A Dissertation on

HAEMATOLOGICAL MANIFESTATIONS IN CHRONIC OBSTRUCTIVE LUNG DISEASE IN RELATION TO FEV1

COIMBATORE MEDICAL COLLEGE HOSPITAL

Dissertation Submitted to

THE TAMILNADU Dr.M.G.R. MEDICAL UNIVERSITY

CHENNAI - 600 032

With partial fulfillment of the regulations
for the award of the degree of

M.D. GENERAL MEDICINE

BRANCH-I

COIMBATORE MEDICAL COLLEGE, COIMBATORE

APRIL 2015

CERTIFICATE

Certified that this is the bonafide dissertation in "**HAEMATOLOGICAL MANIFESTATIONS IN CHRONIC OBSTRUCTIVE LUNG DISEASE IN RELATION TO FEV1"** done by **Dr. GOWRI SANKAR. M** and submitted in partial fulfillment of the requirements for the Degree of **M.D., General Medicine**, Branch I of **The Tamilnadu Dr. M.G.R. Medical University, Chennai.**

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DECLARATION

I solemnly declare that the dissertation titled **"HAEMATOLOGICAL MANIFESTATIONS IN CHRONIC OBSTRUCTIVE LUNG DISEASE IN RELATION TO FEV1"** was done by me from AUGUST 2013 to JULY 2014 under the guidance and supervision of Professor **Dr. S.USHA .M.D.** This dissertation is submitted to **The Tamilnadu Dr.M.G.R. Medical University towards** the partial fulfillment of the requirement for the award of MD Degree in **General** Medicine (Branch I).

Place: Coimbatore **Dr. GOWRI SANKAR.M**

Date:

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LIST OF ABBREVIATIONS USED

1. COPD	-	Chronic Obstructive Pulmonary Disease
2. HB	–	Haemoglobin
3. MCV	-	Mean Corpuscular Volume
4. MCH	-	Mean Corpuscular Haemoglobin
5. MCHC	-	Mean Corpuscular Haemoglobin Concentration
6. SABA	-	Short-Acting Beta-Agonist
7. SAMA	-	Short Acting Antimuscarninic Agent
8. LAMA	-	Long-Acting Antimuscarninic Agent
9. LABA	-	Long-Acting Beta-Agonist
10.ICA	-	Inhaled Corticosteroids
11. FEV1	-	Forced Expiratory Volume in One Second
12. FVC	-	Forced Vital Capacity
13. WHO	–	World Health Organisation

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ABSTRACT

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a complex disease characterized by airflow limitation resulting from airway inflammation, parenchymal destruction and the development of emphysema. COPD results from inflammatory mechanisms, the **"SPILL-OVER"** of inflammatory mediators like IL6, IL-8,

TNF-∝ leads to systemic inflammation and also initiates or worsens comorbid diseases. About 15-30% of COPD patients have anemia and this is noticed especially in patients with severe disease, and the occurrence of polycythaemia is only 6%. Anemia is related to depressed functional capacity and for low quality and standard of life.

METHODOLOGY

This clinical observational study was undertaken to investigate the pattern and magnitude of Hematological parameters in the relationship with the severity of the disease by Forced Expiratory Volume1 (FEV1). 60 Patients were included in this study.

All the Patients were subjected to detailed History and Physical examination. Lung function parameters were assessed with the help of spirometer. All patients underwent Haematological parameters with particular reference to HB%, MCV,

MCH, MCHC, HAEMATOCRIT, and PERIPHERAL SMEAR along with routine tests.

RESULTS AND DISCUSSION

In our study, 56% of patients had anemia, among them 70% had Normocytic Normochromic Anemia and 30% had Microcytic Hypochromic Anemia. Patients with duration of illness 6-10 yrs had 57% risk for anemia and > 10 yrs had 87% risk for anemia. Patients with history of exposure to Cotton mill dust had 71% risk for anemia.

CONCLUSION

Patients with decreased FEV1, having prolonged duration of illness and increasing grades of dyspnea had high risk for anemia. Patients with exposure to Cotton Mill dust and with the history of smoking had increased risk for anemia, but its association was not statistically significant.

KEY WORDS

- COPD
- ANEMIA
- FEV1

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a complex disease characterized by airflow limitation resulting from airway inflammation, parenchymal destruction and the development of emphysema. COPD results from inflammatory mechanisms, the **"SPILL-OVER"** of inflammatory mediators like IL6, IL-8, and TNF-∝ leads to systemic inflammation and also initiates or worsens comorbid diseases. So the comorbities complicate the management of COPD and need to be evaluated carefully[1]. Diseases with higher morbidity in COPD are associated with an increased risk of hospital admissions,mortality and healthcare costs[2-3]. According to World Health Organisation, in 2020 COPD will become the 3 rd leading cause of death. Meta-analysis study in India suggests that the prevalence of COPD above 30 yrs in males is 5% and in females is 2.7%.

According to a study, about 15-30% of COPD patients have anemia and this is noticed especially in patients with severe disease, and the occurrence of polycythaemia is only 6% [4-6] . Anaemia is related to depressed exercise capacity along with functional dyspnea.This is a significant reason for depressed functional capacity and for low quality and standard of life.[7-9]

Forced Expiratory Volume in one second (FEV1) is an objective measurement,viewed as the most accurate predictor of severity of airway obstruction. The advantage of FEV1 is that it requires lesser effort to measure and can be carried out in all stages of COPD patients .COPD progression can be assessed by serial measurements of FEV1. In this dissertation an effort is made to assess the hematological manifestations in COPD Patients and the relationship to FEV1.

OBJECTIVES OF THE STUDY

To study the Haematological manifestations in relation to FEV1 in 60 cases of Chronic Obstructive Pulmonary Disease.

REVIEW OF LITERATURE

HISTORY OF COPD

- Empysema goes for description with hierarchial reference as follows: "Voluminous lungs" was widely explained by **Bone**t in 1679;
- **Morgagni's** (1769) showed the lungs were "turgid", particularly from air.

FIG 1: TEOFILO BONET (1620 -1689)

FIG 2: GIAMBATISA MORGAGNI (1682-1771)

Baille's descriptions about the emphysematous lung, was exactly similar to Samuel Johnson's one.

The emergence of our clinical apprehension of the chronic bronchitis rose from **Badham** (1814), who attributed chronic cough and mucus hypersecretion jointly to the word "catarrh".[10]

Percussion probably got developed as a way of ascertaining how much fluid remained in barrels of wine by Austrian physician **Josef Leopold Auenbrugger** who applied percussion of chest, having learned this method in his father's wine cellar.[11]

FIG 3: Josef Leopold Auenbrugger (1722-1809)

Laennec, the inventor of Stethescope ,in 1821, explained exhaustively about Emphysema in his "**Treatise of Diseases of the Chest**"

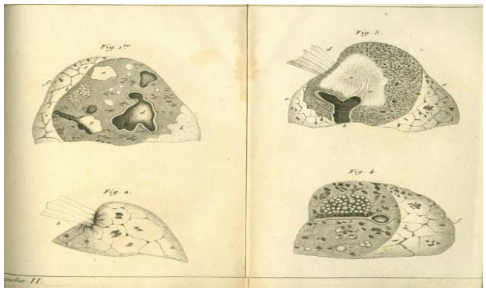


FIG 4: "Treatise of Diseases of the Chest".

In 1846, **John Hutchinson** (1811-1861) invented spirometer and he described the values of vital capacity in measurement.

FIG 5 : SHADOW IMAGE OF JOHN HUTCHINSON AND HIS SPIROMETRY

- **Tiffeneau** ten decades later, mentioned Timed Vital Capacity as a parameter in Spirometry.
- **Gaensler** introduced the concept of Forced Vital Capacity ,which forms the foundation for FEV1 and FEV1/FVC.[10]
- In 1916, Osler's **Principles and Practices of Medicine** describes "EMPHYSEMA"
- In 1956, **Barach and Bickerman** presented the first comprehensive text book on "**Pulmonary Emphysema"**.

DEVELOPMENT OF THE RESPIRATORY SYSTEM[12-17]

The human developmental phases between fertilization and birth are generally divided into

Embryonic Period **Fetal period**

The **embryonic period** occurs during the first 8 weeks and is traditionally organized into 23 stages. During the embryonic period, all major organs begin their development.

The **fetal period** occurs during the remaining32 weeks of gestation. During this period, the organs continue to develop and refine their structure and function.

There are three embryologically germinal tissue layers that eventually forms all tissues and organ.

- **ENDODERM**
- **MESODERM**
- **ECTODERM**

During the development, lung bud forms the trachea and two lateral out pocketings, the bronchial buds. At the beginning of the fifth week, each of these buds enlarges to form right and left main bronchi. The right then forms three secondary bronchi, and the left forms two secondary bronchi.

The mesoderm, which covers the outside of the lungs, develops into the visceral pleura. The somatic mesoderm layer, covering the body wall from the inside, becomes the parietal pleura. The space between the parietal and visceral pleura is pleural cavity.

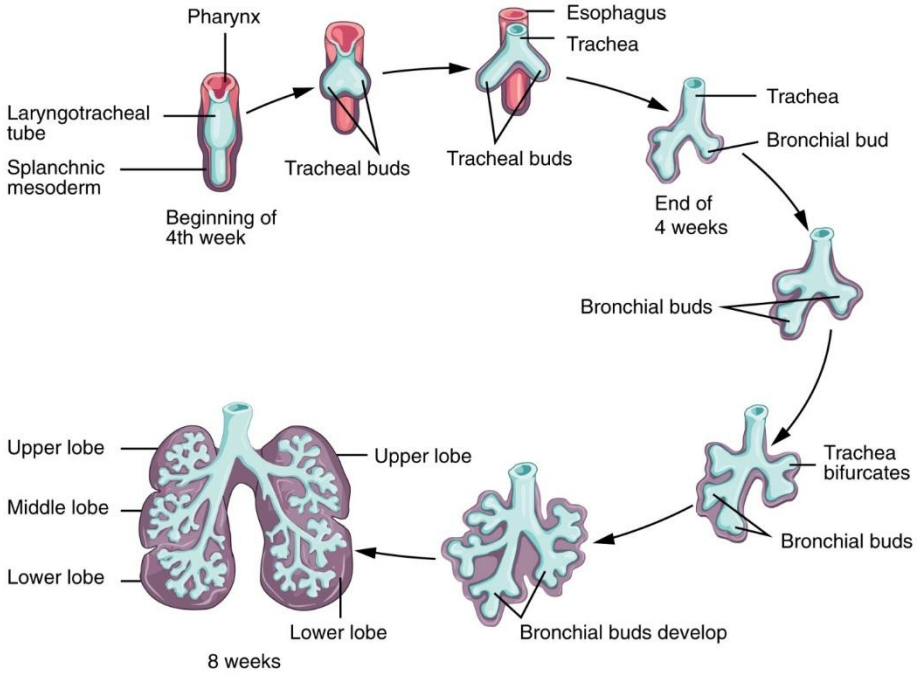


FIG 6: DEVELOPMENT OF THE RESPIRATORY SYSTEM

During further development, secondary bronchi divide repeatedly in a dichotomous fashion, forming ten tertiary (segmental) bronchi in the right lung and eight in the left, creating the bronchopulmonary segments of the adult lung. By the end of the sixth month, approximately 17 generations of subdivisions have formed. Before the bronchial tree reaches its final shape, however, an additional six division's form during postnatal life.

MATURATION OF THE LUNGS

Up to the seventh prenatal month, the bronchioles divide continuously into more and smaller canals, and the vascular supply increases steadily. Respiration becomes possible when some of the cells of the cuboidal respiratory bronchioles change into thin, flat cells. These cells are intimately associated with numerous blood and lymph capillaries, and the surrounding spaces are known as terminal sacs or primitive alveoli. During the seventh month, sufficient numbers of capillaries are present to

guarantee adequate gas exchange, and the premature infant is able to survive.

During the last 2 months of prenatal life and for several years thereafter, the number of terminal sacs increases steadily. Mature alveoli are not present before birth. In addition to endothelial cells and flat alveolar epithelial cells, another cell type develops at the end of the sixth month. These cells, type II alveolar epithelial cells, produces surfactant, a phospholipids-rich fluid capable of lowering surface tension at the air-alveolar interface.

Before birth, the lungs are full of fluid that contains a high chloride concentration, little protein, some mucus from the bronchial glands, and surfactant from the alveolar epithelial cells. The amount of surfactant in the fluid increases, particularly during the last 2 weeks before birth.As concentration of surfactant increase during the 34th week of gestation.

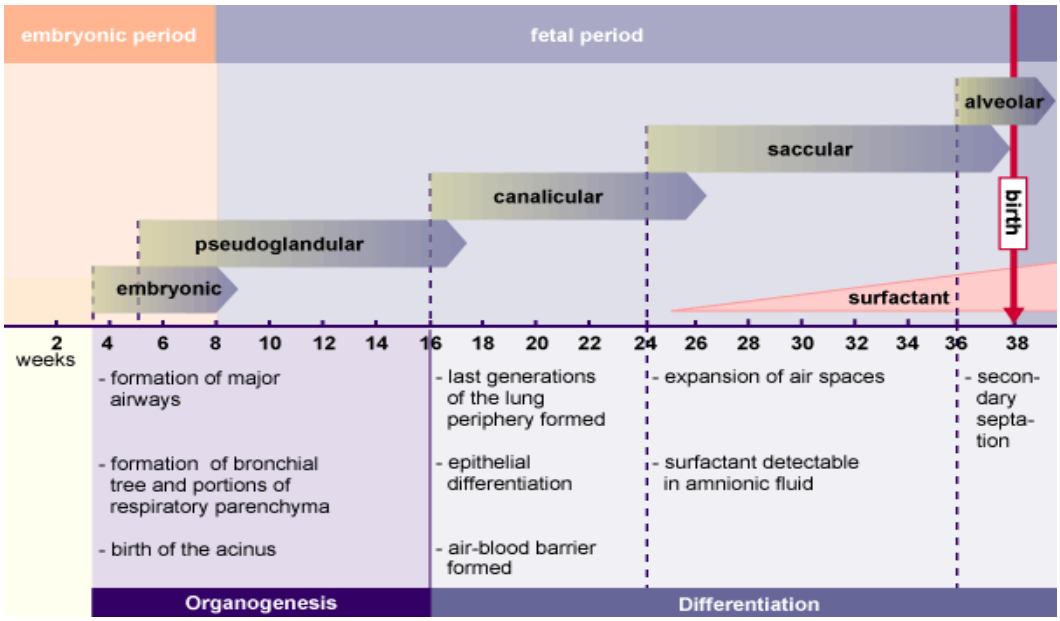


FIG 7: MAJOR PHASES OF RESPIRATORY DEVELOPMENT

Throughout the developmental period, lung growth is similar in male and female fetuses. A full-term newborn has about 50 million alveoli, and the number continues to elevation for about 2 to 3 years after birth.

The developmental branching process of the airways and blood vessels of the lung is highly regulated by the timely activation various genes in different locations. Of the approximate 22,000 genes in the human genome, about 40 are required for normal respiratory development.

ANATOMY[18]

UPPER RESPIRATORY TRACT

The Upper respiratory tract includes nose, pharynx, paranasal sinuses and larynx. The nose is lined with ciliated epithelium helps in clearance of particles and microorganisms.

Functions of the upper airway:

- Passageway for gas flow
- Filter
- Heater
- Humidification
- Phonation
- Protection of airways
- Sense of smell and taste

LOWER RESPIRATORY TRACT

Trachea

It extends down from cricoid cartilage and bifurcates in the superior mediastinum at the level of angle of Louis. The adult trachea is approximately 12cm long and has inner diameter of 2cm. It is lined by pseudo stratified ciliated columnar epithelium containing goblet cells. The adult has 16 to 20 cartilaginous ring.The cartilaginous rings armor the trachea so that it does not collapse during exhalation.

BRONCHI AND THEIR DIVISIONS

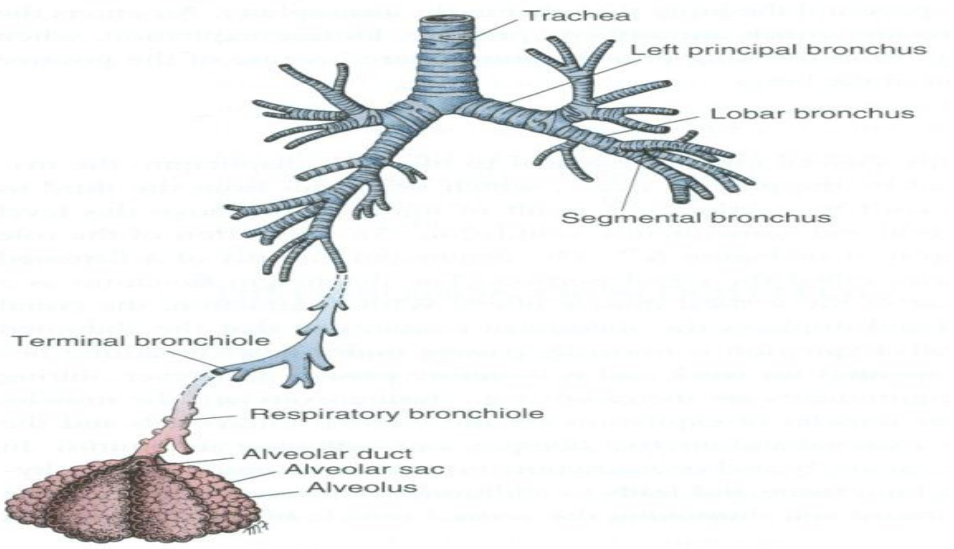
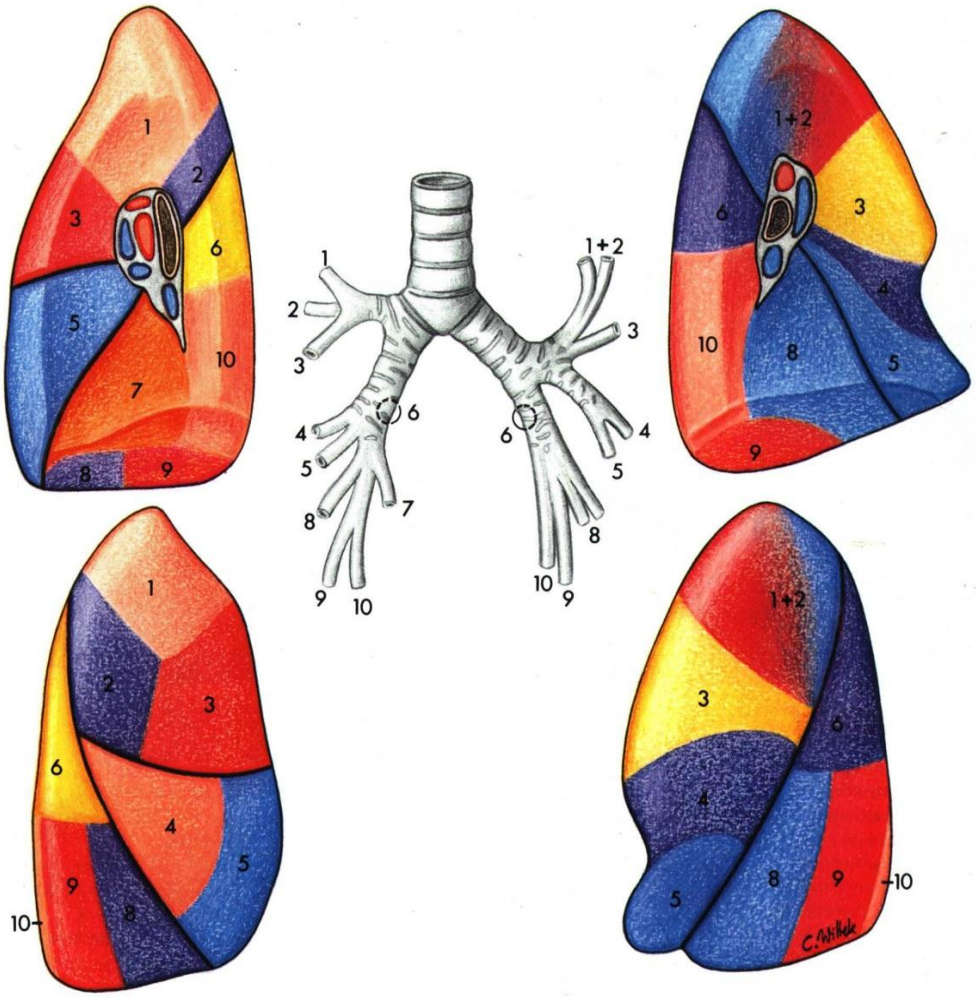


FIG 8: BRONCHI AND THEIR DIVISIONS

At the base of the trachea, Carina is a landmark where the trachea bifurgates. The left bronchus branches with an angle of about 45 to 55 degrees and runs more horizontally and is about 5 cm long. The right main bronchus branches with an angle of about 20 to 30 degrees and it is only about 1 to 2.5 cm and is a more direct continuation of trachea.

BRONCHOPULMONARY SEGMENTS



Distribution of bronchopulmonary segments of the lungs and their relation to the bronchial tree (after J. F. Huber).
The bronchopulmonary segments are morphologically and functionally separate independent respiratory units of the lung tissue. Each segment is surrounded by connective tissue which is continuous with the visceral pleura. The segmental bronchi in a segment are central, closely accompanied by branches of the pulmonary arteries whereas the tributaries of the pulmonary veins run **between** the segments. Thus, the veins serve two adjacent segments which drain for the most part into more than one vein. A bronchopulmonary segment is therefore not a complete vascular unit, but segmentation is the result of a specific architecture of the lung vasculature.

