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TO STUDY THE EFFICACY OF ARSENICUM ALBUM IN THE TREATMENT OF ATOPIC ASTHMA IN PAEDIATRIC AGE GROUP

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ABSTRACT:

Atopic asthma has highest incidence in morbidity and treatment expenditure along with limitation and side effect of drugs of Allopathy. Homoeopathy possesses a better scope in treating Atopic Asthma. Homoeopathic treatment became less expensive compare to other system of medicine and it offers completely cure the disease, gives better future to the patient. Homoeopathy uses medicines prepared from natural substances that are similar to the illness unlike conventional medicines which treat, and often suppresses the patient's symptoms. Homoeopathy is much safer and quicker than other mode of treatment.

KEYWORDS: Atopic asthma, Arsenic Album, Paediatrics, Homeopathy and Homeopathic Medicine.

INTRODUCTION:

As far as I know, homeopathy is the only medicine system that seeks to "treat" this disease instead of trying to provide symptomatic relief. When dealing with the case of asthma, homeopath not only records the symptoms of the disease, but also studies medical history, family history, physical and psychological qualities of man. This helps to find the cause, colliding factors and hereditary tendencies, etc. A special interest in homeopath is the history of suppression of skin disease. Homeopaths believe that when there is a tendency or predisposition for disease - first it is reflected in less vital organs towards the periphery (such as the skin). If this manifestation is suppressed before the disease moves inside, towards vital organs (such as lungs, heart, brain, etc.).

Allopaths observe the fact that eczema often precedes asthma in children. This is written in all their medical textbooks. They say that children often "move" eczema and asthma "Movement-Into". But they are unable to make a correlation. Homeopaths believe that suppression of eczema does not cure the person's disease/sensitivity, only drives it in.

Now, after finding symptoms and cause, homeopath is trying to find a medicine that corresponds to the symptoms and the general characteristics of the person. The selected medicine is given to the patient.

Homeopaths are often (not necessarily) observed that when a real medicine is served, asthma disappears, but the old eczema (if it was originally there) will again appear for some time before it finally disappears. This recreation of old symptoms is considered to be the vomiting of the disease process and homeopaths are considered a very good prognostic feature.

In homeopathy, there are plenty of medicinal products for asthma and it is not possible to mention them all here. Some of the common drugs are ARS-ALB, IPECAC, Lachesis, Pulsatilla, Spongia, Sulfur, Ignatia, Antim-Tart, Hepar-Sulph, Nat-Sulph, Tuberculinum, etc. The selection of medicine differs from the patient to the patient.

The word "asthma" is derived from Greek, which means "catching after catching" or "stressed breathing". Asthma is a condition characterized by paroxysmal whistling shortness of breath (difficulty in breathing), mainly exhale.

According to etiology, bronchial asthma is divided into the following groups:

1. Allergic (external/ atopic) - this type of asthma usually begins in childhood and often precedes eczema. But most young adults (<35 years) develop asthma also fall into this category. Genetic factors also play an important role. In this type of asthma, the allergen leads to the production of excessive (IgE) immunoglobulins.
2. Infectious or internal - this is not hereditary or allergic, but may be caused or at least associated with the upper airways or bronchial infection that is usually viral.

3. Psychological factors (such as anxiety, emotional stress, etc.) are often considered the only cause of some asthma attacks, but it is still not certain whether it may be the only cause or it is just a colliding factor.
4. Asthma Profession - this may occur in some industries in which metal dust (especially platinum salts), biological detergents, tolueen disocyanate, polyurethane, flour and grain dust, etc.

Whatever the cause, this leads to the paroxysms of the bronchial obstruction produced by the extended bronchial spazd emphasized by the connection of bronchi with excessive mucus.

Repetitive episode of paroxysmal dyspnoea (difficulty breathing)

Breathing is strenuous, with sand sound, especially after.

Asthma attacks often occur in the early morning hours (when there is no immediate cause). During the attack, patients often prefer to sit and lie down.

The basic idea in homeopathic arsenic is terrible anxiety, restlessness and nervous exhaustion.

Arsenicum-osicality are great organizers; They work hard and efficiently. They are ambitious people, competitive; They like the best. They are proud of their achievements. The delegation is difficult for them because they want to remain under control. They are convinced that they are right. Arsenicum - bosses or -parents or -teachers will try to get the best of their employees, children or students, but they will probably do it in a dominant way. If they notice that you try to give the best, they will support you 100 %, but if they find out that you are not going well, they can disappoint you completely and consider it very justified. They are very critical people who will clearly inform you about their compassion and their antipathy - and make sure there is nothing in between.

