronchopulmonary nodes.

1. Superficial vessels drain the peripheral lung tissue lying beneath the pulmonary pleura. The vessels pass round the borders of the lung and margins of the fissures to reach the hilum.

2. Deep lymphatics drain the bronchial tree, the pulmonary vessels and the connective tissue septa. They run towards the hilum where they drain into the bronchopulmonary nodes. The superficial vessels have numerous valves: the deep vessels have only a couple of valves or no valves in the least. Though there's no free anastomosis between the superficial and deep vessels some connections exist which might open up, in order that lymph can flow from the deep to the superficial lymphatics when the deep vessels are obstructed in disease of the lungs or of the lymph nodes.

NERVE SUPPLY OF THE LUNGS:

1) Parasympathetic nerves are derived from the vagus. These fibres are:

(i) Motor to the bronchial muscles, and on stimulation cause bronchospasm;
(ii) Secretomotor to the mucous glands of the bronchial tree; and
(iii) Sensory, the sensory fibres are accountable for the stretch reflex of the lungs, and for the cough reflex.

2) Sympathetic nerves are derived from second to fifth spinal segments. These are inhibitory to the smooth muscle and glands of the bronchial tree. That's how sympathomimetic drugs, like adrenalin, cause bronchodilatation and relieve symptom of Bronchial Asthma.

PATHOPHYSIOLOGY:

Airway obstruction in Asthma is caused by-

(i) Oedema and inflammation of mucosa lining the airways;

(ii) Excessive secretion of mucus, inflammatory cells and cellular debris and

(iii) Spasm of the smooth muscle of bronchi. Obstruction is diffuse but not uniform.

Asthma has been classified as extrinsic (IgE mediated, triggered by allergens), intrinsic (non IgE mediated, triggered by infection), mixed, exercise induced or aspirin induced.

However, all asthmatics have a common basic disorder, which can be related to some form of IgE reaction but the precise mechanism remains obscure. Inhalation of an allergen, leads to a biphasic response with early and late reactions ultimately causing broncho constriction.

Early reaction starts within 10 minutes of the exposure I to allergen. It is characterized by release of histamine, leukotrienes C, D, and E, prostaglandins, platelet activating factor and bradykinine from the mast cells that follows the interaction of allergen with specific mast cell bound IgE.

All these substances cause bronchoconstriction, mucosal edema and mucus secretion, which manifests as airway obstruction. This phase is inhibited by P2 agonist drugs. Late phase occurs in about two-third of patients. It develops 3-4 hours later with peak at 8-12 hours. Again there's a release of mastocyte mediators. This phase isn't prevented by premeditation with P2 agonist drugs. However, it's inhibited by premedication with steroids suggesting that airway narrowing is being mainly due to an inflammatory reaction and mucosal edema. This phase presents as clinical asthma. Airway resistance is increased more so

during exhalation because airways close prematurely during expiration. As a result lungs are hyper-inflated; elasticity and frequency dependent compliance of the lungs is reduced. Breathing involves more work leading to dyspnoea. Perfusion of inadequately ventilated lungs causes low PaO2. In early stages of illness, PaCO2 also falls due to hyper-ventilation caused by dyspnea. When obstruction becomes more severe, alveolar hypoventilation supervenes. This results in retention of CO2 with an increase of PaCO2. With the exhaustion of buffer mechanisms, pH of blood falls (respiratory acidosis).

Bronchial hyper-responsiveness and Asthma: Bronchial hyper responsiveness is one among the foremost characteristic features of asthma.

This is often attributed to one or more of the subsequent abnormalities:

(i) Defect within the airway,
(ii) Abnormal neural control of the airways, and
(iii) Bronchial inflammation: It is suggested that an imbalance between excitatory (cholinergic, alpha-adrenergic and non- cholinergic) and inhibitory mechanisms (Beta adrenergic and non-adrenerrgic) increases bronchial reactiveness.

Broncho constriction results from increased cholinergic activity causing bronchial smooth muscle to contract. Broncho dilation results from non-adrenergic system and endogenous catecholamines acting on the beta-adrenergic receptors and prostaglandin E2. There are both inhibitory and excitatory non adrenergic non-cholinergic nerves, which secrete certain neuropeptides. Two of those peptides have been well studied. Vocative intestinal peptides (VIP) relax smooth muscles of bronchi while substance-P increases I-smooth muscle tone, mucus hypersecretion and microvascular leakage.

PATHOLOGY:

In a patient who has died of acute Asthma, the most striking feature of the lungs at necropsy is their gross over distension and failure to collapse when the pleural cavities are opened. When the lungs are cut, numerous gelatinous plugs of exudates are found in most of the bronchial branches down to the terminal bronchioles. Histological examination shows hypertrophy of the bronchial smooth muscle, hyperplasia of mucosal and sub mucosal vessels, mucosal edema, denudation of the surface epithelium, pronounced thickening of the basement membrane, and eosinophilic infiltrates in the bronchial wall. In Asthmatic patients who die from trauma and causes other than Asthma itself, mucus casts, basement membrane thickening, and eosinophilic infiltrates are frequently observed. In both situations there is an absence of the well-recognized forms of destructive emphysema. In a small proportion of Asthmatics that die, the eosinophilic infiltration is replaced by neutropils, and mucus plugging is conspicuously absent. The reasons for these differences are not yet clear [8].