Right lung		**Left lung**	
1 Apical segment 2 Posterior segment 3 Anterior segment	Upper lobe bronchus	1+2 Apico-posterior segment 3 Anterior segment	Superior division → Upper lobe bronchus
4 Lateral segment 5 Medial segment	Middle lobe bronchus	4 Superior lingular segment 5 Inferior lingular segment	Inferior division → Upper lobe bronchus
6 Superior (apical) segment 7 Medial basal segment 8 Anterior basal segment 9 Lateral basal segment 10 Posterior basal segment	Lower lobe bronchus	6 Superior (apical) segment 7 Absent 8 Anteromedial basal segment 9 Lateral basal segment 10 Posterior basal segment	Lower lobe bronchus

FIG 9: BRONCHOPULMONARY SEGMENTS

INTRA PULMONARY AIRWAYS are divided in to three major groups

1) BRONCHI-has cartilage and serve as a airway

2) MEMBRANOUS BRONCHIOLES- non-cartilaginous airways

3) RESPIRATORY BRONCHIOLES-serve as a airways and for gas exchange

There are about 28 generations of Tracheobronchial tree which serve as a Conducting zone and Respiratory zone

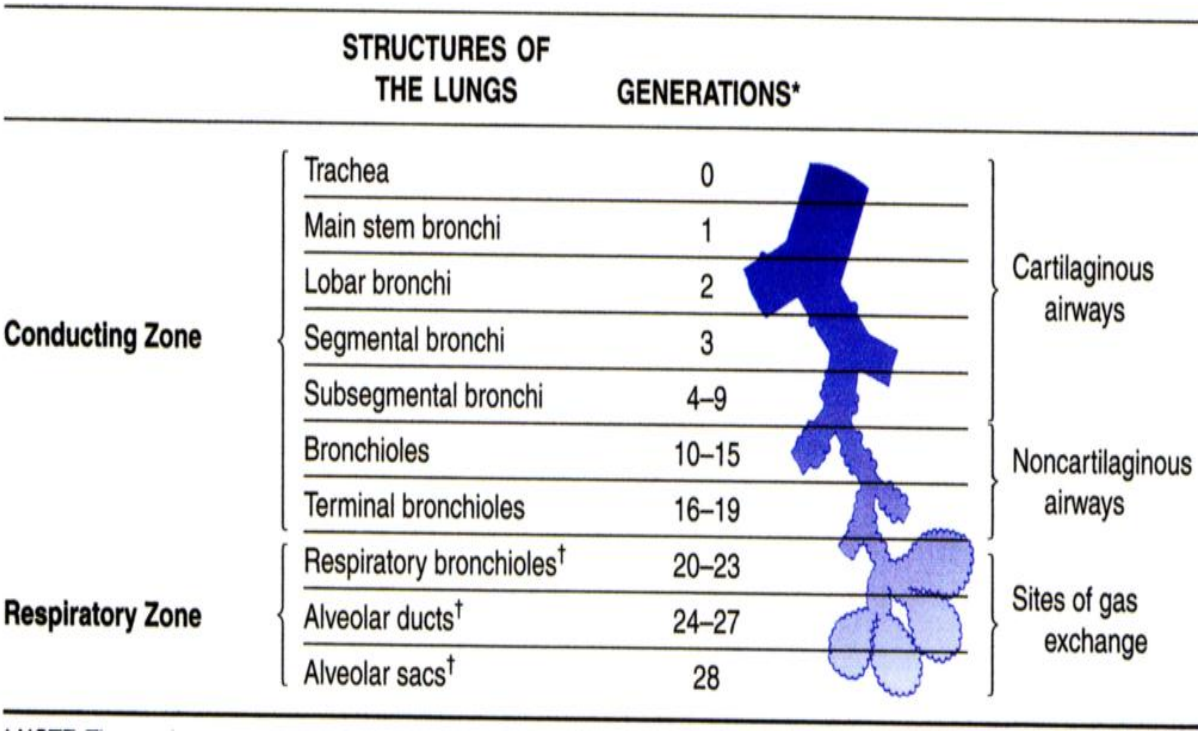


FIG 10: STRUCTURES OF THE LUNGS

ALVEOLI

There are about two hundred to six hundred million alveolus, which is called as the terminal respiratory unit.

There are two types of epithelial cells lining the alveoli.

- Type 1 cells are flat squamous epithelial cells and are the primary lining cells.
- Type II cells Granular Pneumocytes are rounded secretary cells and contain numerous lamellar inclusion bodies. These cells secrete surfactant.

The Bronchi and their Innervations

The trachea and bronchi have cartilage in their walls, but relatively little smooth muscle and it is lined by a ciliated epithelium. It has both mucus gland and serous glands. Ciliated structure is present up to respiratory bronchioles, but glands are absent from the epithelium of the bronchioles and terminal bronchioles and their walls do not contain cartilage. However, their walls contain more smooth muscle and they are innervated by the autonomic nervous system. There are abundant muscarinic receptors and cholinergic discharge causes bronchoconstriction. There are β1 and β2 adrenergic receptors in the bronchial epithelium and smooth muscle and in mast cells. Many are not innervated. Some may be located on cholinergic endings and ganglia, where they inhibit acetylcholine release. In humans, the β2 receptors predominate and inhaled or injected β agonist such as isoproterenol cause bronchodilation and depressed bronchial secretion.

PULMONARY CIRCULATION[19]

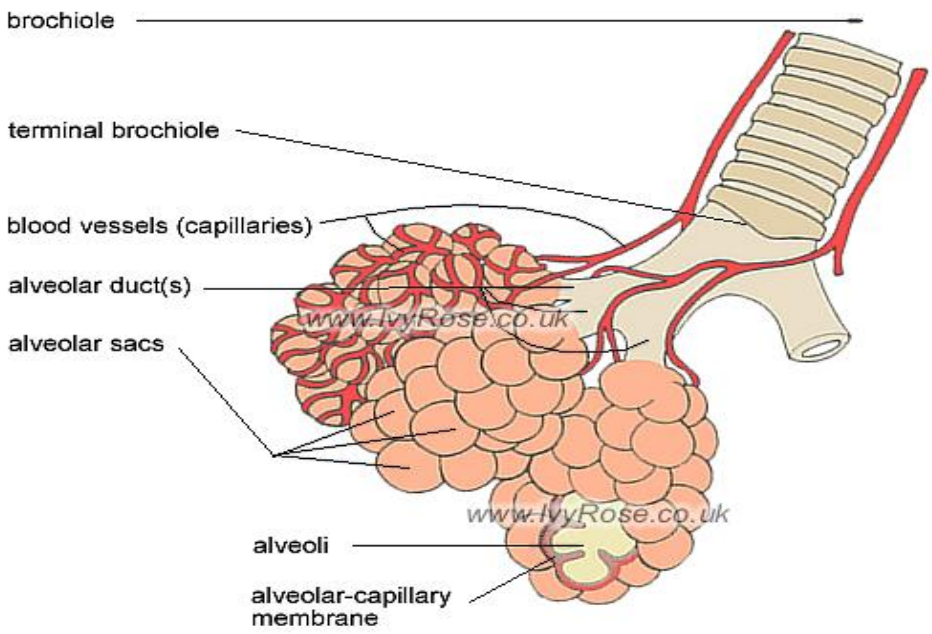


FIG 11: PULMONARY CIRCULATION

All the venous blood in the body enters

RIGHT ATRIUM

⇩

RIGHT VENTRICLE

⇩

PULMONARY ARTERY

⇩

PULMONARY CAPILLARIES –**OXYGENATION OCCURS**

⇩

PULMONARY VEIN

⇩

LEFT ATRIUM

⇩

LEFT VENTRICLE

⇩

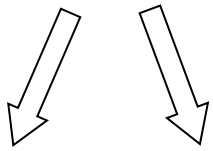
SYSTEMIC CIRCULATION

The separate and much smaller bronchial arteries come from systemic arteries. They form capillaries, which drain into bronchial vein or anastomose with pulmonary capillaries or veins. The bronchial veins drain into the azygos vein. The bronchial circulation nourishes the bronchi and pleura.

LYMPHATIC SYSTEM

Lymphatic channels are more abundant in the lungs than in any other organ.

Lymphatics of the lung is divided in to two plexus



SUPERFICIAL PLEXUS **DEEP PLEXUS**

- Superficial plexus is present in the visceral pleura
- Deep plexus is located in peri broncho vascular connective tissues
- Alveolar wall devoid of lymphatics
- Lymph is flow towards hilum from there it drains in to the extrapulmonary lymph nodes.

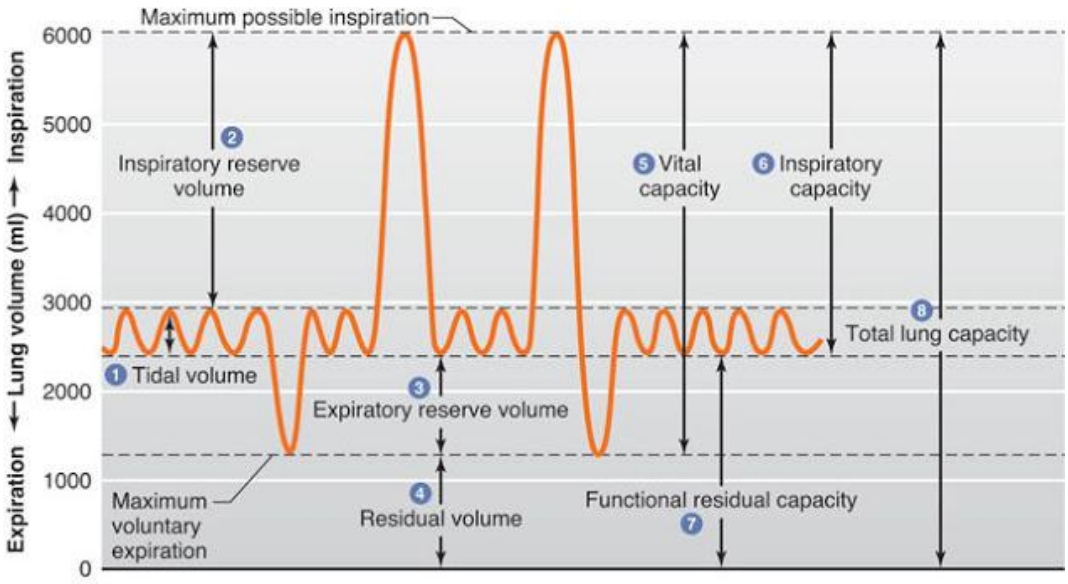


FIG 12: LUNG VOLUMES AND CAPACITIES

Lung volumes

1) **Tidal volume:** The amount of air that moves into the lungs with each inspiration (or the amount that moves out with each expiration) is called the tidal volume.(Value 500-750 ml).
2) **Inspiratory reserve volume:** The air inspired with a maximal inspiratory effort in excess of the tidal volume. (Value -2 litres).
3) **Expiratory reserve volume:** The volume expelled by an active expiratory effort after passive expiration is the expiratory reserve volume.(value -1 litre).
4) **Residual volume:** The air left in the lungs after a maximal expiratory effort (value -1.3litres).
5) **Vital capacity:** The largest amount of air that can be expired after a maximal inspiratory effort, is frequently measured clinically as an index of pulmonary function (value -3.5 litres).
6) **Inspiratory capacity:** The maximum amount of air inspired from the end-expiratory level (IRV+ TV)(Value -2.5 litres).

7) **Functional residual capacity:**The volume of the air remaining in the lungs after expiration of a normal breath (RV+ ERV).(Value - 2.5 litres).
8) **Total lung capacity:** The total lung capacity is composed of all the four components of Tidal volume, Inspiratory reserve volume, expiratory reserve volume and Residual volume (Value - 5 litres).

FEV1**, timed vital capacity:** The fraction of the vital capacity expired during the first second of a forced expiration.

Respiratory minute volume: The amount of air inspired per minute. It is normally6 litres (500ml / breath × 12 breath / min)

Maximal Voluntary Ventilation: It is the largest volume of gas that can be moved into and out of the lungs. In one minute by voluntary effort. The normal MVV is 140-180 L/min for a healthy adult male.

Three measurements are commonly made from a recording of **FORCED EXHALED VOLUME VERSUS TIME,**i.e. a spirogram.

1. **FEV1** (Forced expired volume in one second): the volume of air expired in the first second of maximal expiration after a maximal inspiration. This is a measure of how quickly the lungs can be emptied.
2. **FVC** (Forced vital capacity): maximum volume of air that can be exhaled during a forced maneuver.
3. **FEV1/FVC**: FEV1 expressed as a percentage of the FVC, gives a clinically useful index of airflow limitation.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

DEFINITION[22]

Chronic obstructive pulmonary disease (COPD) by definition goes as abnormalities in expiratory flow tests that do not alter significantly over several months of observation and this helps to distinguish COPD from asthma.

Two disorders are mentioned in COPD:

- **EMPHYSEMA**
- **CHRONIC BRONCHITIS**

American Thoracic Society (ATS) defines COPD as[23]

"A disease state characterized by the presence of airflow limitation due to chronic bronchitis are emphysema; the airflow obstruction is generally progressive, may be accompanied by airway hyperreactivity that may be partially reversible".

The Global initiative for Chronic Obstruction Lung Disease (GOLD) classified COPD as "A disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases".

The components of COPD are Chronic bronchitis and Emphysema24.

Chronic bronchitis is defined as “the presence of a chronic productive cough on most days for three months, in each of two consecutive years”.

Emphysemais defined as “abnormal, permanent enlargement of the distal air spaces, distal to the terminal bronchioles, accompanied by destruction of their walls and without obvious fibrosis”.

Cigarette smoking is the highest risk factor for COPD. The studies have shown a strong relationship between smoking and COPD, in many a high proportion of nonsmokers also develop COPD. There is an inverse relationship between cigarette consumption and expiratory flow rate. The cessation of smoking slows down the speed of pulmonary function deteriorations to that of nonsmokers, but do not normalize the lungs function.

The passive smoking, in other words, environmental tobacco smoke (ETS), or second hand smoke, is associated with COPD. Indoor air pollution due to domestic cooking and heating has been associated with chronic respiratory symptoms as reported from many developed and developing countries.

Occupational exposures to dusts alone or to dusts and in association with fumes and vapors are the risk factors for COPD.

The most important factors were age, daily cigarette consumption, possible alterations of smoking habits and the observed value of FEV1.

Smoking results in an over production of proteases (i.e. elastase). Further, it inactivates the anti-elastases. Thus there is an imbalance between the protease-antiprotease with resultant excess action of protease, which leads on to the destruction.

EPIDEMIOLOGY[25-28]

PREVALENCE: Most data available on COPD statistics are from developed countries, acquiring accurate epidemiological data on COPD is very tough and costs much.

A meta-analysis in performed between 1990 and 2004 in 28 countries showed the prevalence was 11% in men and 5% in women and 3.9% in never smokers and 15.2% in smokers.

Halbert et al. reviewed 32 prevalence studies from 17 different countries and concluded that prevalence of COPD ranged between 4 to 10%.

COPD IN INDIA[29-31]

The inference of the INDIAN STUDY INSEARCH phase II was released and this studies show that the prevalence of COPD in India was 3.7% (4.5% males and 2.9% females).

Wig (1964) et al. showed prevalence of COPD to be 3.36% and 2.54% in men and women with 2:1 smoker non smoker ratio in rural Delhi.

Viswanathan (1966) et al. showed prevalence of 2.12% and 1.33% in men and women respectively in a study in Patna.

In a pilot study in rural area of Mysore, prevalence of COPD was 7.1%, 11.1% in males and 4.5% in females.

Jindal (1993) et al. in a study in rural population of North India showed Prevalence of 6.2% and 3.9% in males and females respectively. In urban population of North India showed prevalence of 4.2% and 1.6% in males and females respectively with smoker to nonsmoker ratio of 9.6.[32]

Ray (1995) et al. in a study in South India showed prevalence of 4.08% and 2.55% in males and females respectively with smoker to nonsmoker ratio of 1.6.

Malik et al. (1986) in a study in rural population of North India showed Prevalence of 9.4% and 4.9% in males and females respectively with smoker to non smoker ratio of 5.5. In urban population of North India showed prevalence of 3.7% and 1.6% in males and females respectively with smoker to non smoker ratio of 3.7.

RISK FACTORS OF COPD

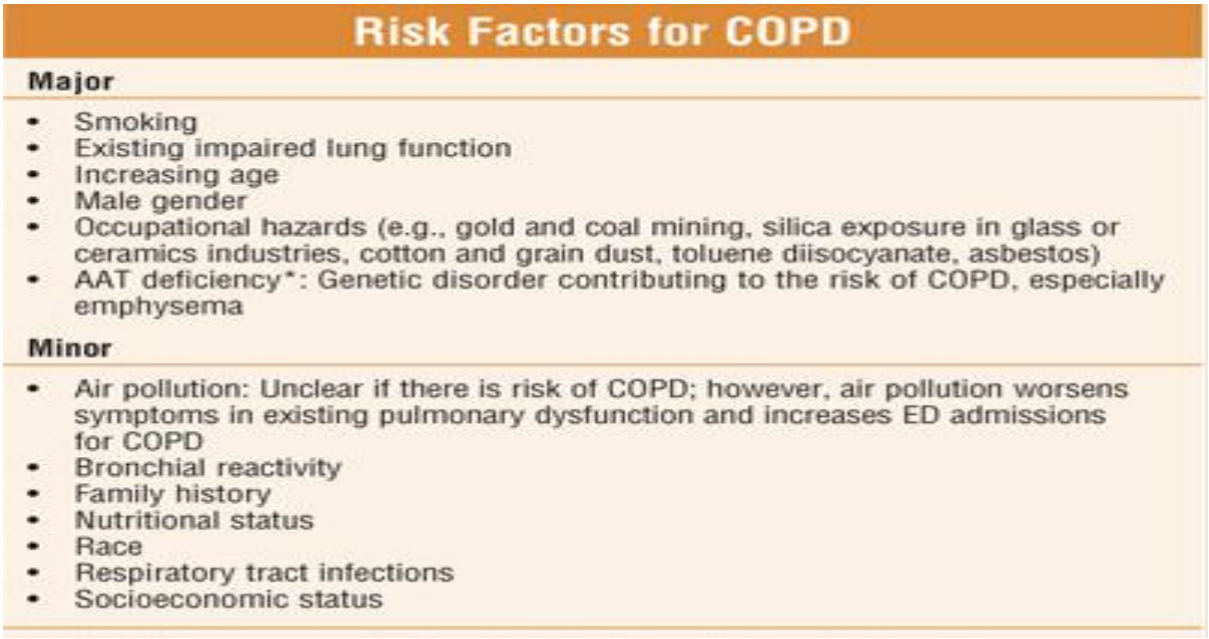


FIG 13: RISK FACTORS OF COPD

Tobacco smoking

It is a familiar fact that Tobacco smoke comprises more than 4000 toxic chemicals, including 50 known carcinogens[33]

A data showed that low-tar cigarettes have irreconcilable effect on COPD and any volume of smoking is harmful [34]

Environmental Tobacco Exposure or Passive Smoking [35]

Exposure to cigarette smoke or passive smoking is gaining more attention because of its appreciable public health effects. Environmental tobacco smoke is a combination of side-stream smoke and exhaled

mainstream smoke. Owing to a lower temperature of combustion, side stream smoke contains larger concentrations of

- Ammonia,
- Benzene,
- Carbon-monoxide,
- Nicotine,
- and various carcinogens(2-naphthylamine, 4-aminobiphenyl, n-nitrosamine, benza-anthracene, and benzopyrene) than the mainstream smoke.

In enclosed spaces, smoke accumulates, and the concentration varies with the number of smokers, with the type of smoking, and with the characteristics of the room, especially the ventilation.

Gupta D et al. in a Multicentric Population Study from India concluded that Passive smoking is associated with increased prevalence of respiratory symptoms.

OCCUPATIONAL EXPOSURE [36]

Epidemiological data by the **Matheson et al.** in their study of 1232 people concluded that occupational exposure to biological dust was associated with elevated risk of COPD which was higher in women.

Bushra Iftikhar and his colleagues in Peshawar studied relationship between silica exposure and COPD and concluded that people exposed to silica dust for more than 10 years, and daily exposure of >8 hours had more risk of developing COPD.

BIOMASS FUEL EXPOSURE [37-38]

The combustion of biomass fuel releases several toxic gases and causing respiratory problems.

Mrigendra Raj Pandey in his study in Nepal showed increased prevalence in females exposed to biomass fuel exposure and the severity of symptoms correlated with duration of exposure to smoke.

Orozco-Levi M et al in a case control study of 120 females in Barcelona, Spain showed increased risk of COPD in exposure to wood or charcoal smoke.

ALPHA-1 ANTITRYPSIN DEFICIENCY [39-40]

The only known genetic cause of emphysema is Alpha-1 antitrypsin (AAT) deficiency. An inherited deficiency of a protein in the blood called the α_1-antitrypsin (AAT). It is the only known genetic disorder that leads to COPD. AAT deficiency accounts for less than 1 percent of COPD in USA. This deficiency is an autosomal hereditary disorder in which there are low levels of α_1-antitrypsin in serum and lungs, with a high risk of development of panlobular emphysema in the third to fifth decade. There is an increased risk of development of liver disease in the childhood associated with this condition.

AAT is a glycoprotein coded for by a single gene on chromosome 14. It is a serine protease inhibitor with primary function of inhibiting neutrophil elastase. Emphysema results from an imbalance between the neutrophil elastase in the lung and the anit-elastases.

