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Review Article

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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ABSTRACT

Chronic Obstructive Pulmonary Disease (COPD) is a common, preventable and treatable lung disease. It is characterized by chronic respiratory symptoms and airflow limitation due to abnormalities of the airways caused by continuous exposure to noxious particles or gases. COPD occupies the third position for the cause of death worldwide. The major risk factor for the development of COPD is tobacco smoking. The inhalation of toxic particles, biomass exposure and outdoor air pollution along with other host factors including abnormal lung development and accelerated lung aging can also contribute. Spirometry characterized by non-fully reversible airflow limitation i.e., FEV1/FVC < 0.7 post bronchodilation, confirms the diagnosis of COPD. The major goals of management are to reduce risk factors, manage stable COPD, prevent and treat acute exacerbations, and manage associated illnesses. The conservative management comprising of antibiotics, inhaled corticosteroids, mucolytics etc. offer promising results in this condition. But tachycardia, glossitis, glaucoma, nausea are some of the common adverse reactions noticed on chronic use of these group of drugs. The contemporary health scenario demands more contribution in this regard from the indigenous system of medicine. In Ayurvedic view, COPD can be considered under the spectrum of *Pranavaha srotodushti vikaras.* The clinical features and etiology mentioned for diseases mentioned in Ayurveda such as *Kasa*, *Tamaka shvasa, Rajayakshma* etc has similarity with that of COPD. Though COPD cannot be correlated to any single condition of *Pranavaha sroto dushti*, *Tamaka shvasa* can be considered as includes most of the clinical features including cough. When the etiological factors favour, the vitiated and aggravated *Kapha dosha* blocks the channels of *Prana* and *Udana vata* causing the onset and symptoms of the disease.

INTRODUCTION

COPD is an irreversible lung condition characterized by airflow limitation that cause persistent and progressive respiratory symptoms.[1] Data shows above 3 million COPD deaths in 2012.[2] The prevalence of COPD are expected to surge over the coming decades due to continued exposure to the risk factors and aging.[3] COPD is caused by the complex, cumulative and dynamic gene environment interactions over the lifetime that can damage the lungs and/or alter their normal developmental or aging processes.[4] The main environmental exposures leading to COPD are tobacco smoking and the inhalation of toxic particles and gases.

Previous definitions of COPD have emphasized the terms “Chronic bronchitis” and “Emphysema”, which are not included in the definition now but contributes COPD. Chronic bronchitis is defined as chronic cough and sputum production lasting at least 3 months per year for two consecutive years. Emphysema is destruction of the gas-exchanging surfaces of the lung (alveoli). Chronic inflammation in the pathophysiology causes structural changes, narrowing of the small airways and destruction of the lung parenchyma. These changes in turn decrease the lung’s elastic recoil. Mucociliary dysfunction is a characteristic feature of the disease causing the mucus stagnation inside the airways. These changes may end

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up in airflow limitation and reduced capacity of the lungs. Airflow limitation is measured by spirometry.

Ayurvedic approach

Ayurveda – the traditional Indian system of medicine is the knowledge of life, the unique objective of which is the achievement of optimum health which includes maintenance and promotion of health in individuals and cure of illness in the diseased. This can be considered as the earliest scientific reference regarding positive concept of health which can be achieved through a blending of physical, mental, social, moral and spiritual well-being. Symptoms similar to COPD and its management are described in detail in Ayurvedic texts under *Kasa* and *Shvasa*, the *Pranavaha srotodushti vikaras*.[5] The symptoms are manageable and Quality of Life can be improved with the Ayurvedic approach of *Shodhana* followed by *Rasayana*. The lifestyle modifications have an effect in the primordial prevention i.e., prevention of risk factors. Ayurveda have a key role in primordial prevention by means of the lifestyle modification, both physical and mental and also by procedures such as *Rutu shodhana*.[6] However in the severe grades of the COPD, where there is saturation fall and associated obstructive sleep apnea or other systemic diseases, the patient needs oxygen support and emergency care where Ayurveda only has the supportive role. The role in primary and secondary prevention is limited, but helps in improving the quality of life of patients affected with COPD.

The prevention of COPD can be done by creating awareness of the disease and risk factors among the people and promoting changes in the life style like healthy diet, regular exercise, cessation of smoking etc. For fulfilment of this aim coordination among different aspects of health care and cost effective management is to be planned. Even though the present available conventional management is excellent, Ayurveda provides additional benefits such as improvement in quality of life without known side effects.