Arsenicums are perfectionists and participate in every detail. These are people who will not rest before the images hang perfectly or who will verify the work of the maid by checking whether dust remains in the hidden corners. Arsenicum Personality that feels good is worth their weight in gold for the organization, but if they are unbalanced, they will be nervous, restless, deeply worried and can be burned.

REVIEW OF LITERATURE:

Allergic bronchitis

Aetiology - allergic bronchitis includes a heterogeneous group of patients and attempts to find a common denominator between situations such as chronic respiratory inflammations, allergies and psychogenic factors. However, it turned out that the unique common denominator is hyper -RACTIVE airways, which are manifested with variable respiratory obstruction. 3 occurs in response to a wide range of endogenous and exogenous stimuli. 4 Many stimuli may cause allergic bronchitis (asthma) attack.

Preliminary factors

* Air pollutant
* Allergens
* Bronchopulmonary aspergillosis.
* Emotional stress, happy and sad events.
* Environmental factors (cold air, wind, fog, ozone, sulfur dioxide, cigarette smoke, diesel vapors, household chemicals).
* Exercise
* Food and preservative additives (monosodium glutamate, tartrazine, metabisulphite).
* Irrigative dust and vapors.
* Menstruation.
* Pharmacological agents (aspirin, non -steroidal anti - inflammatory drugs)
* Occupational exposure.
* Reflux with food.
* Sinus disease.
* Sleep
* Viral infections.

The respiratory hyperactivity is manifested by susceptibility to extended but reversible narrowing of the airways in response to various stimulating factors. It is associated with narrowing of smooth airways, inflammation and mucosal edema, mucus accumulation and influx of inflammatory cells including neutrophils and eosinophils.

There are many ways these patients are heterogeneous. Includes:

1) Factors responsible for the precipitation of the attack (outer with an attack that has been precipitated allergen immunoglobulin E (LGE), internal or mixed)
2) Placing the airway obstruction (large airways, small airways or both).
3) The degree of reversibility of the airway obstruction (complete with a short or extended wheel - treatment - hours or incomplete.
4) Starting (acute or chronic)
5) Frequency (asthmatic), episodes of allergic bronchitis (once a year, three months, every month, every week, 2, 3 days or daily).
6) The grade of severity:
    a) mild and rarely
    b) frequent and episodic
    C) Chronic: mild (without limiting activity), mild (activity limited by drugs) and heavy (short breath with significantly limited physical activity).
7) Periodicity: seasonal, perennial, day (night or morning immersion) or unstable.
8) The length of the attack (abbreviation due to a spasm of smooth muscle or longer due to grouping and swelling of mucus).

9) Symptomatology: (cough - central airways with abundant cough receptors, breathless - smaller airways with sparse cough receptors or both symptoms.) 10) reactions to therapy (theophylin, beta 2 agonists or glucocorticoids).

From the clinical angle, asthma is categorized into external and internal varieties according to the presence or absence of external comprehensive factors. External asthma is attributed to the patient's immunological response to the exposure to the allergen, the substance to which there is a specific hypersensitivity. It occurs in atopic entities (only a small percentage of asthmatics) who demonstrate the immediate reactions of Wheal & Flare to the allergen battery. This is due to the reaction of hypersensitivity of type I (immediate) hypersensitivity mediated by IgE.

John Donne said: "No man is an island, man has a coast of skin, intestines and airways, places where the external and human internal environment is established. Some people react in a special way when they are exposed to agents either in their external or inner millieu."

External atopic asthma: The exposure of antigen in atopic individuals stimulates peripheral lymphoid tissue to synthesize the resumed antibodies. Again, it belongs to the special class of immunoglobulin - IgE. Normally the average IgE level in adults is 200 ng/ml (1 ng = 1/1 000 000 mg). They will regain 2 pieces of FAB and FC. FC pieces are repaired in mast cells located just below the bronchial epithelium and blood vessels and into the circulating basophilic leukocyte. The matrix of the cytoplasms of mast cells is filled with metachromatically dyeing cytoplasm. Granules appear as WHORLS or parallel lines. Any other exposure to the same antigen leads to the combination of antigen with antigen receptors on the FAB parts of two IgE antibodies. The result is an exocytosis cell deregulation. Contactile cell elements - microtubules and microfilaments are involved in this process. Calcium entering the mast cells induces the contraction of microfilaments and the release of anaphylaxis mediators.