This concept is known as the "elastase-antielastase balance hypothesis of Emphysema."

Many Indian studies have tried to examine the role of alpha-1antitrypsin deficiency in the causation of COPD and is summarized by Malik et al.The heterozygote state (intermediate) was found to be 10.3 to 23.3 percent and homozygous (severe) state in 2.8 to 20 percent of cases of COPD.

Alfa 1-Antitrypsin Deficiency Screening

In patients of Caucasian descent who develop COPD at a young age (< 45 yr) or who have a strong family history of the disease, it may be valuable to identify coexisting α_1-antitrypsin deficiency. This could lead to family screening or appropriate counseling.

Young patients with severe hereditary α_1-antitrypsin with severe hereditary α_1-antitryspin augmentation therapy. However, this therapy is very expensive, not available in most countries, and not recommended for patients with COPD that is unrelated to α_1-antitrypsin deficiency.

THE OVERLAP OF COPD AND ASTHMA

Active asthma patient have TEN fold risk of Chronic bronchitis and SEVENTEEN fold risk of Emphysema.

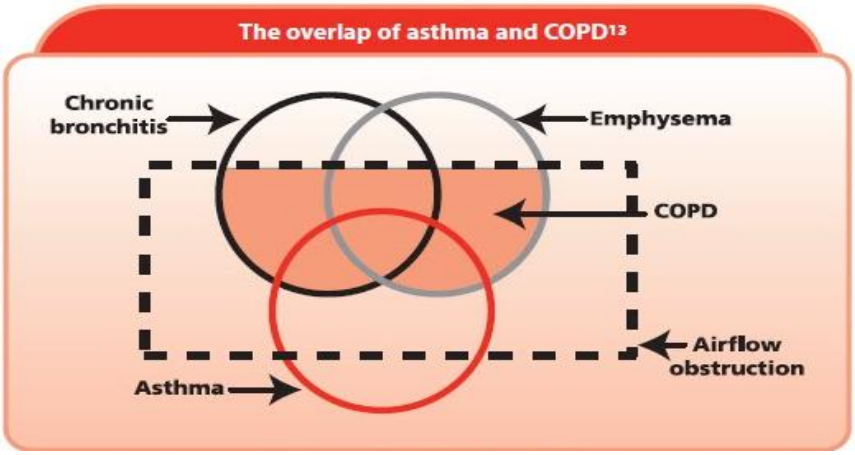


FIG 14a : THE OVERLAP OF COPD AND ASTHMA

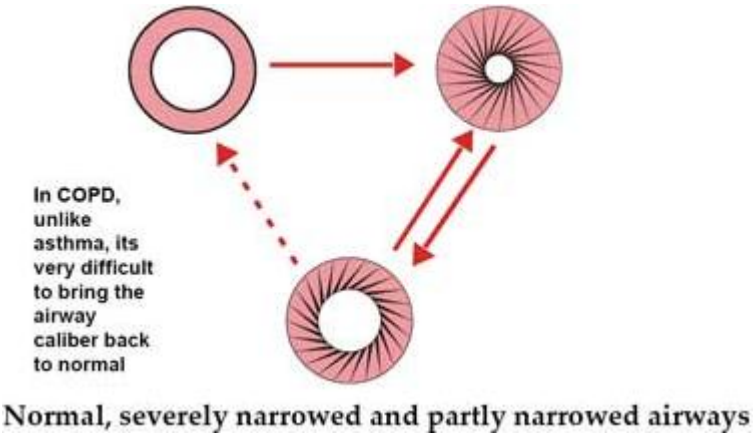


FIG 14b : THE OVERLAP OF COPD AND ASTHMA

Respiratory Infections: Childhood respiratory infections have been assessed as potential predisposing factor for the eventual development of COPD.

MECHANISMS OF AIRWAY OBSTRUCTION IN COPD

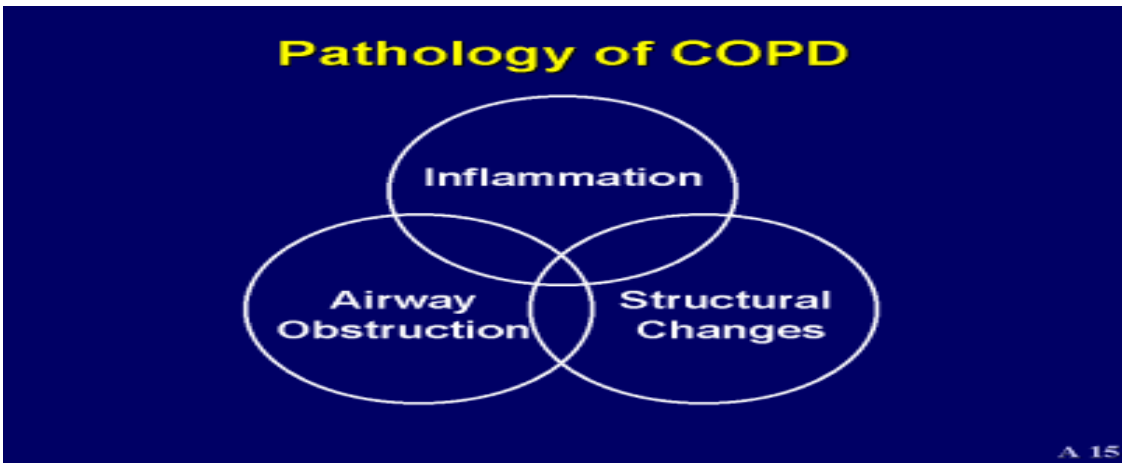


FIG 15 : MECHANISMS OF AIRWAY OBSTRUCTION IN COPD

The three mechanisms account for the elevation in peripheral airways resistance in patients with COPD.

i) The loss of elasticity of the lungs in emphysema allows the airways to narrow

ii) The airways narrow because the supporting alveolar attachment are destroyed by the emphysematous process

iii) The inflammatory process in the airways thickens the wall and narrows the lumen of the distal airways.

PATHOLOGY [41-43]

Several of the abnormalities in chronic bronchitis and emphysema occur in large bronchi (airways with cartilages in their walls and more than 2 mm in diameter), bronchioles (no cartilages in their walls and less than 2 mm internal diameter), and parenchyma. Since emphysema and chronic bronchitis are invariably associated in the same patient, it is common to find changes in all the above three components, although one or the other change may predominate.

The important change in the large bronchi is the hypertrophy of the mucus secreting glands in the sub epithelial layer that are enlarged and are thought to secrete most of the mucus found in the airways. The hypertrophy can be measured as the thickness of the gland layer in histological sections and comparing it to that of the bronchial wall and is expressed as Reid index. The mucous gland hypertrophy is largely seen in the larger bronchi and is equally present throughout the lungs. The mucus secreting goblet cells are also elevated in number in the large as well as in the bronchioli. Bronchial muscle hyperplasia is present in patient with COPD.

The increasing amount of muscle may be responsible for the airway hyper reactivity seen in this patient. Emphysema are classified as,

(a) Centriacinar Emphysema affects the respiratory brochioles . This is probably secondary to bronchiolitis. It is commoner at the lung apices.
(b) Panacinar (or) Panlobular Emphysema. All components of the acinus are involved about equally. usually related with $\propto_1$ antiprotease deficiency. It can also occur in bases of the lung.
(c) Distal Acinar Emphysema (or) Paraseptal emphysema predominantly involvs the alveolar ducts and sacs.

PATHOGENESIS [43]

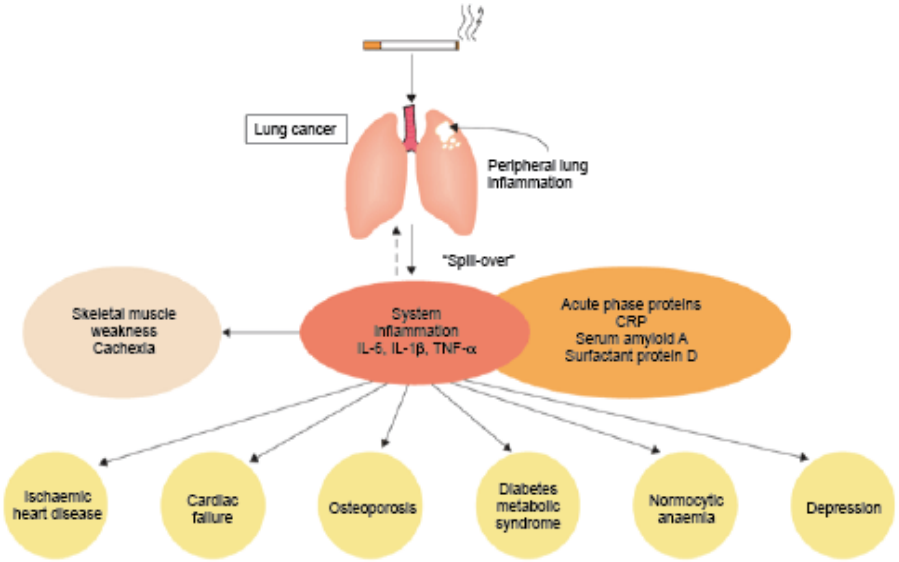


FIG 16: SYSTEMIC MANIFESTATIONS OF COPD

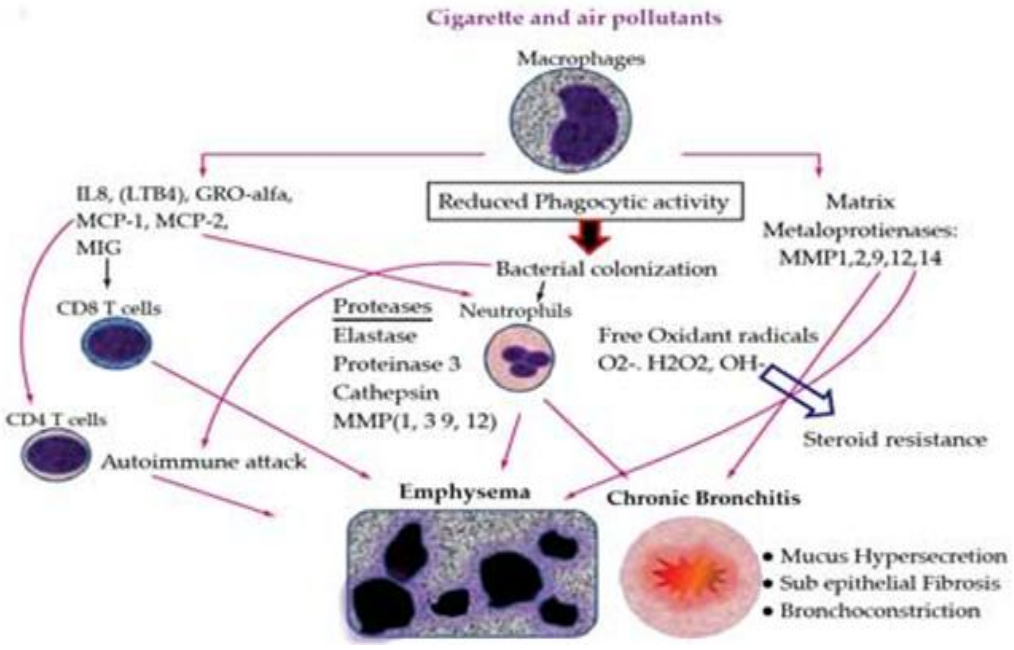


FIG 17 : PATHOGENESIS OF COPD

Inflammatory markers-Cytokines in COPD

- **Interleukin-6**
- **TNFα**
- **IL-1β**
- **Chemokines- CXCL8 (IL-8)** are released during the course of illness and during acute exacerbations of COPD patients and these cytokines are responsible for systemic inflammation and leads to weight loss,cardiac failure ,normocytic anaemia etc.

CLINICAL FEATURES OF COPD [44-49]

The usual presentation is at the fifth decade of life. The characteristic symptoms of chronic bronchitis are cough with expectoration, wheeze, and breathlessness. The cough and expectoration are usually exacerbated from time to time particularly more during the winter.

Pure emphysema is mainly manifested as breathlessness and wheezing, cough and expectoration are less important symptoms. The patient gives a history of progressive dyspnea, sometimes starting apparently after a mild infection and following exertion or exercise. Airflow obstruction causes dyspnea and by the time this present, the FEV is about 1 liter or less than 50 percent of the predicted value. The course progresses over the next 5 year or more with further loss of FEV. The

patient will try to breathe with pursed lips to utilize the respiratory muscles maximum.

Corpulmonale is more common in chronic bronchitis, this is less common in emphysema except terminally. Respiratory failure is the common mode of death of emphysema patients. The emphysema patient maintains a near normal $\boldsymbol{PaO_2}$ and normal $\boldsymbol{PaCO_2}$ by hyperventilation until a late stage of the disease.

Infectious (60-80% of all exacerbations)	Environmental factors
Frequent (70-85% of all infectious exacerbation)	Air pollution
Viruses (influenza and parainfluenza viruses, rhinoviruses, coronaviruses)	Non-adherence to respiratory medication
Hemophilus influenzae	Cold air
Streptococcus pneumoniae	Allergens
Moraxella catarrhalis	Tobacco smoking
Infrequent (15-30% of all infectious exacerbations)	
Pseudomonas aeruginosa	
Opportunistic gram-negative species	
Staphylococcus aureus	
Chlamydophila pneumoniae	
Mycoplasma pneumoniae	

FIG 18: CAUSES OF EXACERBATION OF COPD

m MRC GRADING DYSPNOEA [50]

mMRC Grade 0	I only get breathless with strenuous exercise.
mMRC Grade 1	I get short of breath when hurrying on the level or walking up a slight hill.
mMRC Grade 2	I walk slower than people of the same age on the level because of breathlessness, or I have to stop for breath when walking on my own pace on the level.
mMRC Grade 3	I stop for breath after walking about 100 meters or after a few minutes on the level.
mMRC Grade 4	I am too breathless to leave the house or I am breathless when dressing or undressing.

SYSTEMIC MANIFESTATIONS AND CO-MORBIDITIES OF COPD

- Metabolic disorders
  - Type 2 diabetes mellitus
  - Metabolic syndrome
  - Dyslipidemia
  - Cachexia
  - Obesity
- Skeletal muscle wasting
- Bone diseases: Osteopenia and osteoporosis
- Cardiovascular disease
  - Ischemic heart disease
  - Hypertension,
  - Pulmonary hypertension
  - Corpulmonale
- Cancer: Lung cancer (small cell and non-small cell cancer)
- Obstructive sleep apnea
- Depression and anxiety disorders

PHYSICAL SIGN [44-49]

In early stage of the disease there may not be any findings on examination. In later stages, there will be emaciation, cyanosis, anaemia, edema, and raised jugular venous pressure, if there is associated corpulmoale and heart failure.

The chest is barrel shaped with kyphosis, elevated anteroposterior diameter, ribs being set more horizontal and these changes are permanent and one may feel an inspiratory tracheal tug due to the contraction of low, flat diaphragm. The movement of the chest wall is reduced with limited expansion. The patient may use his accessory muscles of respiration.

JVP may be seen during expiration. In the more severe cases, the costal margins will be drawn inwards on inspiration, paradoxically, due to the pull of the low, flattened diaphragms. On percussion reveal hyperresonant due to hyperinflation of lungs. On auscultation there is a prolonged expiratory phase, wheeze and crepitations are heared.

Airflow obstruction is detected by placing the chest piece of the stethoscope over the trachea and timing of a forced expiration. Normally it is possible to empty the lungs in 4-5 seconds, but a patient with airflow obstruction shows a prolonged expiratory time. Patients with end stage COPD may adopt positions that relieve dyspnoea, by leaning forward with neck extended called asTripod sign. The accessory respiratory muscle helps in to keep the airway patent. Expiration often takes place through pursed lips. Paradoxical in drawing of the lower intercostals space is often evident (Hoover's sign), cyanosis may be present; clubbing is not associated with COPD. If it present one should rule out bronchiectasis or lung carcinoma. An enlarged, tender liver indicates heart failure due to

elevated intrathoracic pressure. Asterexis may be seen with severe hypercapnia.

COPD patients usually ail from muscle wasting, loss of body weight and low quality of life.

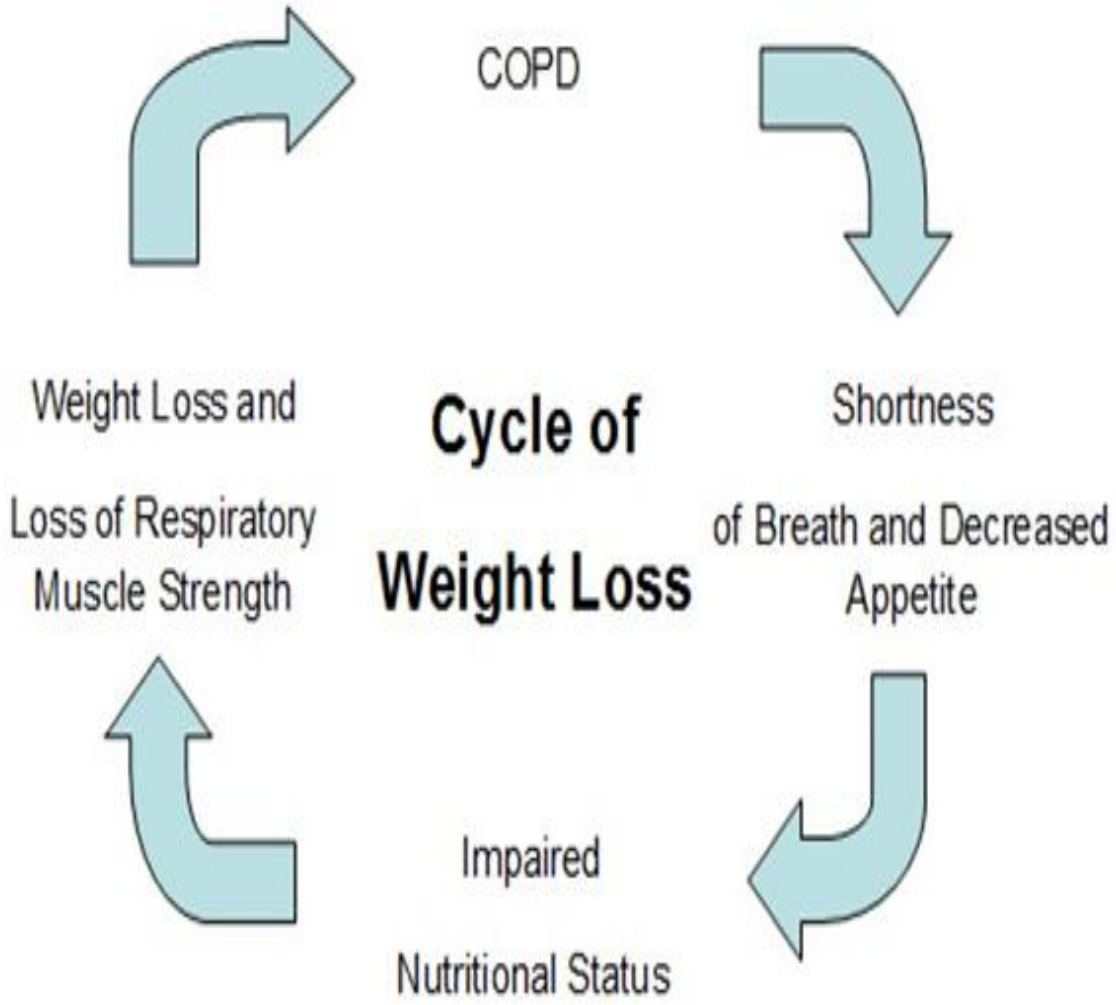
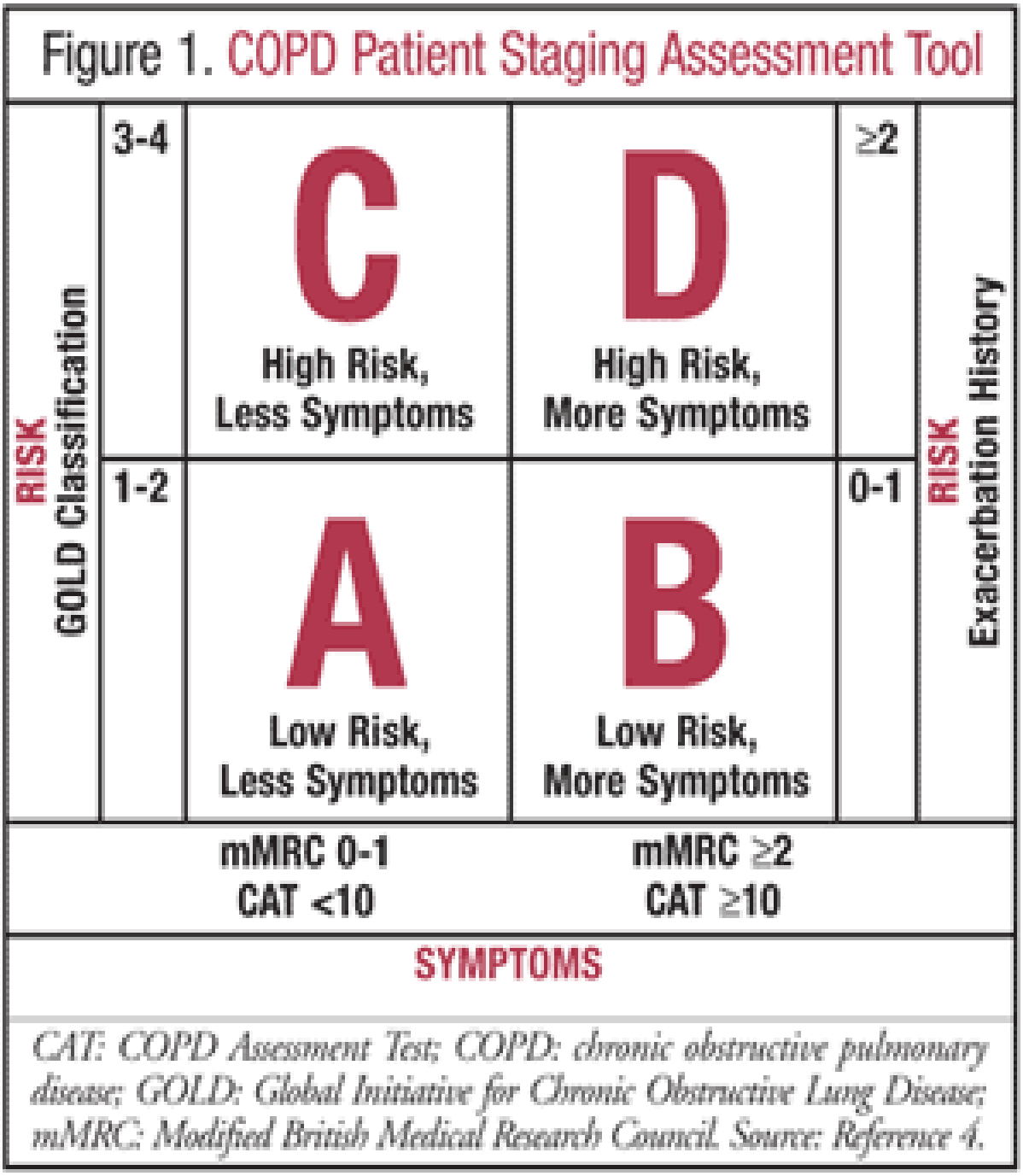


Figure 1. COPD Patient Staging Assessment Tool



CAT: COPD Assessment Test; COPD: chronic obstructive pulmonary disease; GOLD: Global Initiative for Chronic Obstructive Lung Disease; mMRC: Modified British Medical Research Council. Source: Reference 4.