Though COPD cannot be correlated to any single condition of *Pranavaha sroto dushti*, advanced condition of *Doshika kasa* and *Tamaka shvasa* can be considered. *Kapha dosha* getting *Chaya* and *Kopa* in the presence of causative factors, aggravates and blocks the airways leading to airflow obstruction and worsening of disease.[7] Thus a protocol comprising *Agni dipana, Snehapana, Svedana* and *Shodhana* followed by *Rasayana* will be effective in the management. As *Vata kopa* and *Dhatu kshaya* plays major role in this chronic degenerative condition, a protocol including *Shodhana* and *Rasayana* will be effective in managing the symptoms and improving the quality of life.

Etiology

Smoking is the major risk factor for the development of COPD and the prevalence increases as per the chronicity and index. The genetic factor determined for the occurrence of this disease is deficiency for alpha-1-antitrypsin. Chronic cough, sputum production and dyspnoea are the main symptoms of COPD in a patient exposed to risk factors. Clinical diagnosis is confirmed by standardised spirometric tests.

There are two main forms of COPD. Most people with COPD have a combination of both conditions.

Chronic Bronchitis

Chronic bronchitis is characterized by presence of cough and sputum production for at least 3 months in each of two consecutive years. [8] The excessive mucus production causes the bronchi to be narrower than normal creating airway obstruction.

Emphysema

Emphysema is destruction of the gas-exchanging surfaces of the lung (alveoli). As the work needed for expiration is more the lungs get hyper inflated due to residual air which causes dyspnea.

Clinical Features

Cough, shortness of breath and sputum production are the major clinical features of COPD. The symptoms may worsen sometimes according to the climate and diet which need sudden hospitalization and therapeutic intervention, termed as exacerbation. Fatigue, weight loss, muscle mass loss, and anorexia are common problems in patients with severe and very severe COPD. Advanced COPD leads to high pressure on the lung arteries, which strains the right ventricle of the heart leading to right ventricular failure. This situation is referred to as cor pulmonale, and leads to symptoms of ankle swelling and bulging neck veins. COPD often occurs along with a number of other conditions, which include ischemic heart disease, high blood pressure, diabetes mellitus, muscle wasting, osteoporosis, lung cancer, anxiety disorder and depression. [9]

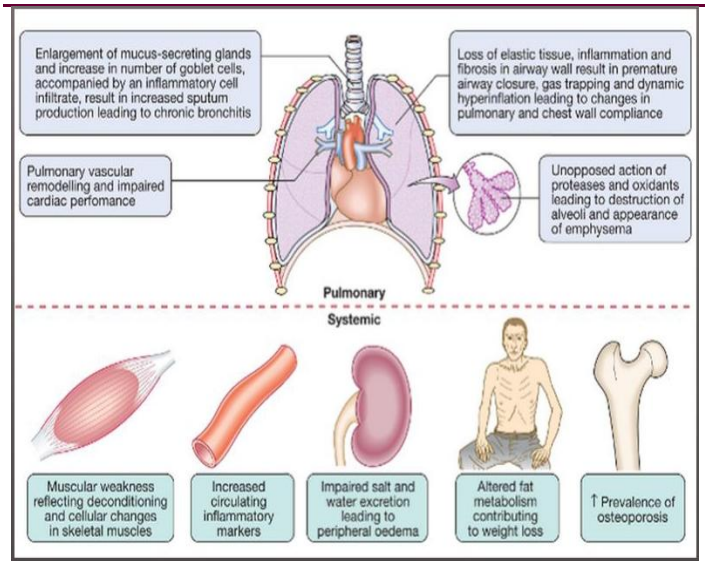

Fig 1: Pulmonary and systemic changes in COPD[10]

Pathology

- ❖ Affects larger airways, alveoli and smaller airways.

  Changes in large airways– Mucous gland enlargement, Goblet cell hyperplasia and squamous metaplasia of the bronchi which disrupts the mucociliary clearance.
- ❖ In smaller airways (<2mm in dia.) - Goblet cell metaplasia, replacement of surfactant releasing clara cells by mucous secreting and infiltrating mononuclear inflammatory cells. Smooth muscle hypertrophy occurs. Excess mucus, edema and infiltration cause luminal narrowing. Reduction of surfactant causes increased surface tension at air tissue interface – airway narrowing and collapse. Loss of elastic recoil causes high resistance to airflow in small airways.
- ❖ Lung parenchyma changes - Destruction of gas exchanging airspaces (respiratory bronchioles, alveoli and alveolar ducts). Macrophages accumulate in respiratory bronchioles of smokers. Broncho alveolar lavage fluid of smokers contains >95% macrophages (5 times as of non-smokers), neutrophils (1-2%) and T-lymphocytes (CD8+ Tcells).
- ❖ 4 events in the pathology of emphysema
  1. Inflammatory cell recruitment in terminal airspace of lung.
  2. Release of elastolytic proteinases.
  3. Damage to extra-cellular matrix of the lung.
  4. Structural cell apoptosis causes airspace enlargement and pulmonary emphysema.