CLINICAL FEATURES:

The symptoms of Asthma consist of a triad of dyspnea, cough, and wheezing, the last often being regarded as the sine qua non. In its most typical form, all three symptoms coexist. At the onset of an attack, patients experience a sense of constriction in the chest, often with a nonproductive cough. Respiration becomes audibly harsh; wheezing in both phases of respiration becomes prominent; expiration becomes prolonged; and patients frequently have tachypnoea, tachycardia, and mild systolic hypertension. If the attack is severe or prolonged, there may be a loss of adventitial breath sounds, and wheezing becomes very high pitched. These two signs Status asthmaticus is a clinical

diagnosis defined by increasingly severe asthma not responsive to drugs clinical feature: Dyspnea and cough unable to speak properly lack of proper sleep.

ON EXAMINATION:

Severe dyspnea, patient tripod position, accessory muscles of respiration, cyanosis, patient is agitated or in coma.

Investigations: There is no single satisfactory diagnostic test for all asthmatic patients. Respiratory function tests Measurements of peak expiratory flow (PEF) on waking, prior to taking a bronchodilator and before bed after a bronchodilator, are particularly useful in demonstrating the variable airflow limitation that characterizes the disease. The diurnal variation in PEF is a good measure of Asthma activity and is of help in the longer-term assessment of the patient's disease and its response to treatment. To assess possible occupational asthma, peak flows need to be measured for at least 2 weeks at work and 2 weeks off work

1) Spirometry is useful, especially in assessing reversibility. Asthma can be diagnosed by demonstrating a greater than 15% improvement in FEV1 or PEF following the inhalation of a bronchodilator. However, this degree of response may not be present if the asthma is in remission or in severe chronic asthma when little reversibility. The carbon monoxide transfer test is normal in Asthma.

2) Exercise tests: These have been widely used in the diagnosis of Asthma in children. Ideally, the child should run for 6 minutes on a treadmill at a workload sufficient to increase the heart rate above 160 beats per minute. Alternative methods use cold air challenge, isocapnoeic hyperventilation (forced over breathing with artificially maintained PaCO2) or aerosol challenge with hypertonic solutions. A negative test does not automatically rule out asthma.

3) Histamine or methacholine bronchial provocation test: This test indicates the presence of airway hyperresponsiveness, a feature found in most asthmatics, and can be particularly useful in investigating those patients whose main symptom is cough. The test should not be performed on individuals who have poor lung function (FEV1< 1.5 L) or a history of 'brittle' asthma.

4) Trial of corticosteroids: All patients who present with severe airflow limitation should undergo a formal trial of corticosteroids. Prednisolone 30 mg orally should be given daily for 2 weeks with lung function measured before and immediately after the course. A substantial improvement in FEV1 (> 15%) confirms the presence of a reversible element and indicates that the administration of inhaled steroids will prove beneficial to the patient. If the trial is for 2 weeks or less, the oral steroids can be withdrawn without tailing off the dose.

5) Blood and sputum tests: Patients with Asthma may have an increase in the number of eosinophils in peripheral blood (> 0.4 × 109/L). The presence of large numbers of eosinophils in the sputum is a more useful diagnostic tool.

6) Chest X-ray: There are no diagnostic features of Asthma on the chest X-ray, although over inflation is characteristic during an acute episode or in chronic severe disease. A chest X-ray may be helpful in excluding a pneumothorax, which can occur as a complication, or in detecting the pulmonary shadows associated with allergic bronchopulmonary aspergillosis.

7) Skin tests: Skin-prick tests should be performed in all cases of Asthma to help identify allergic causes.

8) Allergen provocation tests: Allergen challenge is not required in the clinical investigation of patients except in cases of suspected occupational asthma. If the patient has asthma without any other systemic features, then food allergy is most unlikely to be the cause .

CASSIA SOPHERA:

It is indigenous to India and has been frequently used by ancient Indian physicians for its efficacy in respiratory disorders. Its sanskrit name is, Kasamarda "which means destroyer of cough". It is introduced by WEST BENGAL physician DR. N. K. BANERJEE.

The medicine belong to vegetable kingdom. It is a shrub, glabrous, about 3 m. in height. The compound leaves with 8-12 paired leaflets acute and tapering; bear rachies with single gland at the base. It has yellow flowers in carymbose racemes.Symptoms - Difficult breathing, worse dust, smoke[9]. Dyspnea, worse winter, change of weather, Dyspnea, worse cold drinks, walking. Thick yellow expectoration, Better by rest and pressure. better lying quietly, better cold, cold applications. Better cold air. Better cold drinks. Worse by warmth. Worse by motion [10]

It is a medicine of Bronchial asthma in children below 12 year of age. It is used as Bronchodilator and a powerful expectorant. It is a great antisyphilitic medicine i.e. –Baby have got syphilitic asthma—there may be bloody sputum with a history of head injury during birth. There is pale , cyanotic face with grasp of fresh air.

AGG. — Cold room ; Wet basement ; Rainy weather ; Forward bending. AME.– Open air; Rubbing ; Carring rapidly.

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