PULMONARY FUNCTION TESTING.[51-52]

Pulmonary function evaluation not only establishes the diagnosis of COPD but also assesses the progression and severity of COPD.

FIG 19: SPIROMETRY

Spirometry is performed in the following individuals

- Unexplained dyspnea or suspicion of COPD
- Screening of habitual smokers for early detection
- Repeat spirometry after medications to detect the reversibility.
- Guidelines for rational therapy

Classification of severity:	FEV_1% (or FEV_1/FVC)	Post-bronchodilator FEV_1
Mild	<.70	≥80% predicted
Moderate	<.70	50% ≤ FEV_1 <80% predicted
Severe	<.70	30% ≤ FEV_1 <50% predicted
Very severe	<.70	FEV_1 <30% predicted or FEV_1 <50% predicted plus chronic respiratory failure*

* Chronic respiratory failure defined as Pao_2 <60 mm Hg with or without a $PAco_2$ <50 mm Hg while breathing room air.

FIG 20: CLASSIFICATION OF SEVERITY OF COPD

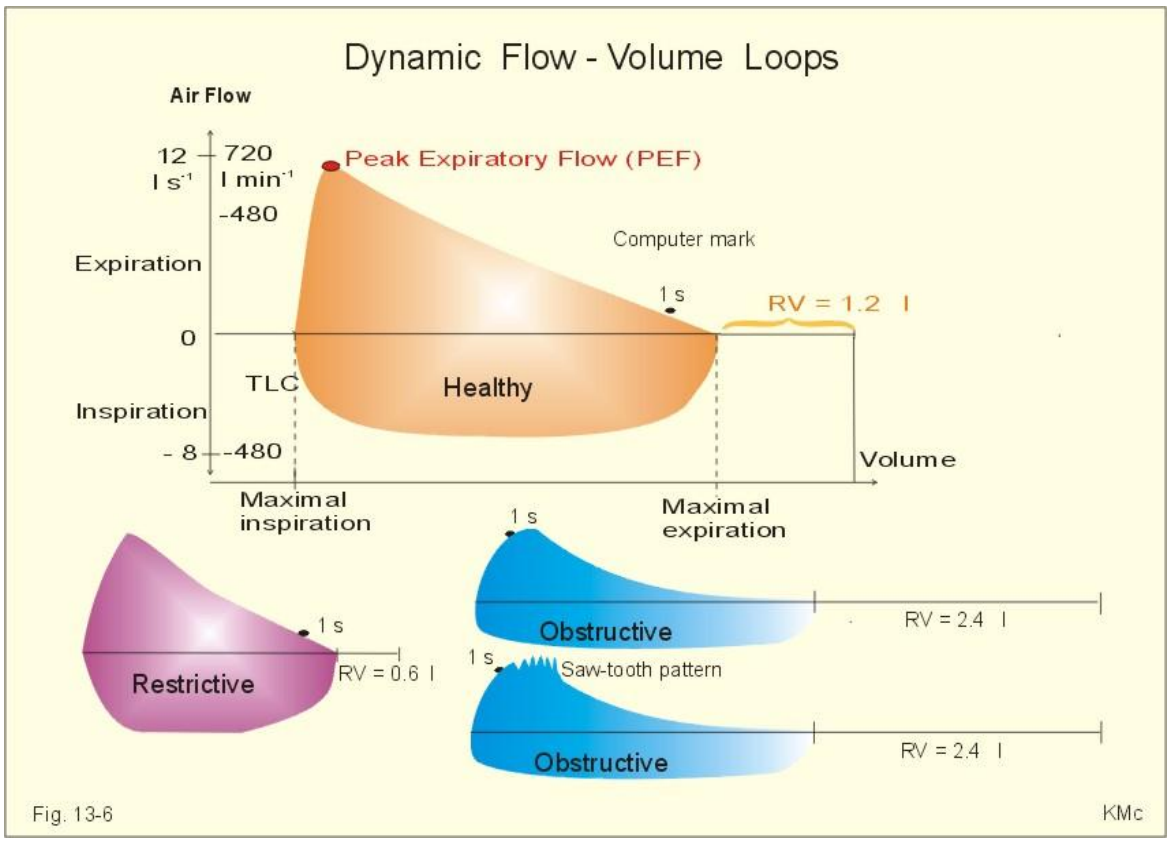


FIG: 21 DYNAMIC FLOW –VOLUME LOOPS

PROGNOSIS [53-54]

Long term prospective studies in patients with severe COPD with FEV of less than 1 liter have shows

- 5 yr survival rates - 69 %
- 10 yr survival rates - 40%

The presence of radiological evidence of emphysema or of bullae in one study resulted in five-year mortalities of 53 percent and 70 percent respectively. Right ventricular failure carries a poor prognosis with a five mortality of 60 to 80 percent reported in different studies. Right ventricular systolic pressure of > 35 mm H, FEV of < 30 percent of predicted and age > 70 years is other poor prognostic factors.

Three risk factors have been useful in predicting the outcome of a patient with COPD age, smoking status, and FEV. The studies of prognosis in the National Institutes of Health Intermittent Positive Pressure Trial confirmed the findings of earlier studies that age and initial FEV are powerful predictors of outcome. The only consistent predictor of decline in FEV besides the initial FEV is the bronchodilator response of the patient. The larger the response the slower the decline in FEV and this relation is not dependent on the initial FEV.

In persons with FEV < 0.75 L, mortality rate at

- 1 yr - 30 %
- 10 yr - 95%

However, some patients with severe airflow obstruction may survive longer, and even up to 15 years. Some other data suggest that next to cessation of smoking, a higher degree of reversibility of airflow obstruction and a lower degree of airway reactivity are the two most important predictors of a slower decline in FEV.

BODE INDEX FOR STAGING COPD [55]

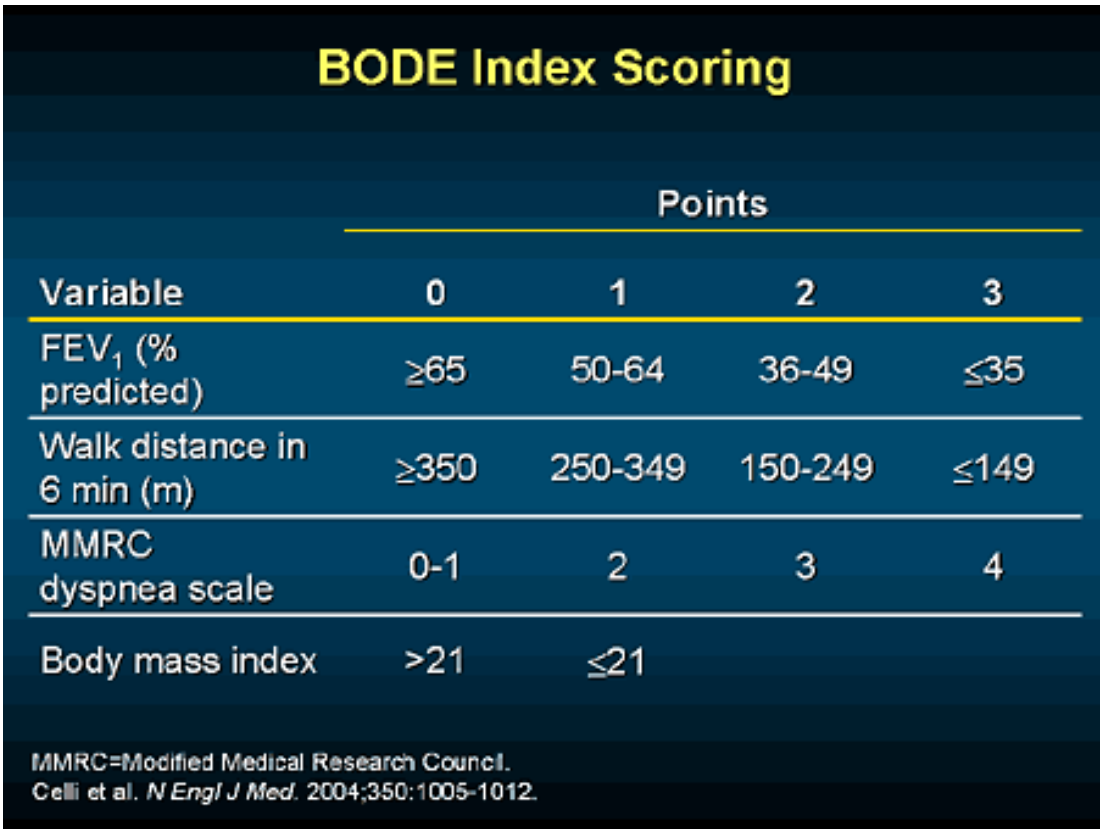


FIG 22: BODE INDEX FOR STAGING COPD

BODE INDEX PREDICTS

- PROGNOSIS
- DEATH AND HOSPITALIZATION BETTER THAN FEV1 ALONE

INVESTIGATIONS

Sputum Examination [56-58]

Sputum Examination is done to all patients with cough of > two wks as per RNTCP Guidelines because Smoking not only increases the risk of COPD and tuberculosis (TB).

Pulse Oximetry [59-60]

Pulse oximetry is to asses hypoxaemia during acute exacerbation of COPD and in respiratory failure cases.

Chest X-Ray[61-62]

A plain Chest X-Ray in both posteroanterior and lateral view to be done.

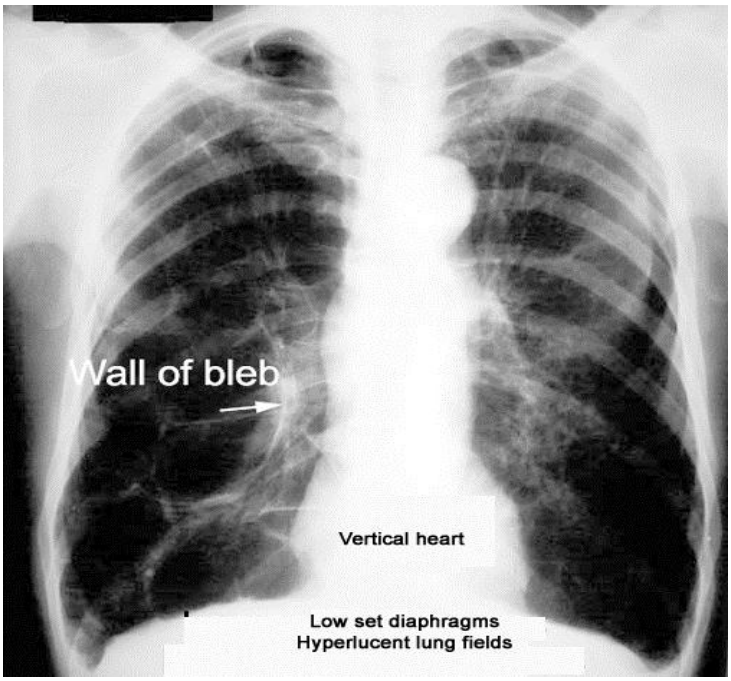


FIG 23: CHEST X-RAY OF COPD

Five radiologic criteria have been described to diagnose emphysema.

(i) A retrosternal space (greatest distance from the sternum to the anterior heart silhouette) more than 2.54 cm(lateral radiograph)

(ii) Regular or irregular hyper-lucency of lung fields reflecting attenuated pulmonary vessels

(iii) Low (mid diaphragm below tenth posterior intercostals space) and flat diaphragm (for two thirds of their length) on lateral films

(iv) Low (mid diaphragm below tenth posterior intercostal space) and flat (for two thirds of their length) on PA radiograph

(v) Bullae one or more clear walled lesions not considered to be a cavity.

Radiologic emphysema is considered to be present when two criteria are present.

CT CHEST [63-64]

Even in normal study of chest xray, CT Scan identifys emphysema.

Computerized tomography is usually not necessary routinely in patients with uncomplicated emphysema.

Early emphysema is identified and quantified by HRCT.

Detailed cardiopulmonary exercise testing (CPET) is helpful in assessing[65-66]

- Prognosis
- Functional status for exercise restrictions
- Impact of therapeutic interventions
- 6-minute walk test (6MWT) is the surrogate for CPET

Alpha -1 Antitrypsin Deficiency[39]

Western data shows that 3% of COPD patients have AAT deficiency and there is no such data available in our country.

TREATMENT GOALS IN A PATIENT WITH STABLE CHRONIC OBSTRUCTIVE PULMONARY DISEASE

NON-PHARMACOLOGICAL MANAGEMENT OF COPD [67]

- **SMOKING CESSATION**
- **PULMONARY REHABILITATION**
- **OXYGEN THERAPY**
- **NON-INVASIVE VENTILATION**
- **SURGERY**

SMOKING CESSATION [68-73]

The most important method to prevent COPD and reduces the frequency of exacerbations.

A variety of pharmacotherapies are available to quit smoking.

Nicotine replacement therapy:

Available in the form of

- Chewing gums
- Tablets
- Patches
- Nasal sprays
- Lozenges
- Inhalers

FIG 24 : NICOTINE PATCH

FIG 25: NICOTINE INHALER

Bupropion:

It is the nicotine receptor antagonist; a weak norepinephrine - dopamine reuptake inhibitor in the brain.

Carenicline:

Acts by partial agonist of the nicotinic receptor as well as simultaneously blocks it.

ROLE OF PHYSICAL REHABILITATION[74]

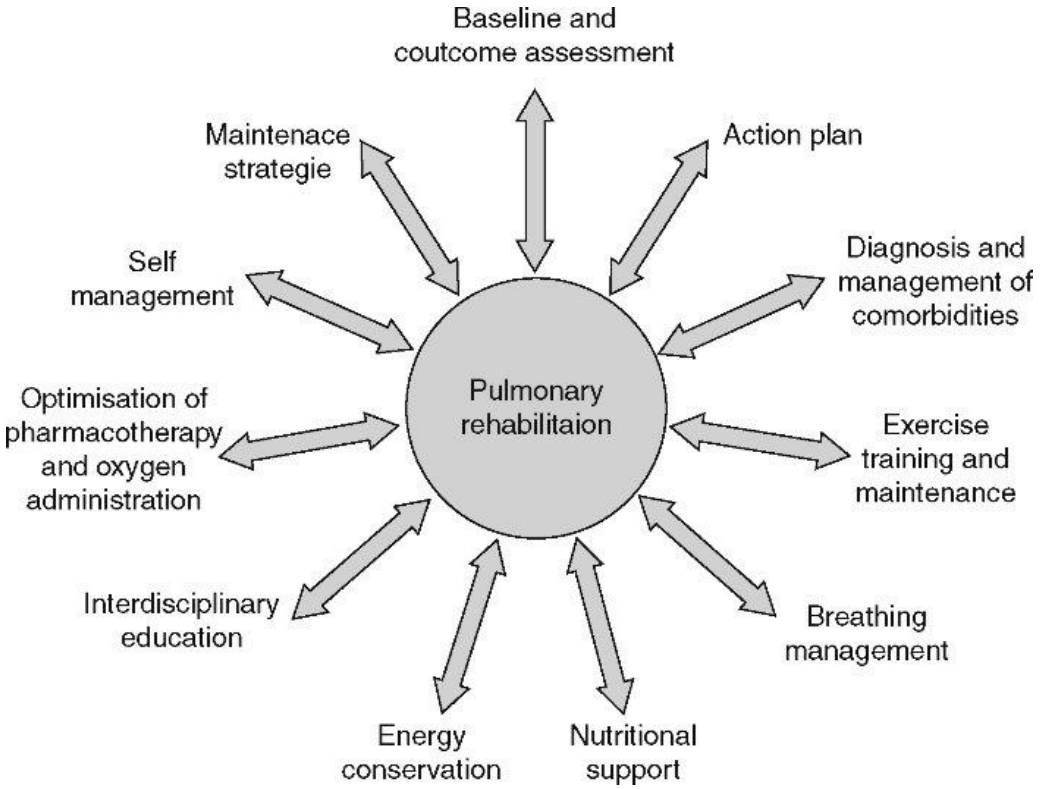


FIG 26: ROLE OF PHYSICAL REHABILITATION

OXYGEN THERAPY [75-77]

The oxygen therapy is helpful in reduction of symptoms especially in patients with systemic complications.

INDICATIONS FOR LONG TERM OXYGEN THERAPY

I .Continous O_2

1. Resting pa O2<_ 55 mm Hg
2. Resting pa O2 56-59 mm Hg or SaO2 89% in the presence of any of the following
   - **A.** Dependent oedema suggesting Congestive Cardiac Failure
   - **B.** P pulmonale in ECG(P wave > 3mm in standard leads II, III, aVf)
   - **C.** Erythrocytosis (heamatocrit>56%)

(a) Reimbursable only with additional documentation justifying O_2 prescription and a summary of more conservative therapy that has failed.

II. Non - Continous O_2

1. O2 flow rate and number of hours per day must be specified.
2. During exercise Pa O2<55mmHg or Sa O2 <88% with associated complications, such as pulmonary hypertension, daytime somnolence or cardiac arrhythmias.

NON-INVASIVE VENTILATION [78-81]

NIV is well established in the treatment of Acute Exacerbation of COPD patients, and its controversial in stable COPD.

Benefits of NIV are

- Maximum inspiratory pressure
- Gas exchange

The indications for NIV in stable COPD are

1. Diagnosed case of COPD, optimization of other therapies and exclusion of sleep apnoea if required.
2. Presence of both symptoms like fatigue, dysponea, morning headache, etc.), and physiologic criteria (one of the following):
   (a) PaCO2≥55 mmHg or PaCO2 of 50-54 mmHg and nocturnal desaturation (oxygen saturation by pulse oximeter≤88% for five continuous minutes while receiving oxygen therapy at 2L/min); and
   (b) PaCO2 of 50-54 mmHg and hospitalization related to recurrent (≥2 in a 12-month period) episode of hypercapnic respiratory failure.

ROLE OF VACCINATIONS[81-82]

Two vaccines are recommended

- Influenza vaccine
- Pneumococcal vaccine

These two vaccines are much beneficial in stage 3 and stage 4 COPD Patients or Patients with recurrent exacerbations.