Provocation of the airways with allergen extracts can cause a rapid onset of bronchoconstriction, reaching a maximum of 15 to 30 minutes after the challenge and recovery in the next two hours. Many individuals show the second wave of bronchoconstriction, which starts 4 to 6 hours after the challenge and lasts for 12 hours. These reactions are the result of cellular and biochemical mechanisms and are accompanied by increased sensitivity. The condition may be seasonal or permanent depending on the timing of exposure to an offensive allergen.

External atopic (asthma) allergic bronchitis is a condition observed in children and young adults. The condition may have a late onset of adults with decreasing atopic condition. This is due to known external allergens and the onset is determined by constitutional factors. There is a congenital ability to produce again in large quantities very easily to the substances of everyday exposure in life. The prick test reveals the existence of immediate sensitivity to the skin to the wide range with which they commonly occur. Often there is a family history of allergy such as hay, asthma, eczema or rhinitis.

In terms of bronchial challenge, the allergen causes asthma attack in ten minutes. Generally inhalants act as allergens. However, food can act similarly in childhood. Occasionally there are paroxysmal attacks of shortness of breath and can show seasonal variations. History is often extended. Remise is often recorded in puberty.

The mechanism - the human airways are controlled by myogenic, neurogenic and chemical mechanisms. Activity comes from smooth muscles. Neurogenic control occurs through vagus nerves innervate muscles. Blood - carried and locally - derived substances bring chemical control.

Bronchial asthma is a complex and heterogeneous disorder characterized by episodic respiratory obstruction, hyper response and respiratory inflammation. The pathogenesis of asthma is not well introduced. Multifactorial stimuli, such as allergens, infections and chemicals, are responsible for respiratory hyperactivity, which leads to obstruction that over time changes either spontaneously or in response to treatment.

Bronchial hyper sensitivity - attempts to clarify the bronchial asthma mechanism have shown that it is a chronic inflammatory disease of the airways. The causal substance must be able to cause persistent airway inflammation with non -specific bronchial hyperactivity (BHR) and subsequent airflow obstruction.

Inductors of asthma are in a genetically suitable host. They include antigens that operate through the immunological mechanism, infections of respiratory tract (rhinoceros, respiratory syncytial virus, influenza and para -chip) and sometimes chlamydia, chemical sensitizing agents (toluene di - isocyanate) and organic dust, noxic gases (dioxides). Many triggers are able to produce asthma in situations of underlying airway inflammations and bronchial hyperactivity. They include exercise, inhalation of cold, dry air, physical or emotional stress, hyperventilation or inhalation of irritants.

Airway inflammation - allergic bronchitis (asthma) was considered a bronchospastic disease with treatment directed to the smooth respiratory muscle. The histopathological study of the respiratory tract from patients with asthma who died during a serious attack by asthma has shown that the inflammatory response plays a dominant role than the response of the smooth respiratory muscles.

In the airways, the presence of significant inflammation with infiltration of inflammatory cells, especially eosinophils, epithelial cells that slipped into the lumen and the connection of lumen airways by viscous mucus. There is a smooth muscle hyperplasia. A biopsy respiratory study in mild asthma revealed the airways of the release of ciliary epithelium, the deposition of collagen under the basement membrane, partial granulation of mast cells and infiltration of eosinophilic cells of laminate.

Changes maintained a look at the Cozer, which wrote a century ago that bronchial asthma in many cases is a special form of inflammation of smaller bronchiols.

Respatter types - respiratory reaction to sensitization agent can be 3 types: isolated instantaneous, isolated late or dual reaction. In sensitized individuals, inhalation of allergen has immediate bronchoconstriction and can be solved in 1-3 hours. Some individuals are followed by a late increase in respiratory resistance. It develops 4-6 hours after inhalation and can persist for 8 to 12 hours or more. Early and late reactions develop through various mechanisms. Early response is caused by acute bronchoconstriction in response to mediators released from sensitized caustic cells.

Immune mechanism - inhalation of allergen substances into the lower respiratory tract can produce in a small group of immunoglobulin antibodies of immunoglobulin E (IgE). Any subsequent exposure to such a substance creates a number of immunological and biochemical reactions that lead to asthma. Abnormalities will bind allergens to cells - bound IgE on caustic cells or basophilic cells. There is a cross connection between two cellular - bound molecules of IgE molecule allergen and subsequent tasting of bassophiles of mast cells and release of mediators, leading to immediate hypersensitivity.