  ❑ Cigarette smoke causes loss of cilia in airway epithelium which in turn leads to mucuous accumulation and bacterial infection with neutrophilia. Smoking leads to increase in collagen content of lung and airspace enlargement with loss of alveolar units. It decreases the ability of adult lung to repair damaged alveoli and functional elastic fibres remain limited.

Pathophysiology

- Persistent decrease in FEFR
- Increased residual volume(RV)
- Increase RV/TLC ratio
- Ventilation- Perfusion mismatching
- Airflow obstruction - Decrease in FEV1 and decrease in FEV1/FVC. If FEV1<25% of predicted, chronic hypoxemia results which causes pulmonary hypertension leading to cor pulmonale and RVF. Nitrogen wash out during breathing is delayed due to poor ventilation.

Systemic examination - Respiratory System

- **Inspection** reveals tachypnoea, use of accessory muscles for breathing, discolouration of nails and fingers (cigarette smoking), Raised JVP in complication of COPD (cor pulmonale) and clubbing. Shape of chest shows barrel shaped (hyper inflation) in which AP diameter > transverse diameter. Chest movements move as a whole (en bloc). Intercostal recession occurs as the result of hyperinflation. Chest expansion will be bilaterally diminished. Characteristic appearance with purse-lip breathing and tripod posture.
- **On Palpation** there will be widening of intercostal spaces on both sides. Tactile vocal fremitus will be decreased and there may be palpable wheeze or rhonchi. Apex beat may be difficult to localise. In advance COPD there will be inward movement of abdomen during inspiration indicating diaphragmatic paralysis.
- **On Percussion**, note will be hyper resonant in emphysema. Resonant note below right 5th rib is indicative of hyper inflation.
- **Auscultation** reveals diminished intensity of breath sounds due to decreased air flow and poor transmission of sounds. Vesicular breath sounds with prolonged expiration is characteristic of COPD. Vocal resonance will be decreased. Added sounds like wheezes, rhonchi, crackles may be present. Widespread polyphonic wheezes are the commonest type particularly during expiration.

Prognosis

COPD progresses and contributes to high mortality and morbidity. Estimates show that around 3% of the disability is contributed by this disease. [11] Home oxygen support has contributed to reduction in morbidity and improved quality of life. BODE Index comprising of BMI, obstruction by spirometry, dyspnea grading and exercise test by 6

mt walking is the scoring used for predicting the prognosis of COPD patients.

Management

Once the diagnosis of COPD has been confirmed by spirometry, the following four fundamental aspects guide the selection of therapy:

- Severity of airflow limitation
- Nature and magnitude of current symptoms
- Previous history of moderate and severe exacerbations.
- Comorbidities

Presence of the symptoms and the history of exacerbations decide the management protocol in stable COPD patients.

- Smoking cessation - All individuals who smoke should be strongly encouraged and supported to quit.
- Vaccination - Include influenza vaccination once a year, pneumococcal vaccination once every 5 years.

Management strategies include pharmacologic and non-pharmacologic interventions. [12]

Reducing the symptoms, frequency, severity of exacerbations and thereby improving the exercise tolerance and health status is the aim of pharmacological therapy.

- Bronchodilators cause dilation of the airways and improve spirometric variables. They act by altering airway smooth muscle tone and the improvements in expiratory flow which reflect widening of the airways rather than changes in lung elastic recoil. [13]
- Beta2-agonists - Relaxes airway smooth muscle. Stimulation of beta2-adrenergic receptors increases cyclic AMP and acts antagonistic to bronchoconstriction. There are short-acting (SABA) and long-acting (LABA) beta2-agonists. [14]
- Antimuscarinic drugs block the bronchoconstrictor effects of acetylcholine on M3 muscarinic receptors expressed in airway smooth muscle. [15]
- Methylxanthines may act as non-selective phosphodiesterase inhibitor. Eg-Theophylline.
- Inhaled corticosteroids (ICS) – Advised to use in higher blood eosinophil count. [16]
- Triple therapy (LABA+LAMA+ICS)
- Oral glucocorticoids play a role in the acute management of exacerbations[17]
- Phosphodiesterase-4 (PDE4) inhibitors- The principal action of PDE4 inhibitors is to reduce inflammation by inhibiting the breakdown of intracellular cyclic AMP. [18]
- Anti-inflammatory drugs, antibiotics, anti-oxidants and mucolytics are other groups.