PHARMACOLOGICAL MANAGEMENT OF COPD [83]

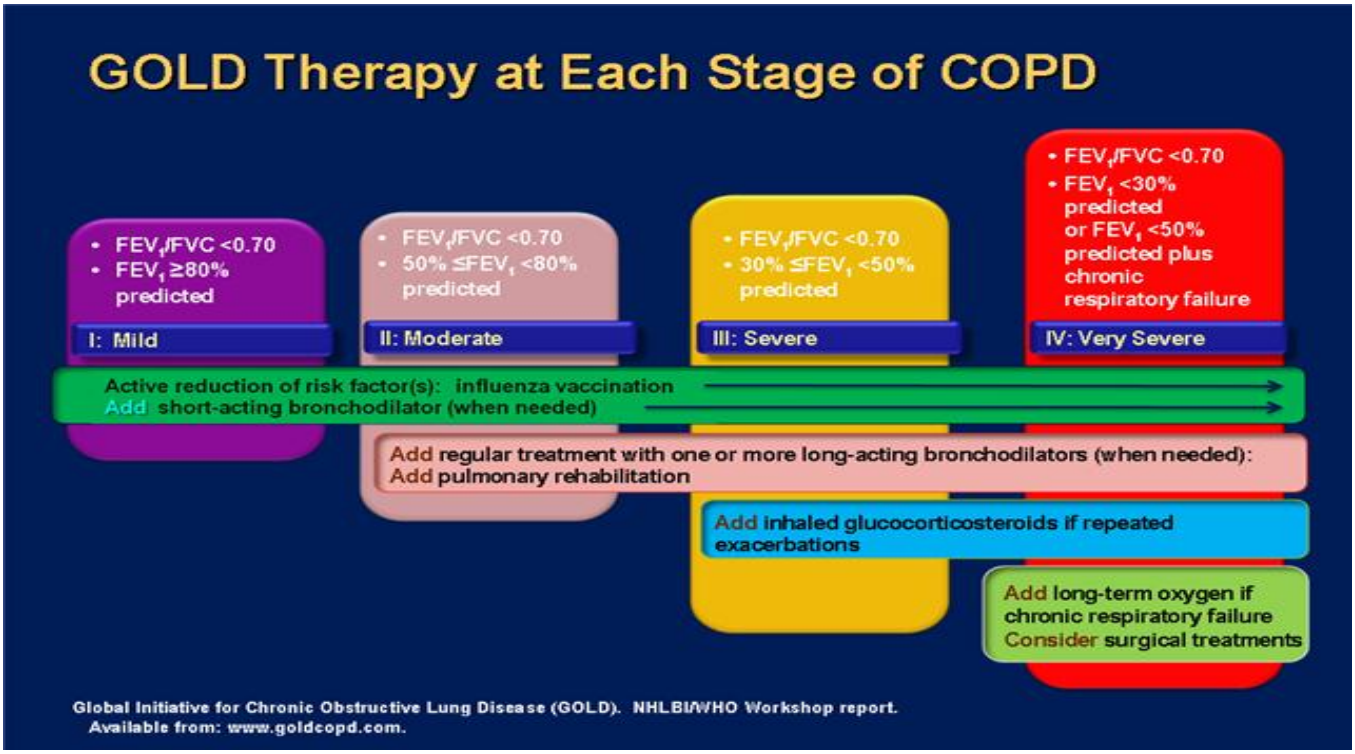


FIG 27: GOLD THERAPY AT EACH STAGE OF COPD

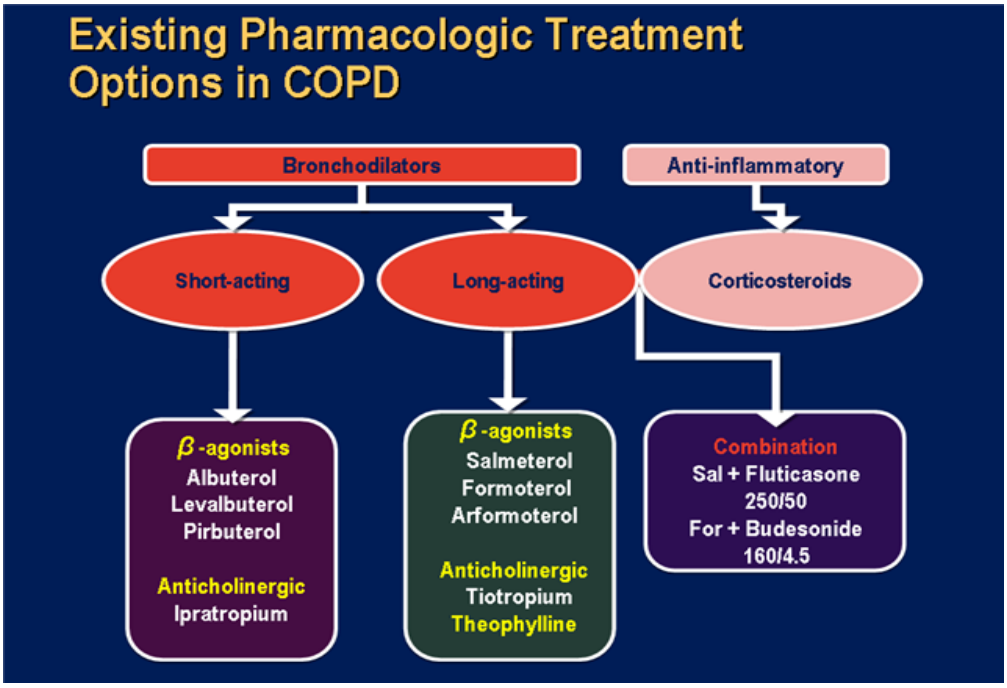


FIG 28: PHARMACOLOGIC TREATMENT OPTIONS IN COPD

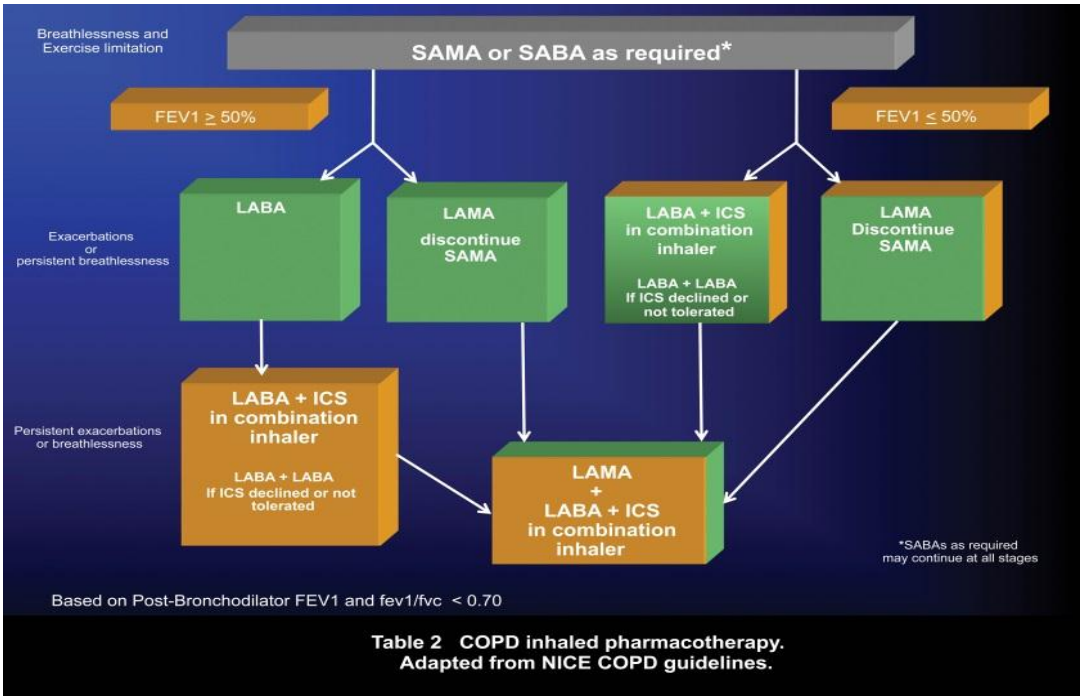


FIG 29: COPD INHALED PHARMACOTHERAPY

INDIAN STRATEGY FOR THE MANAGEMENT OF COPD

SUGGESTED TREATMENT GUIDELINES FOR PATIENTS WITH STABLE COPD

Category	Initial therapy		Add-on therapy (if patient continues to have symptoms)
	First choice	Alternative choice	
Mild	SABA or SAMA prn	Methyl xanthines	-
Moderate	LAMA	LABA	Methylxanthines to LAMA/ LABA
Severe	ICS plus LABA	LAMA	Methylxanthines to LAMA or ICS plus LABA

Indications for hospital assessment or admission for exacerbations of COPD

- Marked increase in intensity of symptoms, such as sudden development of resting dyspnea, change in vital signs
- Severe underlying COPD
- Onset of new physical signs (e.g. cyanosis, peripheral edema)
- Failure of exacerbation to respond to intial medical management
- Significant comorbidities
- Frequent exacerbations

- Newly occurring arrhythmias
- Diagnostic uncertainty
- Older age
- Insufficient home support

Indications for intensive care unit admission of patients with exacerbations of COPD

- Severe dyspnea that responds inadequately to initial emergency therapy
- Changes in mental status (confusion, lethargy, coma)
- Persistent or worsening hypoxemia (Pa_{o2} <5.3 kPa, 40mm Hg), and/or severe/worsening hypercapnia (Pa_{co2} >8.0 kPa, 60mm Hg, and/or severe/worsening respiratory acidosis (pH < 7.25) despite supplemental oxygen and noninvasive ventilation.
- Need for invasive mechanical ventilation
- Hemodynamic instability-need for vasopressors
- Local resources need to be considered.

Management of severe but not life-threatening exacerbations of COPD in the emergency department or the hospital

- Assess severity of symptoms, blood gases, chest X-ray
- Administer controlled oxygen therapy and repeat arterial blood gas measurement after 30-60 min
- Bronchodilators:
  - Increase doses and/or frequency
  - Combine2-agonists and anticholinergics
  - Use spacers or air-driven nebulizers
  - Consider adding intravenous methylxanthines, if needed
- Add oral or intravenous glucocorticosteroids
- Consider noninvasive mechanical ventilation
- At all times:
  - Monitor fluid balance and nutrition
  - Consider subcutaneous heparin
  - Identify and treat associated conditions (e.g., heart failure, arthythmias)
  - Closely monitor condition of the patient.

World COPD Day takes place each year on the second or third Wednesday in November World COPD Day 2013 theme

"It's Not Too Late"

This positive message was chosen to emphasize the meaningful actions people can take to improve their respiratory health, at any stage before or after a COPD diagnosis

WORLD COPD DAY 2014 LOGO

FIG 30: WORLD COPD DAY-2014 LOGO

MATERIALS AND METHODS

SOURCE OF DATA

All patients who presented with history of cough, sputum, breathlessness or wheezing of more than 3 months duration to the Medical Outpatient Department or admitted in the medical wards of Coimbatore Medical College Hospital were subjected to pre and post-bronchodilator Pulmonary Function Testing. Those patients whose post-bronchodilator FEV1/FVC was less than 0.7 were included in this study. This study period was from AUGUST 2013 to JULY 2014.

COLLABORATING DEPARTMENTS

Department of TB & Chest Medicine, Coimbatore Medical College Hospital.

Design of Study	: Observational Clinical study
Period	: AUGUST 2013 to JULY 2014
Sample size	: 60 Patients
Ethical Committee Approval	: Obtained
Consent	: Informed consent was obtained

INCLUSION CRITERIA

- Both in-patients and out-patients were included in the study.
- Both new and previously diagnosed cases were included in the study.
- Patients with post-bronchodilator FEV1 / FVC < 0.7.

EXCLUSION CRITERIA

- Patients with systemic illness like Diabetes, Coronary artery heart disease, Cardiac failure, renal failure, Liver diseases, Malignancy, Collagen vascular disease.
- Patients with history of present or past pulmonary tuberculosis
- Patients with other lung diseases like interstitial lung disease, Bronchiectasis, Pneumonia, Lung abscess.
- Data was collected using a pretested Proforma meeting the objectives of the study. Detailed history, physical examination and necessary investigations were undertaken.
- Pulmonary Function Testing was done using spirometer. Three satisfactory efforts were recorded and the best effort was considered. Bronchodilatation was done using 200 μg of inhaled salbutamol using a metered dose inhaler and the test was repeated after 15 min.

Patients were subjected to the following investigations:

- Complete Haemogram
- Peripheral smear
- Blood urea, serum creatinine
- Blood sugar
- Spirometry (pre and post bronchodilator therapy)
- Sputum for gram stain and AFB
- Chest X-ray PA view
- Urine: Albumin, Sugar
- ECG in all leads

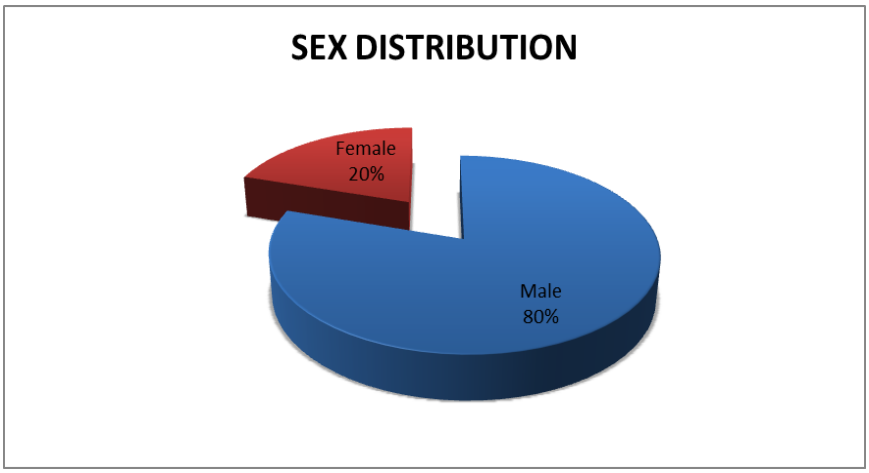
RESULTS

Sixty cases were studied and the following observation and analysis were made.

TABLE 1: SEX DISTRIBUTION

SEX	NUMBER OF PATIENTS	PERCENT
Male	48	80
Female	12	20
Total	60	100

CHART 1: SEX DISTRIBUTION

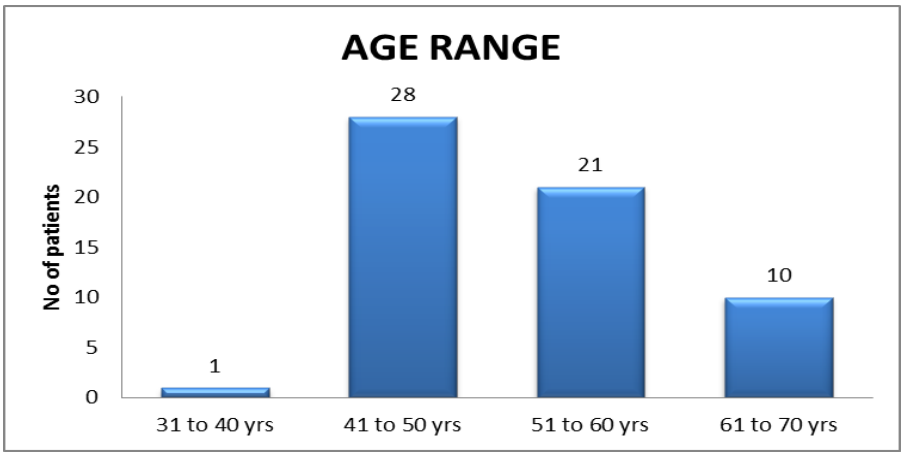


Out of the 60 cases studied, 48 Patients **(80%)** were Males and 12 Patients **(20%)**were Females.

TABLE 2: AGE DISTRIBUTION

AGE GROUP	NUMBER OF PATIENTS	PERCENT
31 to 40 yrs	1	2
41 to 50 yrs	28	47
51 to 60 yrs	21	35
61 to 70 yrs	10	17

CHART 2: AGE DISTRIBUTION



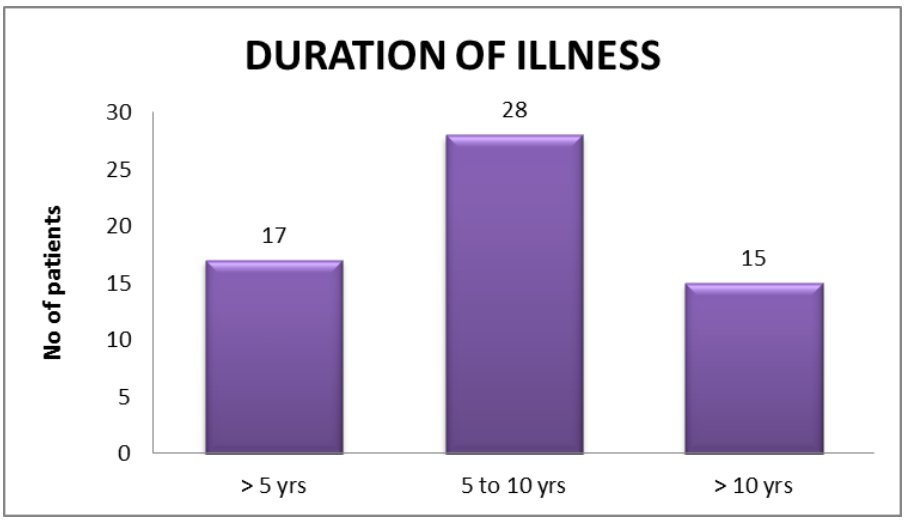
Majority of the patients were in the age group of **41-50 years**.

Minimum age being 38 years and Maximum age being 68 years.

TABLE 3.DURATION OF ILLNESS

DURATION OF ILLNESS	NUMBER OF PATIENTS	PERCENT
0-5 yrs	17	28
6 to 10 yrs	28	47
> 10 yrs	15	25

CHART 3. DURATION OF ILLNESS

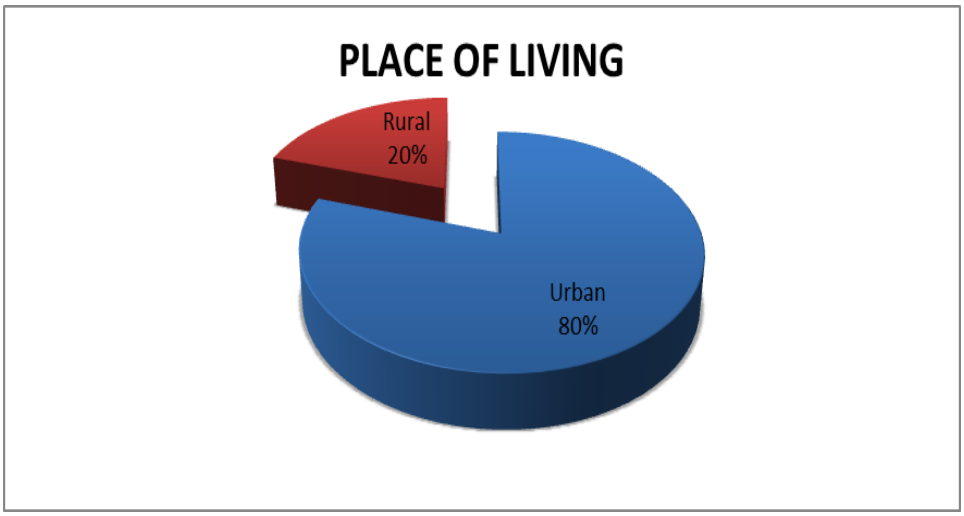


Majority of patients, i.e. 28 patients **(47 %)** had duration of illness 6-10 years, 15 patients **(25%)** had duration of illness of >10 years and 17 patients **(28%)** had duration of illness 0-5 years.

TABLE:4 PLACE OF LIVING

PLACE OF LIVING	NUMBER OF PATIENTS	PERCENT
Urban	48	80
Rural	12	20

CHART: 4 PLACE OF LIVING

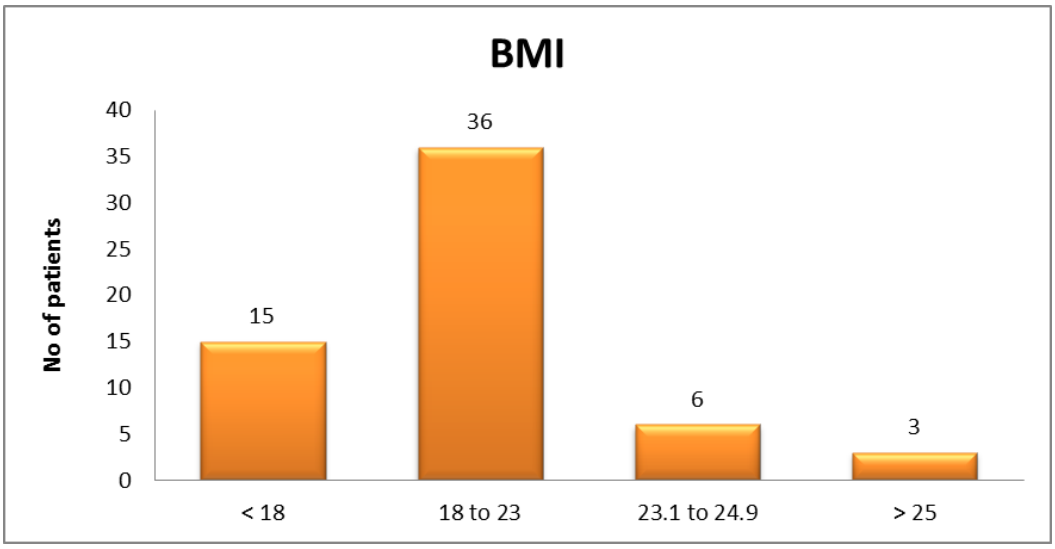


48 Patients **(80%)** were living in urban areas whereas **12** Patients **(20%)** were living in rural area.

TABLE: 5 BODY MASS INDEX

BODY MASS INDEX	NUMBER OF PATIENTS	PERCENT
< 18	15	25
18 to 23	36	60
23 to 24.9	6	10
> 25	3	5

CHART: 5 BODY MASS INDEX

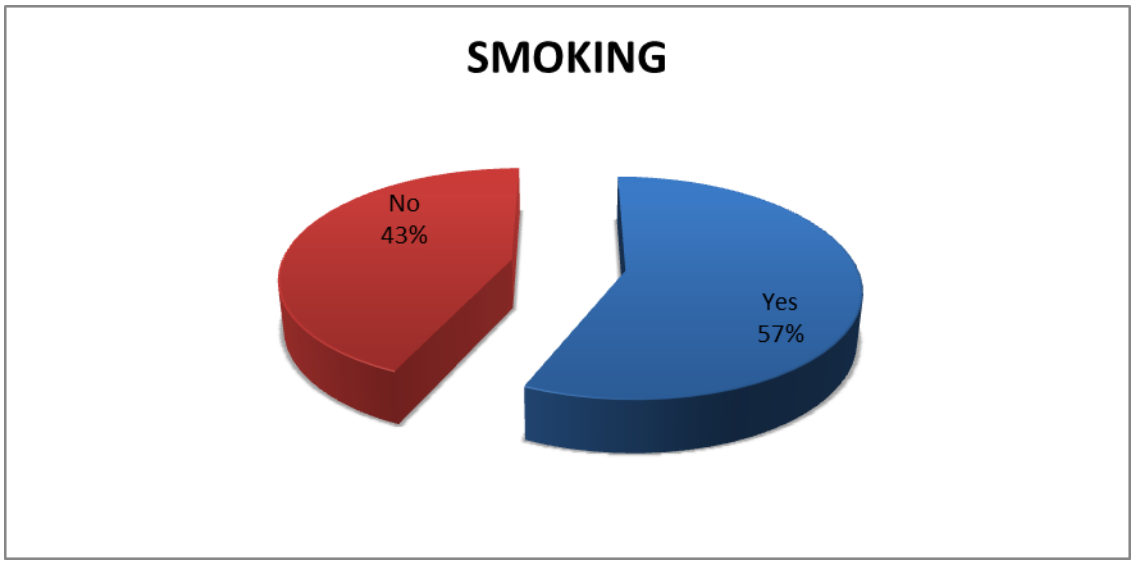


Majority of Patients **(36%)** had BMI within normal limits (18-23kg /sqcm).