Protein substances such as pollen grains, weeds, forms, domestic dust, mite, animal and non -protein substances such as platinum salts, toulene disocyanate and trimellitic anhydride cause an immunological response through IgE dependent interaction.

Spring cells are present in the lining of bronchiols and bronchi under the basement membrane and adjacent to capillaries. Driving cells are probably also involved in an immediate answer to other non -mediated stimuli. However, cell ointment is not involved in infection and internal asthma. Preliminary

treatment stabilizers of mast cells (beta - 2 agonists and sodium cromoglycate) can prevent immediate reaction and cannot reduce the bronchial hyper sensitivity. The late response characterized by airway inflammation is inhibited by preliminary processing of corticosteroids. Corticosteroids reduce bronchial sensitivity. They have no effect on mast cells and an early response. Bronchoalvelor Lavage has shown that the late reaction is associated with the influx of eosinophils and neutrophils into the airways.

Inflammatory cells - previously allergic bronchitis (asthma) were perceived only as tasting of move cells induced by allergen, which has led to the release of mediators such as histamine and slow reacting anaphylaxis (A) and chemotactic factors from neutrophils (NCF) & eosinophil). Asthma is now considered a chronic inflammatory disease involving multiple interactive cells. Each cell speaks with the other and sends messages. These cells release a wide range of inflammatory mediators that activate several trigger cells in the airways, resulting in bronchoconstriction, microvascular leakage and edema, secretion of mucus and stimulating nerve reflexes. Inflammation and inflammatory mediators seem to support bronchial hyper sensitivity. Neural mechanisms can contribute to these inflammatory events.

Inflammatory stimuli on the airways activate cells that normally occur in lungs referred to as primary effectors cells. They are caustic cells, basophils and alveolar macrophages, epithelial and endothelial respiratory cells. When activated, they release mediators that are chemotactic for cells derived from circulation. They are referred to as secondary effectors cells and include eosinophils, neutrophils, t - lymphocytes and plates. These cells and their mediators module the respiratory reactivity. Mediators can be pre -remembered or generated.

Macrophages present in the airways are activated by IgE -dependent mechanisms. They release a large number of mediators such as thromboxane, prostaglandins, and factors of activation of plate (PAF). Corticosteroids inhibit the release of mediators from macrophages, unlike caustic cells. Macrophages are likely to start late response and bronchial hyper sensitivity.

Asthma respiratory tracts are characterized by eosinophils. The condition is often considered to be chronic eosinophilic bronchitis. At the time of the late response after the allergen challenge, there is a significant increase in activated eosinophils of Hypodense in the bronchoalvellor of the fluid. There is a close relationship between the number of eosinophils and the bronchial hyper sensitivity and C4, paf, oxygen radicals and basic proteins (the main part and eosinophilic cationic protein). The latter causes damage and relaxation to the airway epithelium. Eosinophils are sensitive to corticosteroids. Corticosteroid management causes reduction of tissue and circulating eosinophils.

Although there is an infiltration of neutrophils, their role is not determined. Plates by release of mediators such as serotonin, thromboxane and lipoxygenase products can cause bronchial hyper sensitivity. Lymphocytes may have a regulatory effect on an inflammatory response.

Obstruction of the respiratory tract - it turned out that six factors were functional in the production of airway obstruction in bronchiols in asthma: spasm of smooth muscle, mucosal infiltration, mucus secretion, epithelium damage and membrane reinforcement. The release of mediators together with infiltrate inflammatory cells, especially eosinophils, and to some extent neutrophils in the airway wall provide a strong stimulus for pathological changes in asthma.

Inflammatory mediators -

Wide inflammatory respiratory mediators participate in asthma and cause inflammatory changes and cause the respiratory sensitivity. Histamine, prostaglandins, sulfidopeptide leukotrienes (formerly referred to as SRS -A) and thromboxans cause the contraction of the smooth respiratory muscles, increase microvascular leakage, stimulate the secretion of mucus mucus, attract and active inflammatory cells that release the media.

Histamine is a pre -remembered substance associated with a powerful bronchospastic mediator and is found in mast cells and bassophils. Its action is mediated by the H1 receptors associated with the membrane. Bronchial smooth muscle contraction, increased vascular permeability, increased airway mucus production and prostaglandin production are available. Cross connections to cells bound to cells activate a number of enzymes that mobilize arachidonic acid from membrane phospholipids. The oxidation pathway of cyclooxygenase produces prostaglandins PGE2, PGF 2, PGD2 and PGI2) and thromboxans (TX) or lipooxygenase pathway is produced by hydroxyl fatty acids and sulfidopeptide leukotrienes (LTC4, LTD4 and LTE4).