Non-pharmacological Therapy

- Smoking cessation
- Pulmonary rehabilitation (PR) – Comprises of exercise, disease management and counselling to improve the overall quality of life. [19]
- Long term oxygen therapy (LTOT).
- Non-invasive positive pressure ventilation (NPPV).
- Lung transplantation and lung volume reduction surgery (LVRS)

Ayurvedic view of COPD

Nidana panchaka

Nidana[20]

- *Rajodhuma anila seva*
- *Guru, Abhishyandi bhojana*
- *Seetaambu pana, Snana*
- *Visha vata*
- *Apatarpana*
- *Beejadushti*
- *Kapha kopa kala (Poorvahna)*

Poorva rupa[21]

- *Gurutwa of Kanda & Uras*
- *Hrit peeda*
- *Parswasoola*

Roopa[22]

- *Uro alparuk*
- *Hridayam sthimitam, guru*
- *Kandopalepa*
- *Sadanam*
- *Ghana snigdha swetha sleshma pravrithi*
- *Hrit parswa sula*
- *Shteevanam*
- *Niswasochwasa samgraha*
- *Anaha*

Upasaya[23]

- *Kaphavata hara*
- *Usna*
- *Vatanulomana*

Samprapthi

Nidanas mentioned above causes *Kapha kopa* which leads to obstruction of airway *(Pranavaha srothorodha)* further leading to *Vata kopa (Prana, Udana)* and getting localised *in Uras* and *Kanta* manifesting as *Kasa* and *Svasa.*

Samprapthi ghatakas

- *Dosha: Kapha, Vata*
- *Dhathu: Rasa, Medas, Majja*
- *Agni: Mandam/Vishamam*

- *Srothas: Pranavaha (Athi badha, Kupitha, Alpa uchwasa, Sasabdhsa sula uchwasa)*
- *Annavaha (Anannabhilasha, Arochaka)*
- *Udakavaha (Sushka talu, Kanta)*
- *Rogamarga: Abhyanthara*
- *Sadhyasadyata: Yapyam-asadhyam*

Table 1: Ayurvedic correlation of COPD [24]

Chronic bronchitis	**Features of *vataja & kaphaja kasa***
1) Dry cough/ productive cough	1) *Sushka kasa/Sakapha kasa*
2) Thick white sputum/ little sputum with great difficulty	2) *Ghana snigdha svetha sleshma pravrithi / Krichrat muktva alpatam vrajet*
3) Loss of appetite, anorexia	3) *Agnimandya, Aruchi, Angasada*
4) Chest discomfort, fatigue	4) *Uro alpa rujatva, Hrudaya stimita*
5) Rhinitis	5) *Peenasa*
Emphysema	**Features of *Tamaka svasa***
1) Shortness of breath, wheezing	1) *Rudha swasa, Ghurghurakam*
2) Cough	2) *Muhur muhur kasa, Kasavegat pratamyati*
3)Disturbed sleep	3) *Sayananthe swasadikam/Asino labate soukhyam*
4) Increased complaints on cold, dust & smoke	4) *Meghambu seeta pragvathe sleshmalaischa vivardhate*
5) some relief after spitting sputum	5) *Nishtutyanthe kshanam sukhi*

Sadhyasadhyata

Though *Tamaka shvasa* is a palliable disorder, in the initial stage of the disease and in patients of good strength it is expected to be cured completely. With the prolongation of the illness the patient usually develops *Dhatu–kshaya* which is either be generalised or localised in *Pranavaha srotas*, therefore the prognosis in very chronic cases is unfavorable. [25]

Treatment schedule *(Cikitsa Sutra)*

Yukti plays pivotal role in treatment of any disease. *Yukti* means use of drug and its form after consideration of *Dosha, Dushya, Anala* and *Prakṛti*. While narrating the treatment portion of *Shvasa*, the ancient scholars classified it depending on dominancy of *Dosha* and physical state of patient.

a) *Dosha* dominance 1) *Vataja* 2) *Kaphaja*

b) Physical state of patient 1) *Balavan* 2) *Durbala*

The treatment of *Shvasa* can be explained in three groups as given below:

Samshodhana

Vamana and *Virecana* must be performed in patient having dominancy of *Kapha dosha* and well–built physic.