TABLE 6: SMOKING HABITS AMONG THE PATIENTS STUDIED

SMOKING	NUMBER OF PATIENTS	PERCENT
Yes	34	57
No	26	43

CHART 6: SMOKING HABITS AMONG THE PATIENTS STUDIED

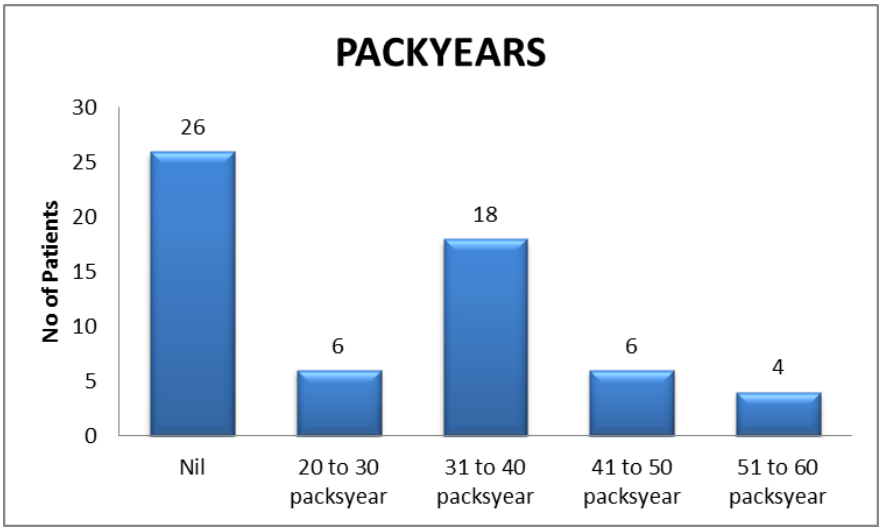


Out of 60 patients, 34 patients (57%) had history of smoking.

TABLE 7: SMOKING PACK YEARS

PACK YEARS	NUMBER OF PATIENTS	PERCENT
NIL	26	43
20 TO 30	6	10
31 TO 40	18	30
41 TO 50	6	10
51 TO 60	4	7

CHART 7: SMOKING PACK YEARS



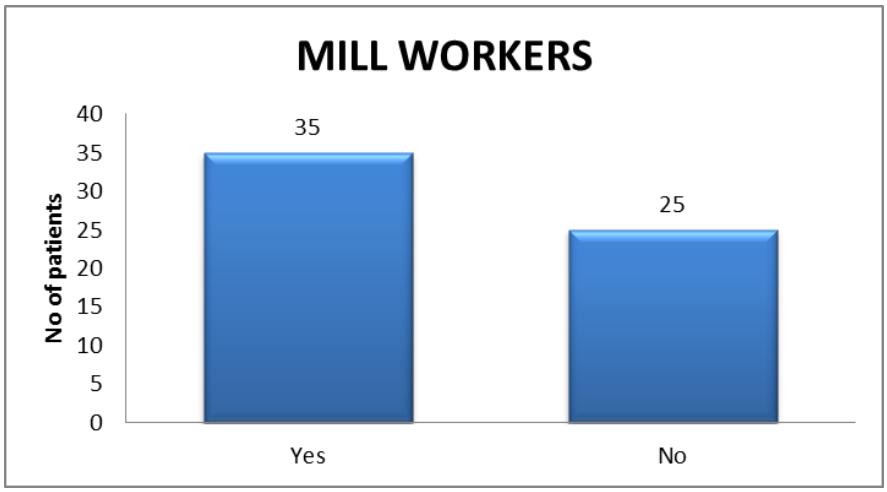
In the present study duration of smoking ranged from 20 to 60 Pack years.

Majority had **31-40 pack years** of duration of exposure.

TABLE 8: OCCUPATIONAL EXPOSURE – COTTON MILL WORKERS

COTTON MILL WORKER	NUMBER OF PATIENTS	PERCENT
Yes	35	58
No	25	42

CHART 8: OCCUPATIONAL EXPOSURE – COTTON MILL WORKERS

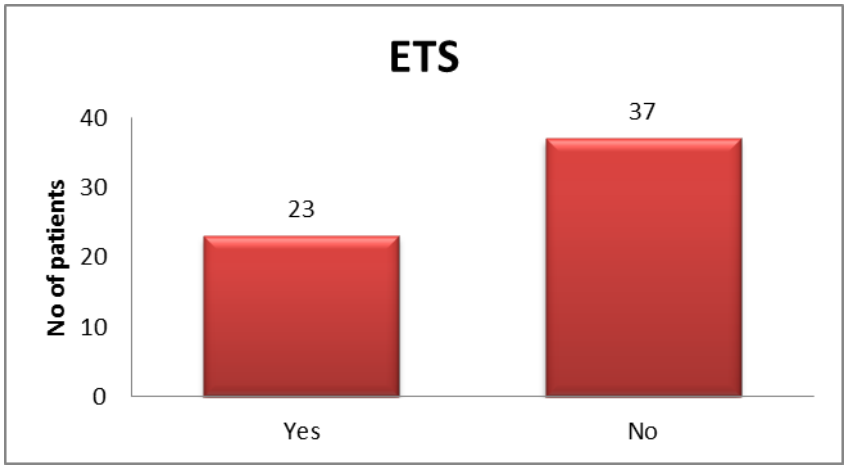


Out of 60 patients, only 35 patients (58%) had history of occupational Exposure. 10 patients had history of exposure for <10 years and 25 patients had history of exposure for >10 years.

TABLE 9: ENVIRONMENTAL TOBACCO SMOKE EXPOSURE

ETS	NUMBER OF PATIENTS	PERCENT
YES	23	38
NO	37	62

FIGURE 9: ENVIRONMENTAL TOBACCO SMOKE EXPOSURE

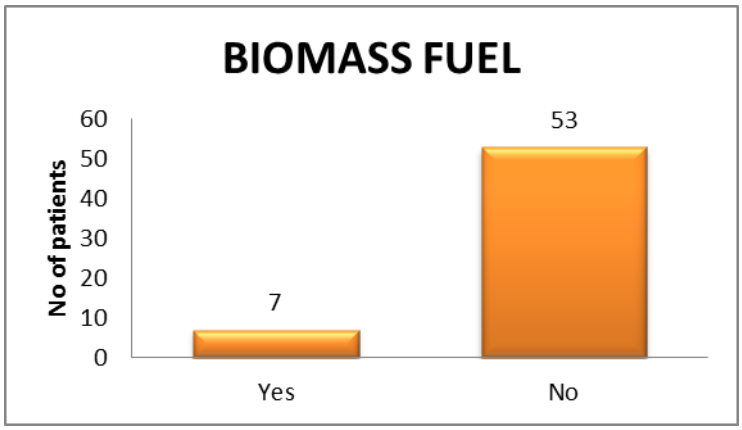


History of exposure to environmental tobacco smoke was present in 23 patients. Most of the patients, i.e. 20 patients had history of environmental tobacco smoke exposure for >3 hours, only 3 patients had history of exposure for <3 hours in a day.

TABLE 10: BIO MASS FUEL EXPOSURE

BIO MASS FUEL EXPOSURE	NUMBER OF PATIENTS	PERCENT
YES	7	12
NO	53	88

CHART 10: BIO MASS FUEL EXPOSURE

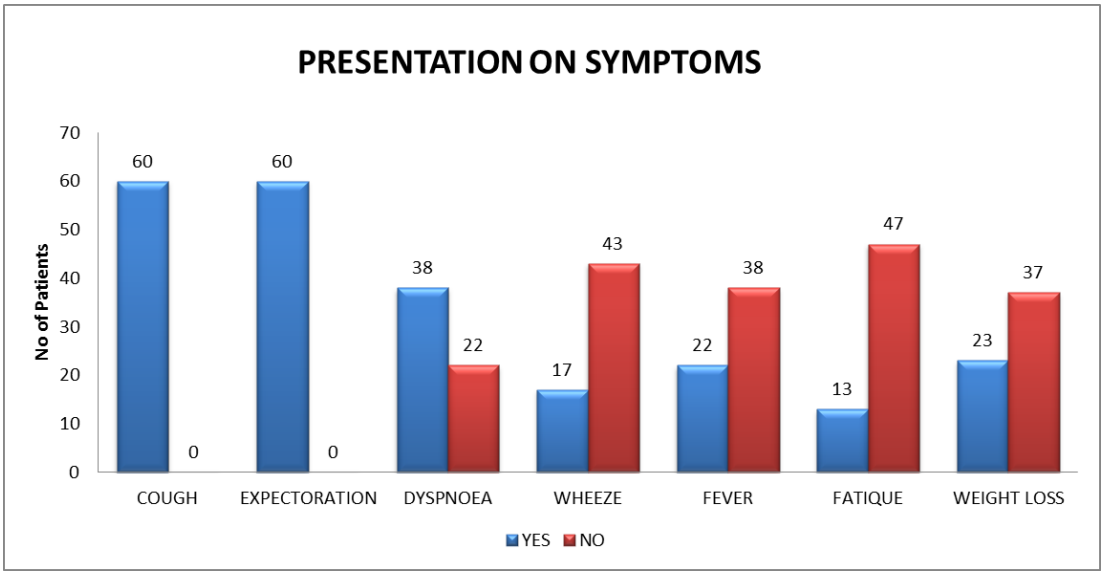


Out of 60 patients, 7 female patients had history of biomass fuel usage and exposure whereas all 48 male patients (80%) did not have biomass fuel exposure.

TABLE : 11 PRESENTATION OF SYMPTOMS

SYMPTOMS	NUMBER OF PATIENTS	PERCENTAGE
Cough	60	100
Expectoration	60	100
Dyspnoea	38	63
Wheeze	17	28
Fever	22	36
Fatigue	13	22
Weight Loss	23	38

CHART 11: PRESENTATION OF SYMPTOMS



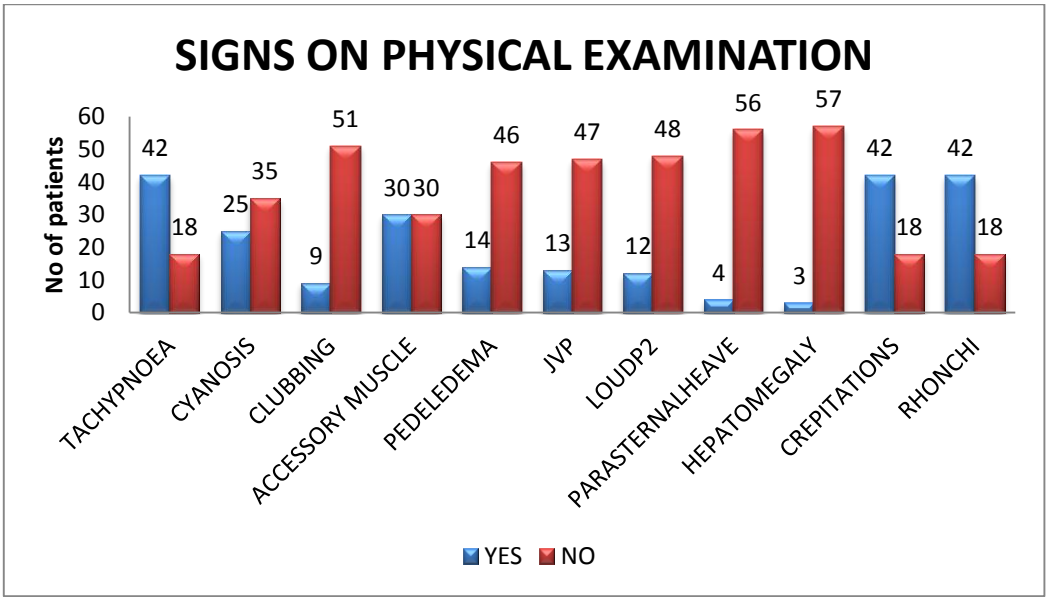
Cough and Expectoration was present in all the 60 patients **(100%).**

Wheeze in 38 patients (63%) and Weight loss in 23 patients (38%).

TABLE 12: SIGNS ON PHYSICAL EXAMINATION

SIGNS	NUMBER OF PATIENTS	PERCENTAGE
Tachypnoea	42	70
Cyanosis	25	42
Clubbing	9	15
Accessory muscle	30	50
Pedel edema	14	23
JVP	13	22
Loud p2	12	20
Parasternal heave	4	7
Hepatomegaly	3	5
Crepitations	42	70
Rhonchi	42	70

CHART 12 :SIGNS ON PHYSICAL EXAMINATION

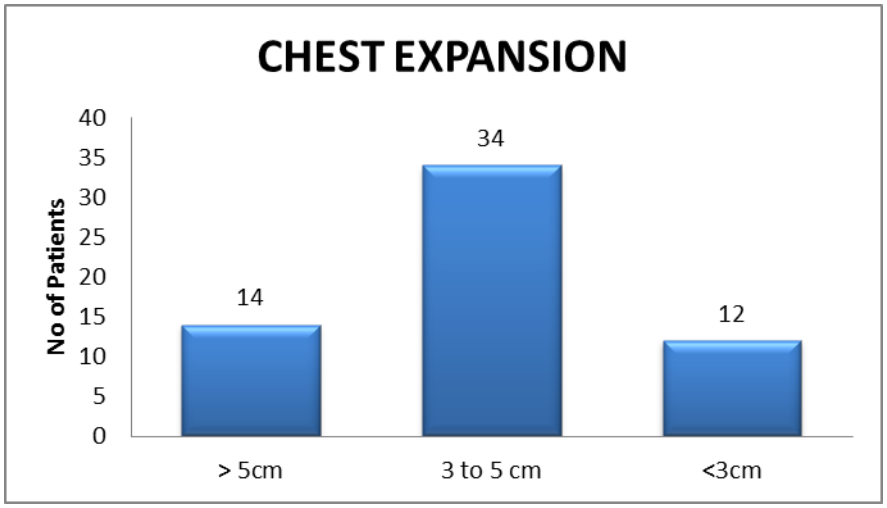


Tachypnea , Rhonchi, Crepitations were present in 42 patients (70%).

TABLE 13: CHEST EXPANSION MEASUREMENT

CHEST EXPANSION	NUMBER OF PATIENTS	PERCENT
> 5CM	14	23
3 TO 5 CM	34	57
<3CM	12	20

FIGURE 13: CHEST EXPANSION MEASUREMENT

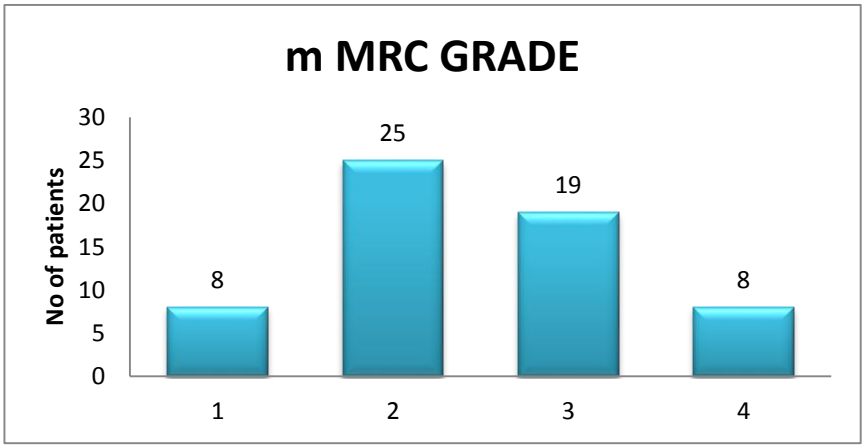


Out of 60 patients ,14 patients **(23%)** had normal chest wall expansion and 46 patients **(77%)** had reduced chest wall movements.

TABLE 14: m MRC GRADING

m MRC GRADING	NUMBER OF PATIENTS	PERCENT
1	8	13
2	25	42
3	19	32
4	8	13

CHART 14: m MRC GRADING

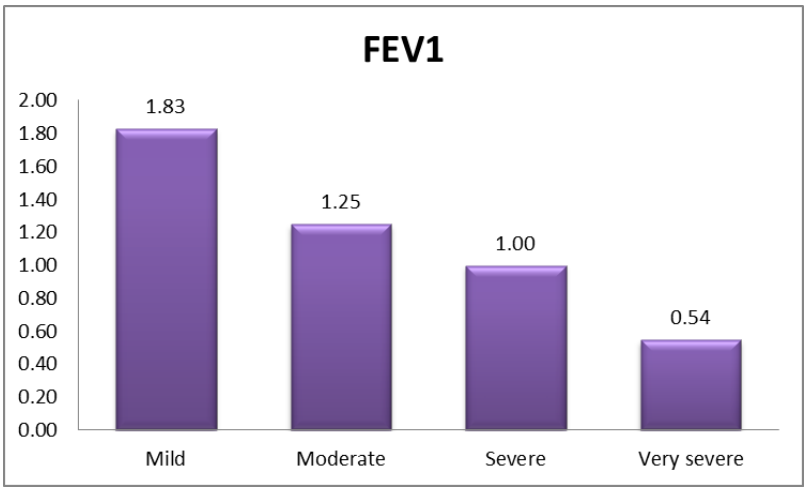


25 patients had Grade 2 Dyspnoea,19 patients had Grade 3 Dyspnoea, 8 patients had Grade 1 Dyspnoea and 8 patients had Grade 4 Dyspnoea.

TABLE 15:FEV1 OF THE PATIENTS STUDIED

FEV 1	NUMBER OF PATIENTS	Mean
Mild	21	1.82
Moderate	23	1.24
Severe	9	0.99
Very Severe	7	0.54

CHART 15: FEV1 OF THE PATIENTS STUDIED

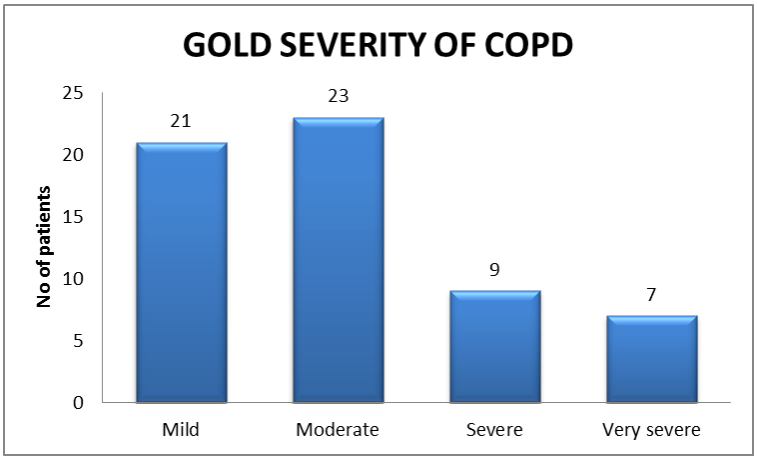


Post bronchodilator FEV1 results shows that 21 patients **(35%)** had mild disease with mean value of 1.83, 23 patients **(38%)** had moderate disease with mean value of 1.25, 9 patients **(15%)** had severe disease with mean value of 1.00 and 7 patients **(11%)** had very severe disease with mean value of 0.54.