Sulfidopeptide and leukotrienes (salts) are produced after an antigen challenge in the sensitized plice. Many macrophages and eosinophils are responsible for their production. LTC4, LTD4 and LTE4 are strong suppliers of smooth airways. They cause vasoconstriction, increased vascular permeability and stimulate mucus production.

Cyclooxygenase products (prostaglandins and thromboxans) are synthesized by inflammatory cells of the lungs, PGE2 and PGI2 cause the airway smooth muscle relaxation, while PGR2x and TXA2 cause smooth muscle contraction. PGE2 and PGI2 cause vasodilation and vasoconstriction is caused by TXA2, PGD2 increases vascular permeability and stimulates chemoinesis of neutrophils. However, they are not considered important mediators of asthma inflammation.

Cytokines are strong substances to induce airway inflammation. Interleukin - 5 (IL - 5) produced by T lymphocytes acts as eosinfilietine and eosinophilic activation factor. Eosinophilic activity is increased by interleukin - 3 and granulocyte macrophage colonies - stimulating factor (GM - CSF) of lymphocytes.

Mediators are a diverse group of highly active biological and pharmacological substances that are able to initiate immediate and delayed local immune inflammatory response. Early reaction causes bronchospasm, exudation of proteins and plasma to bronchial walls and secretion of mucus mucus. The late response is characterized by more intense specific phagocytic infiltrates into bronchial walls that are able to amplify the immuno -inflammatory response initiated by the initiated cells.

The epithelium is damaged by the effects of basic proteins derived from eosinophils and probably also with oxygen radicals released by various inflammatory cells. Inflammatory mediators increase the permeability of the epithelium by disturbing tight connections between epithelial cells. It allows access to large molecules and antigen to floor mast cells.

There is a micro vascular escape in the airways. The leakage occurs in the post of capillary vens. The consequences are swelling of the respiratory tract, release of epithelium and exudation of plasma. Plasma not only provides inflammatory mediators, but also reacts with the edition and obstruction of the peripheral airways.

Damage to the airway epithelium recorded in the exposure of viral infection and allergen plays an important role in the respiratory hypersiviskot. They remove the protection of the smooth respiratory muscles, cause loss of relaxation factors, release chemotactic mediators for

inflammatory cells, and reveal the sensory nerve endings and irritating receptors of the influence of inflammatory mediators and physical substances to activate nerve reflexes.

Factor activating plates (PAF, PAF-ACETHER) is complex phospholipids and is released from the lipid membrane. It is a strong mediator of inflammation. It seems to play a central role in evoking a lasting increase in bronchial sensitivity. It causes bronchoconstriction, increased vascular permeability and vasoconstriction. PAF can be a molecular connection connecting various inflammatory cells involved in asthma. Eosinophils are strongly attracted and activated with paf. PAF is produced by blood plates, vascular endothelial cells and various leukocyte populations such as macrophages, monocytes, eosinophils and neutrophils. It is not released from mast cells.

PAF releases eosinophilic chemoatractants, including eosinophilic chemotactic factor anaphylaxe-a (ECF-A) and leukotrien B4 (LTB4). In addition to PAF, other mediators are released eosinophils superoxide and oxygen, hydrogen peroxide, LTC4 and cationic proteins. PAF has been shown to cause bronchoconstriction, vascular permeability, bronchial edema and hyperssecrection and bronchial epithelial dysfunction.

In addition, various chemotactic mediators, such as eosinophilic chemotactic factor -a (ECF -a) and neutrophilic chemotactic factor (NCF) in lung bradykinin, peptide product of plasma kininogen using enzyme, the cause of vasodilation. Serotonin is released by neuro-epithelial bodies located in bronchial and alveolar walls as well as aggregation of plates. They cause bronchial, increased vascular permeability and increased bronchial secretion.

Histamine, prostaglandins and leukotrienes derived from activated caustic cells are responsible for early reaction with allergen inhalation. Mediators released from activated mast cells induced by an IgE -dependent mechanism are inhibited by stimulants of beta 2 - adrenuceptor, sodium cromoglycate and non -blood sodium.

In the late reaction there is increased bronchial sensitivity and can be inhibited by sodium cromoglycate and corticosteroids. In smaller airways, there is a reduction in air flow. It is characterized by mucous edema, mucus hyperse and exudation of plasma proteins. Contractions of the smooth respiratory muscles play a minor role.