Samshamana

Patients having *Vatika* dominancy and weak in built are not apt for *Samshodhana* therapy. *Samshamana* therapy plays very crucial role in *Vataja* and *Anutklishta kaphaja* dominant *Shvasa* in weak, old and children. As such, *Samshamana* therapy is a broad spectrum therapy which includes *Ushna*, *Kapha-vataghna, Vatanulomaka* drugs and food. More importance is given to *Vatakapha hara* therapy rather than singular treatment of *Vata* and *Kapha*. *Shamana* and *Brimhana* therapy should be applied in *Ashuddha* and *Shuddhavastha* respectively.[26] *Acarya Vagbhata* emphasizes the use of *Kashaya, Leha, Sneha* in the *Samshamana* therapy. *Acarya Caraka* elucidates the use of *Sneha* in *Vata* dominancy, *Avaleha* in *Kaphaja* and *Arishta* in the *Vegavasta*. [27]

Nidana Parivarjana

Tamaka svasa, episodic in nature, occurs after exposure to certain factors like *Raja, Dhuma*, which are termed as aggravating or predisposing factor. Avoidance of such factors is helpful to get rid of attacks of *Shvasa* or else they may produce more *Vigunata* in *Pranavaha srotas* through the provocation of *Doshas*. *Snehana* (oleation), *Svedana* (sudation), *Vamana* (emesis), *Virecana* (purgation), *Kaphahara* and *Vatanulomana* (that which not vitiates *Vata* and *Kapha*) is the mentioned treatment for *Shvasa*. For *Lina dosha, Dhuma* is done. *Sveda* is to be avoided in *Pitta* conditions and *Ruksha sveda* can be done in *Ama* stage. *Vamana* with *Lavanambu* is also advised. [28]

DISCUSSION

COPD is a *Kapha vata* predominant disease. In the *Samprapthi ghatakas* of *Kasa* and *Svasa*, the main *Doshas* involved are *Vata* and *Kapha*. *Agni mandya, Agnijanya ama* and *Pranavaha srotorodha* are having major role in the pathogenesis of the disease. *Nidanas* cause *Dosha kopa* and *Agni mandya* which further leads to *Ama utpathi*. The food not getting digested

properly by the poor digestive fire in the alimentary tract gives rise to more of wastes and less for the nourishment of *Dhatus*. The *Sroto mukhas* are coated and obstructed, especially the vitiated *Kapha* obstructs *Pranavaha srothas* and cause *Pranavaha srothodushti.* There is *Sanga* and *Vimarga gamana* for *Pranavata* which attains *Udanabhava* that produces *Kasa* and *Svasa*. The *Dhatvagni* being poor and the *Rasa* undergoes improper cooking and remaining in its own place produces the various complications. It won't lead to the formation of further *Dhatus*, resulting in *Kshaya roga*. If not properly managed, *Kasa* progresses to *Svasa* and finally to *Kshaya*.

COPD is also progressive in nature with chronic bronchitis if not managed properly leading to emphysema. Above said *Ama* and vitiated *Kapha* leads to hypersecretion, bronchoconstriction and narrowing of airways which manifests in the form of productive cough and shortness of breath. In later stages the fibrosis and destruction of alveoli can be taken as *Vatakopa lakshanas*.

CONCLUSION

COPD – Ayurvedic Concept

We have seen that COPD is having two conditions chronic bronchitis and emphysema. We can see both these conditions together in latter stages in a single patient, i.e., to begin with mostly in bronchitis and leading to emphysema. COPD cannot be directly equated to any Ayurvedic conditions, but considering the similarities in etiology and clinical features, *Pranavaha srotodushti vikaras* like *Kasa, Shvasa, Urakshata, Rajayakshma* etc can be considered. It is said that *'Kasa vridyat bhavet shvasa*!' So we can consider chronic bronchitis as *Kasa* and emphysema as *Shvasa*. Among the types of *Kasa* and *Shvasa*, by analyzing the symptoms, we can come to a conclusion that *Vata–Kaphaja kasa* and *Tamaka shvasa* can be better correlated with chronic bronchitis and emphysema respectively. COPD is considered as *Tamaka shvasa* in this review as dyspnoea is the major presenting symptom in it along with *Kasa*.

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