TABLE 16: GOLD SEVERITY OF COPD

GOLD SEVERITY OF COPD	NUMBER OF PATIENTS	PERCENT
Mild	21	35
Moderate	23	38
Severe	9	15
Very severe	7	11

CHART 16: GOLD SEVERITY OF COPD



Post bronchodilator FEV1 results shows that 21 patients **(35%)** had mild disease, 23 patients **(38%)** had moderate disease, 9 patients **(15%)** had severe disease and 7 patient **(11%)** had very severe disease

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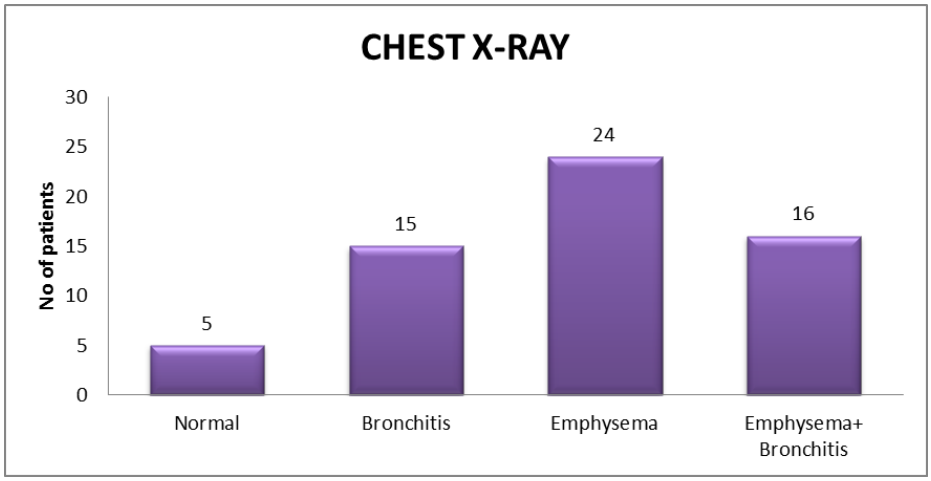
Table 17: CHEST X RAY OF THE PATIENTS STUDIED

CHEST X RAY	NUMBER OF PATIENTS	PERCENT
NORMAL	5	8
BRONCHITIS	15	25
EMPHYSEMA	24	40
EMPHYSEMA + BRONCHITIS	16	27

CHART 17: CHEST X RAY OF THE PATIENTS STUDIED

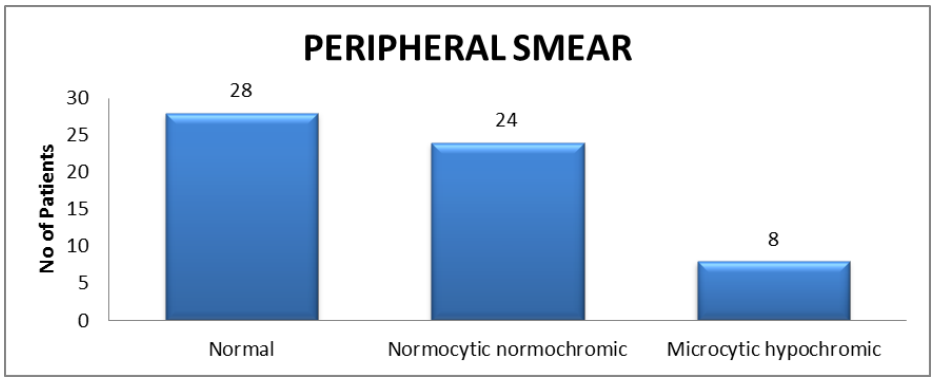


Chest X-ray showed Chronic bronchitis in 15 Patients, Chronic bronchitis with Emphysema in 16 Patients, Emphysema in 24 Patients and was Normal in 5 Patients.

Table 18: PERIPHERAL SMEAR OF THE PATIENTS STUDIED

PERIPHERAL SMEAR	NUMBER OF PATIENTS	PERCENT
NORMAL	28	47
NORMOCYTIC NORMO CHROMIC	24	40
MICROCYTIC HYPO CHROMIC	8	13

CHART 18: PERIPHERAL SMEAR OF THE PATIENTS STUDIED

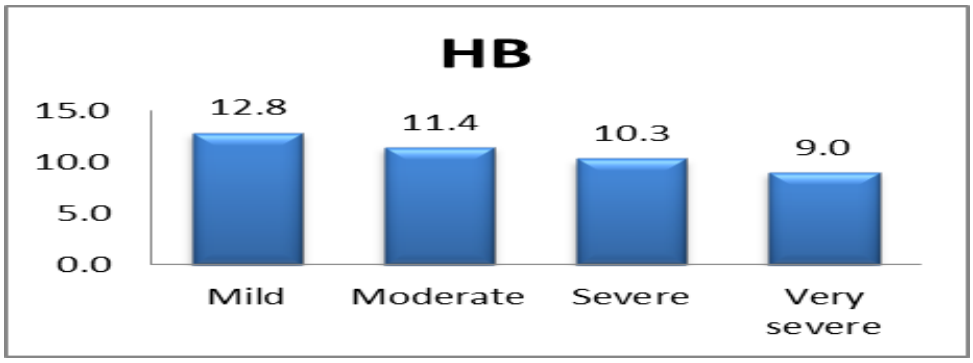


Peripheral smear was Normal in 28 patients, 24 patients had Normocytic Normochromic Anemia and 8 patients had Microcytic Hypochromic Anemia.

Table 19: HAEMOGLOBIN OF THE PATIENTS STUDIED

GOLD CRITERIA STAGING	NUMBER OF PATIENTS	MEAN HB gm%
Mild	21	12.8
Moderate	23	11.4
Severe	9	10.3
Very severe	7	9

CHART 19: HAEMOGLOBIN OF THE PATIENTS STUDIED

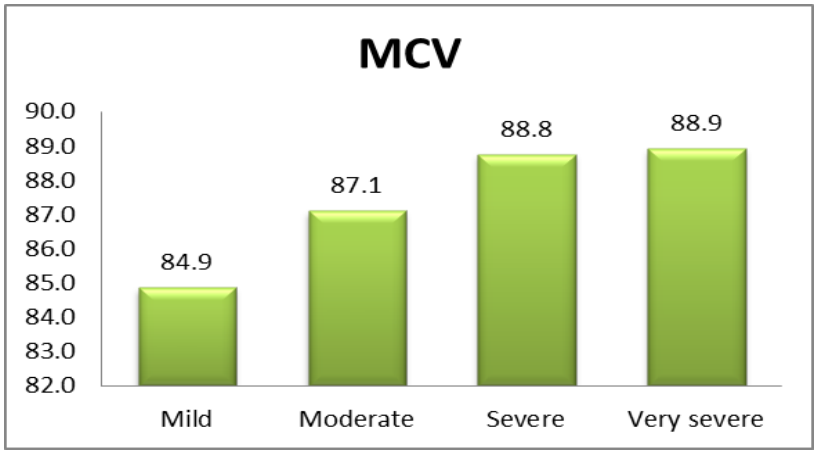


21 Patients with mild disease had Mean Haemoglobin value of 12.8, Moderate disease had 11.4, Severe disease had 10.3 and Very severe disease had value of 9.

TABLE 20: MEAN CORPUSCULAR VOLUME OF THE PATIENTS STUDIED

GOLD CRITERIA STAGING	NUMBER OF PATIENTS	MEAN MCV
Mild	21	84.9
Moderate	23	87.1
Severe	9	88.8
Very severe	7	88.9

CHART 20: MEAN CORPUSCULAR VOLUME OF THE PATIENTS STUDIED

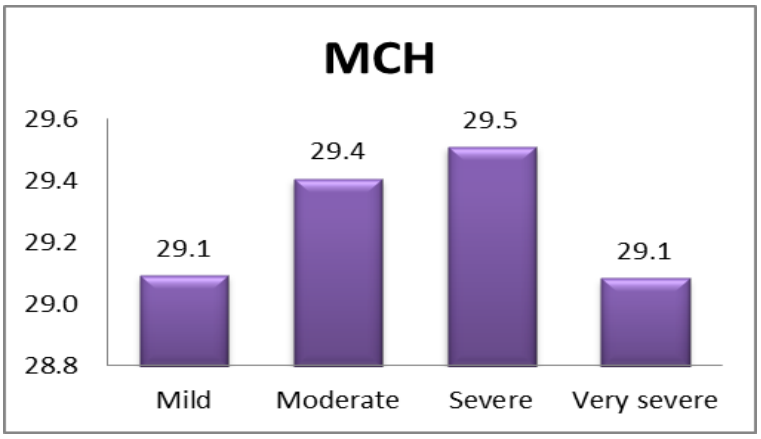


21 Patients in mild disease had Mean MCV value of 84.9, Moderate disease had 87.1, Severe disease had 88.8 and Very severe disease had value of 88.9.

TABLE 21: MEAN CORPUSCULAR HAEMOGLOBIN OF THE PATIENTS STUDIED

GOLD CRITERIA STAGING	NUMBER OF PATIENTS	MEAN MCH
Mild	21	29.1
Moderate	23	29.4
Severe	9	29.5
Very severe	7	29.1

CHART 21: MEAN CORPUSCULAR HAEMOGLOBIN OF THE PATIENTS STUDIED

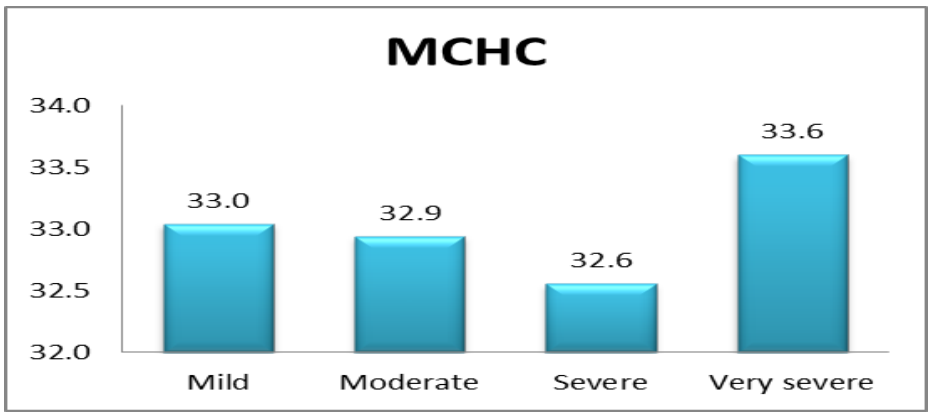


21 Patients in mild disease had Mean MCH value of 29.1, Moderate disease had 29.4, Severe disease had 29.5, Very severe disease had 29.1.

TABLE 22: MEAN CORPUSCULAR HAEMOGLOBIN CONCENTRATION OF THE PATIENTS STUDIED

GOLD CRITERIA STAGING	NUMBER OF PATIENTS	MEAN MCHC
Mild	21	33
Moderate	23	32.9
Severe	9	32.6
Very severe	7	33.6

FIGURE 22: MEAN CORPUSCULAR HAEMOGLOBIN CONCENTRATION OF THE PATIENTS STUDIED

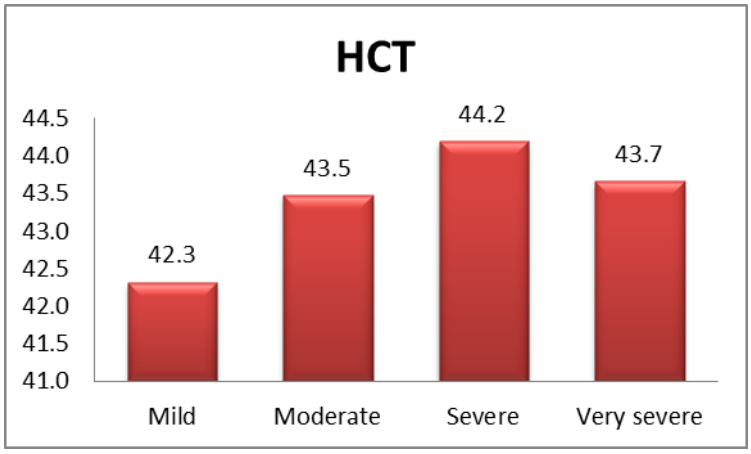


21 Patients in Mild disease had Mean MCHC value of 33, Moderate disease had 32.9, severe disease had 32.6, and Very severe disease had 33.6.

TABLE 23: HAEMATOCRIT OF THE PATIENTS STUDIED

GOLD CRITERIA STAGING	NUMBER OF PATIENTS	MEAN HCT
Mild	21	42.3
Moderate	23	43.5
Severe	9	44.2
Very severe	7	43.7

CHART 23: HAEMATOCRIT OF THE PATIENTS STUDIED

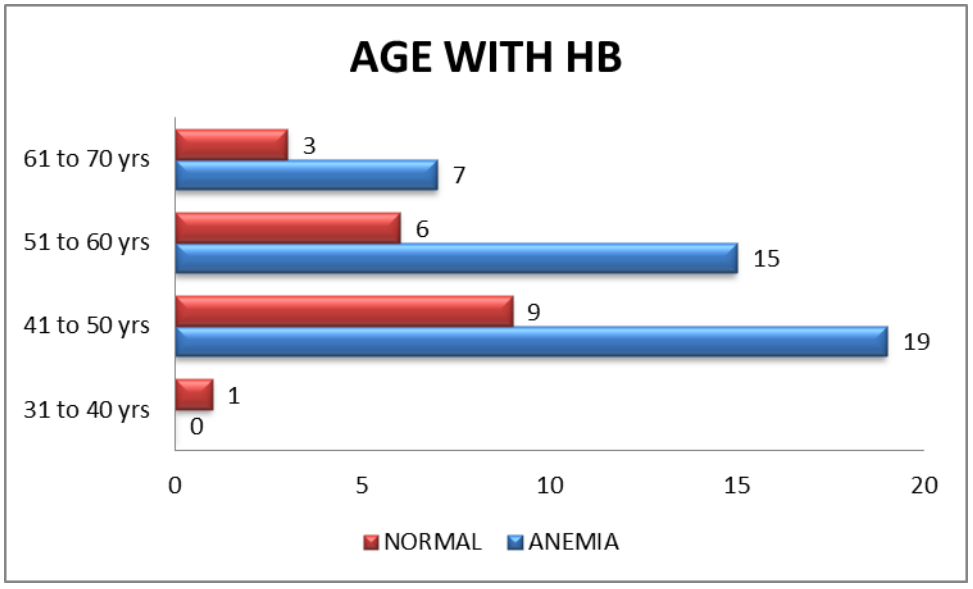


21 Patients in mild disease had Haematocrit value of 42.3, Moderate disease had 43.5, Severe disease had 44.2, Very severe disease had 43.7.

TABLE 24: COMPARISON OF AGE vs HAEMOGLOBIN

AGE	HAEMOGLOBIN	
	ANEMIA	NORMAL
31 to 40 yrs	0	1
41 to 50 yrs	19	9
51 to 60 yrs	15	6
61 to 70 yrs	7	3

CHART 24 :COMPARISON OF AGE vs HAEMOGLOBIN

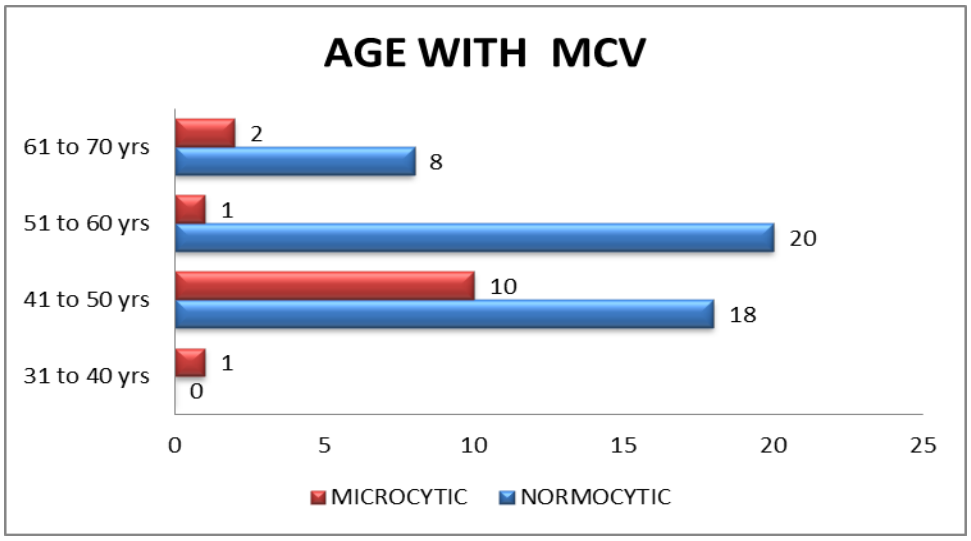


Haemoglobin of the patient were compared with age of the patient. Patient with age group ranged from **41-50 yrs** had 68% chance for development of anemia. . Patient with age group ranged from **51-60 yrs** had 71% chance for development of anemia. Patient with age group ranged from **61-70 yrs** had 70% chance for development of anemia.

TABLE 25: COMPARISON OF AGE vs MCV

AGE	MCV	
	NORMOCYTIC	MICROCYTIC
31 to 40 yrs	0	1
41 to 50 yrs	18	10
51 to 60 yrs	20	1
61 to 70 yrs	8	2

CHART 25: COMPARISON OF AGE vs MCV



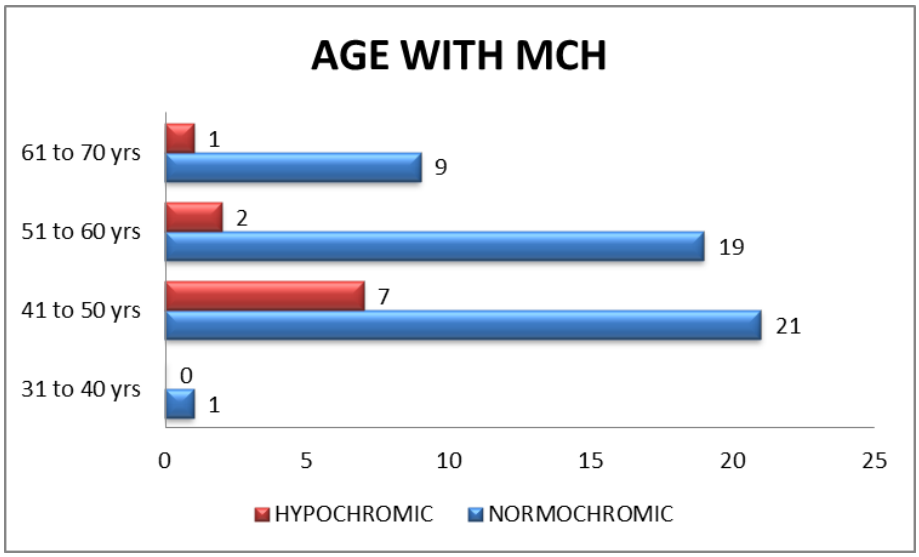
MCV of the patient was compared with age of the patient. Patient with age group ranged from **41-50 yrs** had 64% chance for development of Normocytic Anemia. Patient with age group ranged from **51-60 yrs** had 95% chance for development of Normocytic Anemia. Patient with age group ranged from **61-70 yrs** had 80% chance for development of Normocytic Anemia.

This association was statistically significant with **P value of 0.020.**

TABLE 26: COMPARISION OF AGE vs MCH

AGE	MCH	
	NORMOCHROMIC	HYPOCHROMIC
31 to 40 yrs	1	0
41 to 50 yrs	21	7
51 to 60 yrs	19	2
61 to 70 yrs	9	1

CHART 26: COMPARISION OF AGE vs MCH



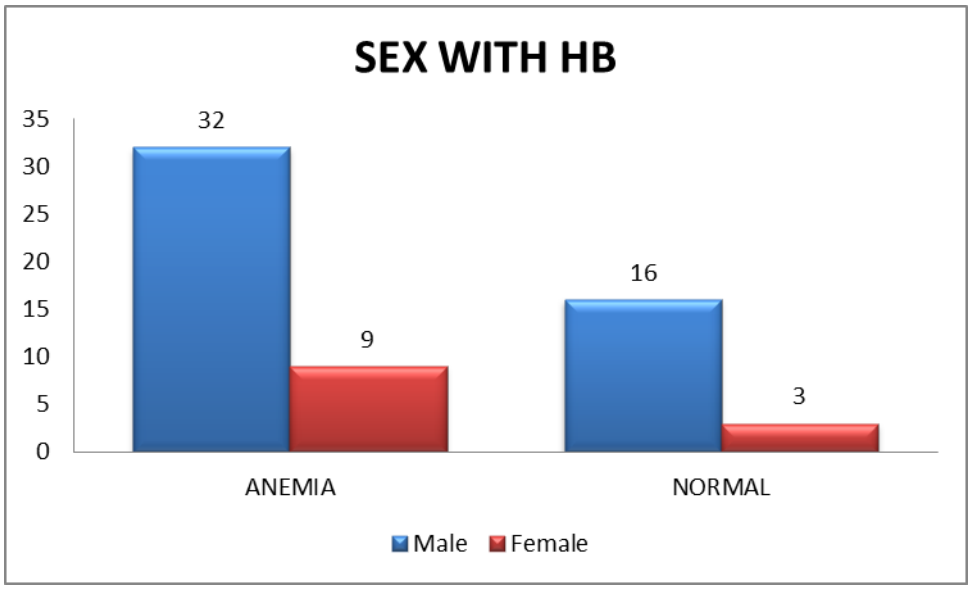
MCH of the patient was compared with age of the patient. Patient with age group ranged from **41-50 yrs** had 75% chance for development of Normochromic. Patient with age group ranged from **51-60 yrs** had 90% chance for development of Normochromia. Patient with age group ranged from **61-70 yrs** had 90% chance for development of Normochromia.