It seems that the late reaction does not seem to be an anus -dependent event. Macrophages, monocytes, eosinophils, t - lymphocytes and plates are able to expresses receptors for IgE on their cellular surface and are able to respond to IgE dependent stimulation by release of mediators after a suitable stimulus. There is an influx of polymorphonuclear leukocyte and eosinophils into the bronchial epithelium and lumen. Leukotrienes and paf - lead to mucous membranes and exudation of plasma proteins.

The non -aginic mechanisms inducing the reaction of type II hypersensitivity are a precipitate mediated and the manifestation of asthma occurs five to six hours after exposure to the allergen. It occurs in no -atopic individuals.

Epithelial damage occurs by the effects of basic proteins derived from eosinophils and probably also oxygen radicals released by various inflammatory cells. Inflammatory mediators increase the permeability of the epithelium by disturbing tight connections between epithelial cells. It allows access to large molecules and antigens to submicose mast cells.

There is a microvascular escape in the airways. The leakage occurs in the post of capillary vens. The consequences are swelling of the respiratory tract with the release of epithelium and exudation of plasma. Plasma not only provides inflammatory mediators, but also responds with mucus to form mucus clogging and peripheral airway obstacle.

Damage to the airways recorded in viral infection and exposure to allergens play an important role in the respiratory hypersiviskot. They remove the protection of smooth respiratory muscles due to loss of relaxation factors, release chemotactic mediators for inflammatory cells and reveal the sensory nerve endings, and some interaction between nerve and inflammatory mediars. Many inflammatory mediators bring release neurotransmitters. Neural mechanisms can contribute to an inflammatory reaction in the airway wall and asthma features. Cholinergic mechanisms form the dominant nervous bronchoconstrictive path in the human airways. Many stimuli that produce bronchospasm also stimulate afferent receptors and can lead to reflective cholinergic bronchoconstriction and facilitate the release of acetylcholine from the end of the motor nerves in the airways. Since anticolinergic drugs are not very effective in asthma, the cholinergic mechanism does not seem to play an important role.

The adrenergic mechanism includes sympathetic nerves. They do not directly check the smooth airway muscle and circulating catecholamine, especially epinephrine, can play an important role in the regulation of bronchoconstrictor tones. Inflammatory mediators also increase the function of alpha adrenergic receptor and cause bronchoconstriction. However, the effect is insignificant.

The vasoactive intestinal peptide (VIP) is a strong airway relaxant, and if enzymes released from inflammatory cells are quickly degraded, it causes an exaggerated bronchoconstrictive response. Other neuropeptides such as the P, Peptide related to the Neurokinin A and Calcitonine gene could be released by the local axon reflex after damage to the airway epithelium that revealed afferent nerves of the effect of inflammatory mediators. The result is bronchoconstriction, microvascular leak and mucus secretion.

The airway hyperspicism mechanism seems to be caused by the interaction of inflammatory cells and meditators with smooth respiratory muscle and its nervous control. Thus, myogenic, neurogenic and chemical mechanisms act together in airway inflammation, hyper sensitivity and obstruction.

Homoeopathic therapeutic atopic asthma

Arsenic album

Astma worse at 12:00 until 2 hours.

Asthma, better sitting upright. Dyspnoea, worse lying, better sitting or bent forward. Feeling as if the lungs were full of smoke or dust. Angina pectoris. Cardiac inflammation from suppressed eruption.

Atrial fibrillation. Congestive heart failure with irregular rhythm, cyanosis and shortness of breath has improved from sitting, restlessness, anxiety, weakness and great cold. Cough worse colds or in the open air, worse drinking, especially cold drinks, worse at 1 or 2:00. Bronchitis, pneumonia, pleural discharge. Empyzema, breast malignancy.

BELLADONNA

Breathing difficult agg - warm, wet weather. Breathing moan. Brief breathing. Breathing is difficult to accompany widespread pupils.

Asthma after a cold.

Drosera

Cough the violent paroxysma coughs, sometimes so serious that the patient captures or becomes cyanotic. Cough, worse after midnight. Cough so serious that it produces epistaxi. Cough worse immediately after lying at night. Vomiting for cough. A painful cough, a patient who has a lot of possession, has a chest in cough. Many authors use Drosera in the case of tuberculosis.

Kali bichromicum

Asthma, worse at night, especially 1 to 2:00.

Productive cough with strong or stringed green sputum in asthma or bronchitis. Rattling breathing in sleep (kali with children)

Cough worse in the morning when waking up, worse eating. Croup. Tickling the feeling of trachea bifurcation. A cold feeling in the heart area. (Kali - n).

Chest pain reaches backwards. Pain in stern.

Kali Carbonicum

Asthma, worse 2 to 3 o'clock in the morning or at 3:00. Difficult breathing, worse lying flat, better sitting upright, better lean forward and elbows resting on the knees better lying. Sewing, tear -off pain that desires the chest. Angina pectoris. Current heart failure. Palpitations with flushing of heat and shortness of breath. A cold feeling in the chest. Breast swelling.

Cough - spasmodic cough, ticking in the throat. Cough, worse night, especially 2 to 4 o'clock in the morning, worse in the morning when waking up. Cough with a thick purulent sputum with a nodd taste. Whooping - cough. Bronchitis. Pneumonia, often left - neighborly, but also lower lobe.

Pneumonia in children, screaming with sharp, sewn pains for inspiration, cannot eat or drink, cannot sleep.

Kali Jodatum

Repetitive pneumonia. Acute pneumonia especially on the right side. Asthma with aggravation at 5:00.

Wakes up with spirit and shortness of breath. The ulcer of the chest and the back.

Lachesis

Asthma after jealousy or strong emotions. Asthma, worse night, worse during sleep, worse morning when waking up, worse from a hot room. Asthma, better in the open air, better colds, better when you sit forward (Kali - C, Ars, Spong). During the crisis of asthma, they must pull out an open shirt or nightgown. They wake up with a suffocating feeling at night, especially when falling asleep. Angina congestive heart failure. Muokard infarction with narrowing in the chest, pain on the left arm. Palpitations, worse lying left side, worse night and worse sleep. Chest pressure, worse lying on the left side.

Rumex crispus

Worse colds or in the open air. Worse revelation. Worse inspiring, breath irregularity, speaking, laughter. Worse especially in the morning when waking up or at 11 am. Worse touch or pressure on the neck or larynx. Worse temperature change (warm to cold or cold to warm up). Cold air causes tickling in the throat and triggers cough. Better because sucking candies, better drink. Dry, tickling cough, krup pertussis.

Spongia tosta

Cough, usually dry, usually worse before midnight, often harsh or barking or cruel figure. A dry cough reminiscent of a "saw passing through wood" or "back of the seal". Croup, worse before midnight. Cough from tickling or irritation in the throat or chest. Cough better eating and drinking, especially hot drinks. Cough worse of cold drinks. Cough, worse eat sweets. Asthma, worse at night or during sleep, worse of respiratory infection or colds. Asthma better tilting forward, better bending the head back, better warm food or drinks.

The suffocating feelings, worse during sleep at night and wake him out of sleep (Lach, Grind, Sulph, OP) angina. Heart valve disorders.

Natrum sulfuricum

Astma worse at 4:00, wet weather, effort. Asthma of children. Asthma after grief. Asthma can stop at puberty and repeat in the thirties or later. Chronic bronchitis, rattling mucus filled with cough. The weak feeling in the chest must hold the chest when cough. Pneumonia, especially lower left lobe, productive cough with greenish sputum. Many authors consider the remedy for tuberculosis.

Chelidonium

Rectangular pulmonary affection of pain at the lower angle of the right shoulder blade, desire for warm beverages, painful cough, hemoptys. AGG - Movement. You can rattle a lot with little expectation.

CONCLUSION:

Respiratory disorders are on the increase worldwide. More than millions of people in India alone suffer from the upper respiratory discomforts. With the increasing level of pollution, the susceptibility increases to the allergy (pollen, dust, food articles) which causes inflammation of mucous membranes of nostrils.

In modern science the treatment of allergic disorders consists of suppressions of the symptoms & cauterization of any growth, if present which causes gradual deterioration of health of the patient. The treatment is essentially only a palliation, not a cure, only there is relief of symptoms during their acute attack.

By increasing knowledge of side effects of this treatment the patients are more likely to incline towards the alternative mode of treatment, which are free from side effects. Out of that Homoeopathic treatment is preferred by these populations.

The study shows that homoeopathy plays very important role in the treatment of Allergic Respiratory disorders. It can be said that homoeopathic medicines are effective in the treatment of Allergic disorders provided exact medicine, exact potency & precautions are maintained by physician & patients both.

All cases were studied in detail for all their trouble and given medicine on the principles of homoeopathy and if needed revised case taking done with medicine till follow up and remedy prescribed were found helpful.

From the above discussion, I came to the following observations:

Allergies are the most common immunological diseases among general population, and increasing evidence suggests that incidence of allergic disorders is rising dramatically

The clinical studies show the effectiveness of Arsenic album in atopic asthma.

Homoeopathy can be used in reducing the intensity of atopic asthma, if each case is taken thoroughly & individually. The frequency of attacks can be minimized with homoeopathic treatment, only if medicines are selected strictly according to the principle & laws of Homoeopathy. Patients with psora as the underlying miasm, tend to have a better prognosis with homoeopathic medicine Arsenic album.

Anti-miasmatic and/or constitutional medicine have a better prognosis in homoeopathy. The effectiveness and duration of cure of treatment will differ from individual to individual. The greatest advantage of treating by homoeopathic medicine is that it improves without hazards of drug dependence which usually happens in conventional medicines.

The prevention is better than cure so people should be educated to avoid dust, smoke, pollution etc. because these environmental factors are the main exciting and/or maintaining causes.

I know that after all level study about miasm that miasmatic consideration is necessary all the way to manage patient in allergic diseases may be atomic asthma or anything.

Psora is the fundamental miasm were diseases have to be managed with homoeopathy always. Fundamental miasmatic defects can be corrected with suitable constitutional remedy.

Follow up assessment of case was based on subjective and objective criteria's following Herring's guidelines which says that cure takes place in direction is above downwards, within outwards, from more important organs to less important, from generals to particulars and from subjective relief to improvement in objective pathological parameters and its clinical correlation.

The appearance of symptom since early stage of disease indicated the remedy .given on basis of totality of symptoms could arrest the further progression of disease, thus homoeopathy acting much faster and efficiently before the patients gets crippled with the disease.

In the assessment the sensitive index includes the temperature, debilitating joint pains, intensity of pains, and clinical correlation along with investigations, from the case study a criteria for the assessment is been put forwarded which has helped to understand better the process of presenting complaints of fever and arthralgia both during the initial presentation and its follow up.

Patient approaching with the initial presentation of fever followed by joint pains, observe the temperature record, indicating the process of disease if the remedy is been prescribed it is homoeopathic similimum if there is an improvement at general level, wait and see the response and note down the changes. If temperature still rising following possibilities:

- Homoeopathic aggravation if associated with sense of well-being and > at general level
- Disease progression with increase in intensity of suffering with no general well being
- Medicinal aggravation if < presenting complaints on which remedy was prescribed, with no improvement in general symptoms, at this junction reassess the case and for the remedy or change in posology.
- Temperature declining and again coming back with same intensity, on the joint pains increase with not much relief
- Indicates change in potency, need for higher potency or either frequent repetition in relation to progression of disease and susceptibility of the patient.
- Search for other causes as said by Dr. Hahnemann, the maintaining causes and if present remove it.
- The remedy may not be the similimum as it is partially affecting and not making any changes at the level of process so reassessed the case in view of remedy.

REFERENCES/BIBLIOGRAPHY:

1. Text book of ENT Dr. Bhargava
2. Diseases of Ear, Nose and Throat By P.L.Dhingra
3. Published by B.I. Churchill Livingstone Second edition, 1998.
4. Synopsis of Otorhinolaryngology By Zakir Hussain
5. Published by Paras Medical Publisher First Edition, 2005.
6. Bailey & Love's Short Practice of Surgery By R.C.G Russell
7. Norman S. WilliamsChristopher J.K Bulstrode
8. Published by Arnold International Students Edition 3rd Edition, 1998
9. Gray Anatomy Published by Churchill Livingstone Thirty Eighth Edition, 2002
10. Therapeutic Pointers to some common Diseases By E. A Farrington
11. Select your Remedy By Bishamber Das
12. Therapeutics By Lilienthel
13. Organon of Medicine translated by William Boericke
14. Synthesis repertory Dr. Fredrick
15. Murphys's repertory Dr. Robin Murphy
16. Kent repertory Dr. J. T Kent
17. Principles and practice of Homoeopathy by Richard Hughes
18. Lectures on Homoeopathic philosophy J.T. Kent
19. www.wikepedia.com
20. www.hpathy.com
21. http://en.wikipedia.org/wiki/CURB-65

22. www.homoeotimes.co
23. Internet websites like- www.google.co.in, www.hpathy.com,
24. www.similima.com.