This association was not statistically significant with **P value of 0.442.**

TABLE 27: COMPARISON OF SEX vs HAEMOGLOBIN

SEX	HAEMOGLOBIN	
	ANAEMIA	NORMAL
MALE	32	16
FEMALE	9	3

FIGURE 27: COMPARISON OF SEX vs HAEMOGLOBIN

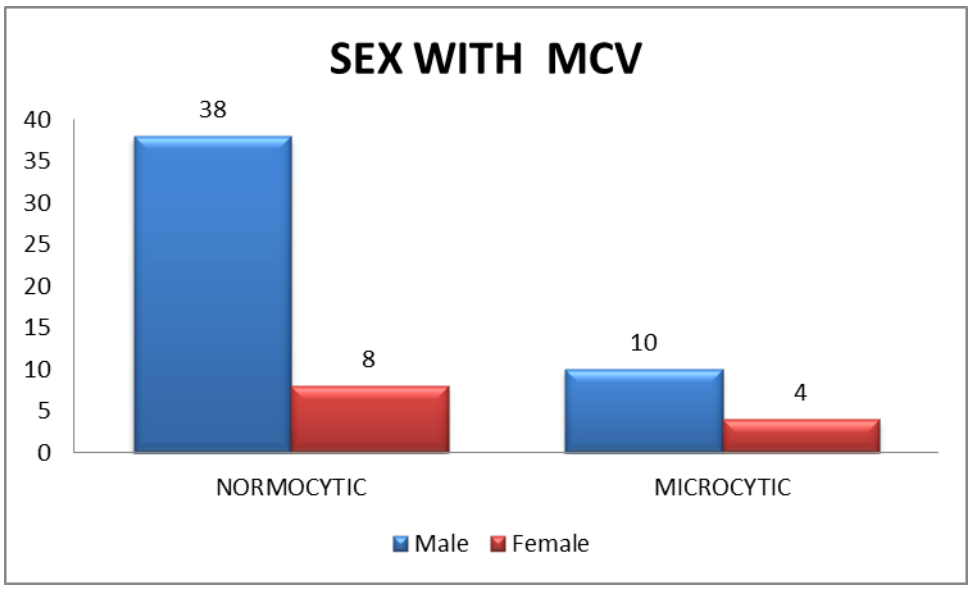


Haemoglobin of the patient was compared with age of the patient. Among 48 male patients 32 (67%) had anemia. Among 12 female patients 9 (75%) had anemia.

TABLE 28 : COMPARISON OF SEX vs MCV

SEX	MCV	
	NORMOCYTIC	MICROCYTIC
MALE	38	10
FEMALE	8	4

CHART 28: COMPARISON OF SEX vs MCV

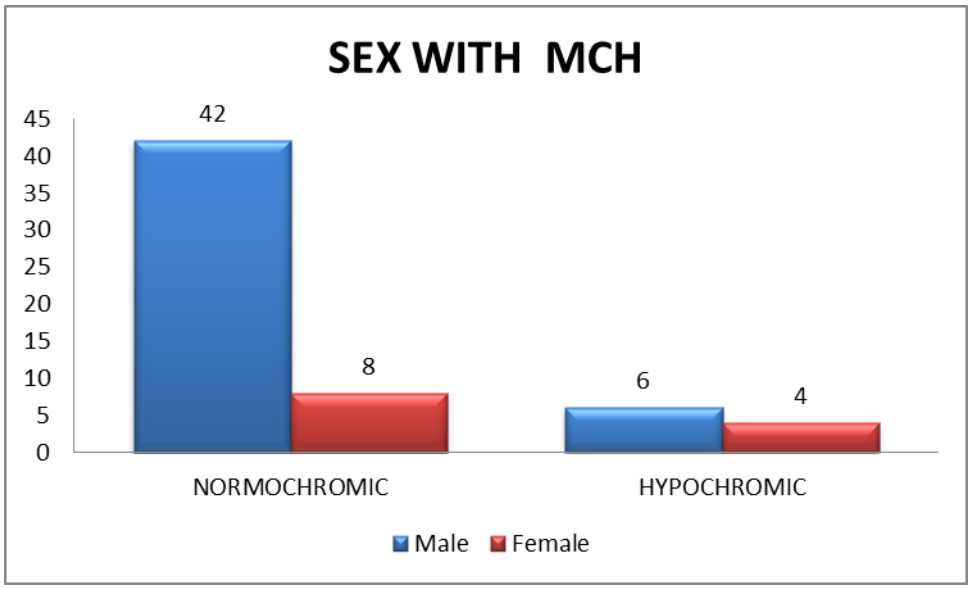


MCV of the patient was compared with age of the patient. Among 48 male patients 38 (79%) had normocytic. Among 12 female patients 8 (67%) had normocytic.

TABLE 29: COMPARISON OF SEX vs MCH

SEX	MCH	
	NORMOCHROMIC	HYPOCHROMIC
MALE	42	6
FEMALE	8	4

CHART 29 : COMPARISON OF SEX vs MCH

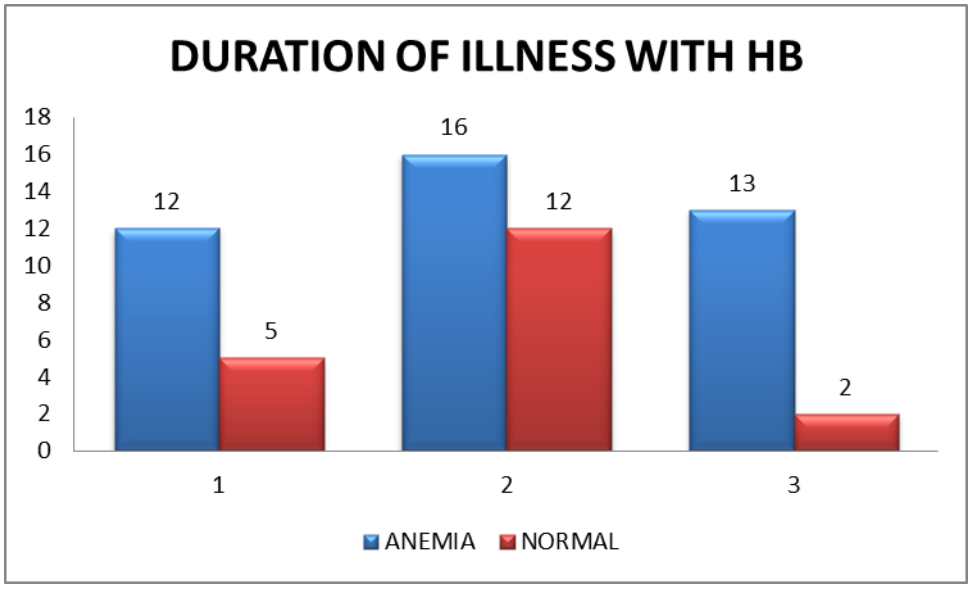


MCH of the patient was compared with age of the patient. Among 48 male patients 42 (88%) had normochromic. Among 12 female patients 8 (67%) had normochromic.

TABLE 30: DURATION OF ILLNESS vs HAEMOGLOBIN

DURATION OF ILLNESS	HAEMOGLOBIN	
	ANEMIA	NORMAL
0-5 yrs	12	5
6 -10 yrs	16	12
>10 yrs	13	2

FIGURE 30: DURATION OF ILLNESS vs HAEMOGLOBIN



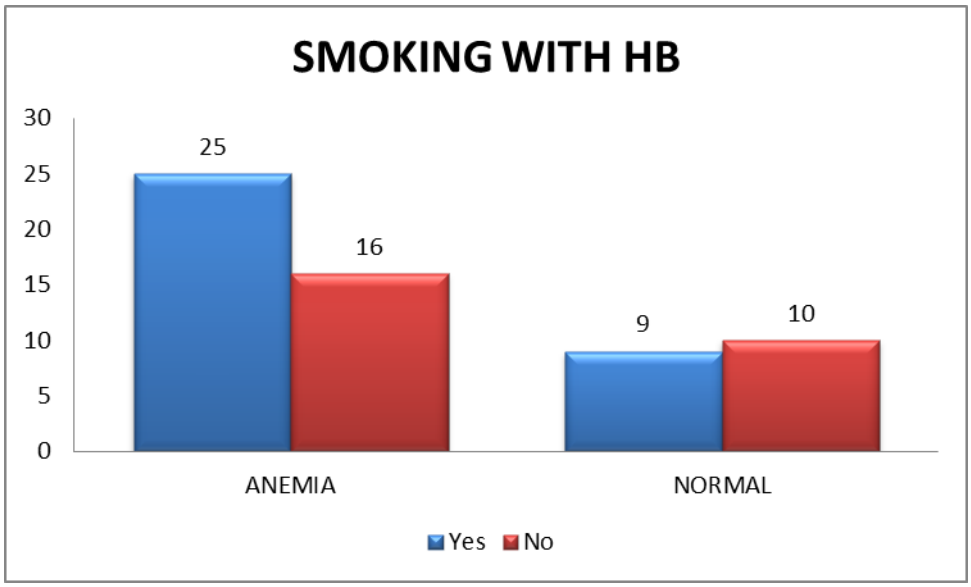
Haemoglobin of the patient was compared with Duration of illness.

Patient with duration of illness more than 6-10yrs had 57% risk for anemia and duration of illness more than 10 yrs had 87% risk for anemia.

TABLE 31: COMPARISON OF SMOKING WITH HAEMOGLOBIN

SMOKING	HAEMOGLOBIN	
	ANAEMIA	NORMAL
YES	25	9
NO	16	10

CHART 31: COMPARISON OF SMOKING WITH HAEMOGLOBIN



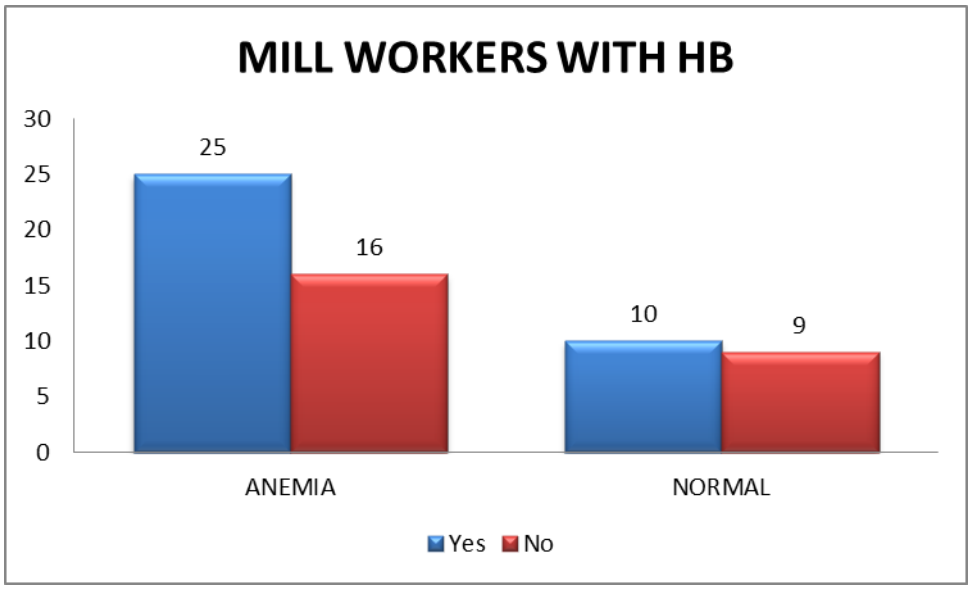
Haemoglobin of the patient was compared with Smoking.

Among the patients with smoking 73% had anemia and in non-smokers 61% had anemia. Although this association was considerable but it was not statistically significant with P value of 0.322

TABLE 32: COMPARISON OF OCCUPATIONAL EXPOSURE – COTTON MILL WORKERS WITH HAEMOGLOBIN

COTTON MILL WORKER	HAEMOGLOBIN	
	ANAEMIA	NORMAL
YES	25	10
NO	16	9

CHART 32: COMPARISON OF OCCUPATIONAL EXPOSURE – COTTON MILL WORKERS WITH HAEMOGLOBIN

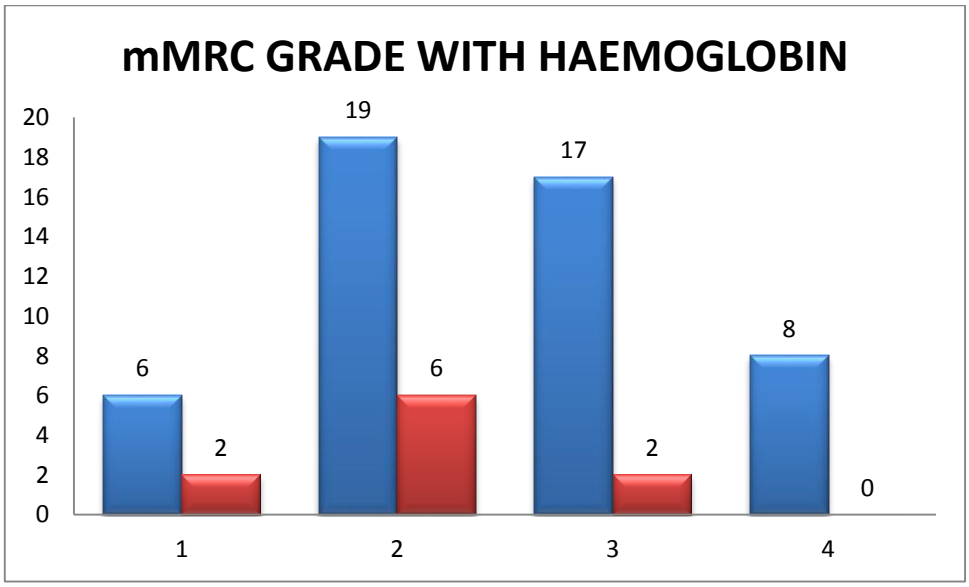


Haemoglobin of the patient was compared with Mill workers.Among the mill workers 71% had anaemia and 64% in non –mill workers. Although this association was considerable but it was not statistically significant.

TABLE 33: m MRC GRADING vs HAEMOGLOBIN

m MRC GRADING	HAEMOGLOBIN	
	ANEMIC	NORMAL
1	6	2
2	19	6
3	17	2
4	8	0

CHART 33: m MRC GRADING vs HAEMOGLOBIN



Haemoglobin of the patient was compared with m MRC GRADING.

Patients with increased Grades of Dyspnea had more risk for anemia and vice versa.

DISCUSSION

This clinical observational study was undertaken to investigate the pattern and magnitude of Hematological parameters in the relationship with the Severity of the disease. The study consisted of 60 subjects, among them 21 Patients had mild COPD, 23 Patients with moderate COPD, 9 Patients had severe COPD and 7 Patients had very severe COPD.

All the individuals in different groups were subjected to detailed History and Physical examination. Lung function parameters were assessed with the help of spirometer. All patients underwent Haematological parameters with particular reference to Hb%, MCV, MCH, MCHC, HAEMATOCRIT, PERIPHERAL SMEAR along with routine tests.

The aim of the present study was to assess the relationship between Physical examination, Hematological parameters and the severity of COPD.

1. COMPARISON OF AGE DISTRIBUTION WITH OTHER STUDIES

STUDIES	MEAN AGE
Behrendt et al.	42.2
Kamat SR et al	50
Our study	44

The mean age of present study population was 44 years which was comparable to Behrendt et al. study.

2. COMPARISON OF SEX DISTRIBUTION WITH OTHER STUDIES

STUDIES	MALE	FEMALE
Behrendt et al.	30.83%	69.17%
Mahesh et al.	11.54%	88.46%
Kamat SR et al	88.8%	11.2%
Our study	80%	20%

In the our study, males account for **80%,** and females account for **20%** with a Male : Female ratio of **4:1**

3. COMPARISON OF PLACE OF LIVING WITH OTHER STUDIES

STUDIES	URBAN	RURAL
Celli B et al.	58.80%	46.20%
Goel S et al.	27.27%	72.73%
Our study	80%	20%

In our study 80% patients were from Urban area .

4. COMPARISON OF DURATION OF ILLNESS WITH OTHER STUDIES

DURATION OF ILLNESS	Thiruvengadam KV (N=30)	Kamat SR (N=50)	OUR STUDY (N=60)
0- 5 yrs	33.3	40	28
6-10 yrs	43.3	40	47
>10 yrs	23.3	20	25

.

The duration of illness in all the study group showed more patients belonging to 6-10 years of duration.

5. PREVALENCE OF SMOKERS AND NONSMOKERS

STUDIES	SMOKERS	NON SMOKERS
Thiruvengadam KV et al.	87%	13%
OUR STUDY	57%	43%

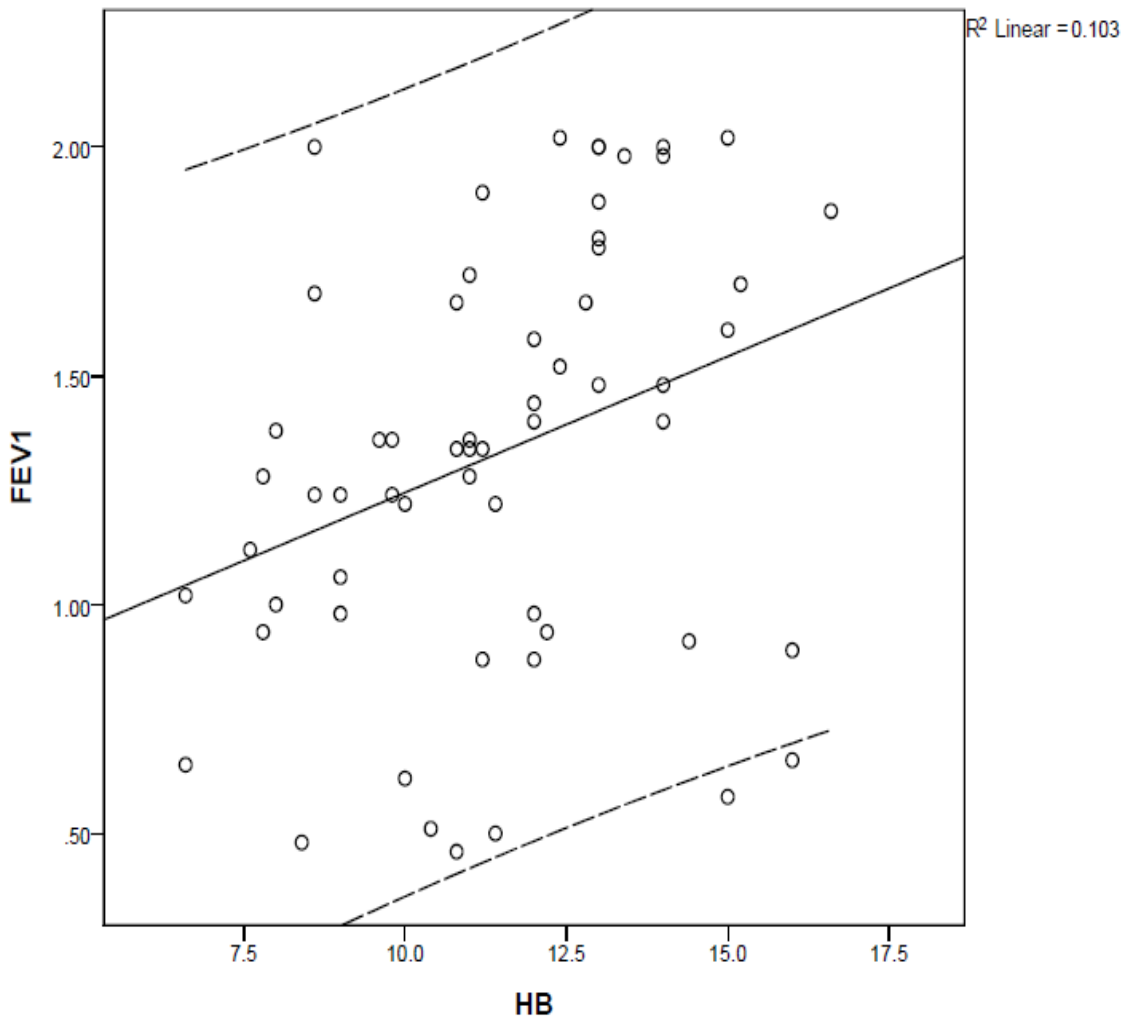
Smoking is being very important risk factor for COPD seen in most of the patients who develop COPD. In the present study 71% males were smokers, when compared with the Thiruvengadam KV et al. study group, who also had smoking history in all males. Among the smokers 73% had anemia and in Non- smokers 61% had anemia. Although this association was considerable but it was not statistically significant with P value of 0.322

6. COMPARISON OF SYMPTOMS WITH OTHER STUDIES

SYMPTOMS	Behrendt et al	Mahesh et al	Our study
COUGH	11.55%	100%	100%
EXPECTORATION	7.05%	100%	100%
WHEEZE	25.57%	90.9%	63%

In our study, cough and expectoration were predominant symptoms, Followed by wheeze. Symptom profile is comparable with Mahesh et al. study.

7. COMPARISON OF FEV1 WITH HAEMOGLOBIN



There is significant positive correlation between FEV1 and Haemoglobin with r=0.322 at P = 0.12 < 0.05 Level.

8. Among the patients studied 58% had exposure to cotton mill industry.Among them 71% had anemia.

9. Patient with increased levels of Dyspnea (m MRC Grading) had more risk for anemia

Grade 2 Dyspnea - 76% had anemia

Grade 3 Dyspnea – 89% had anemia

Grade 4 Dyspnea - 100% had anemia.

CONCLUSION

1. The age of the patients being studied ranged between 30 yrs to 70 yrs and the majority of the patients were in the age group of **41-50 years**.
2. In our study, out of 60 patients 48 (80%) were males and 12 (20%) were females. The Male:Female ratio of this study was **4:1**.
3. In our study, the duration of illness was more in patients with severe COPD as compared to the mild and moderate COPD group. This shows that the severity of COPD increases with the duration of illness.
4. In our study Post bronchodilator FEV1 results showed that 21 patients **(35%)** had GOLD CRITERIA of mild disease who had a mean of 1.82, 23 patients **(38%)** had moderate disease who had a mean of 1.24, 9 patients **(15%)** had severe disease who had a mean of 0.99 and 7 patient **(11%)** had very severe disease who had a mean of 0.54.
5. The most common symptoms were cough and expectoration , Present in all the 60 patients (100%). The most common sign observed was tachypnea in 42 (70%) patients.
6. In our study out of 60 (100%) patients studied chest x ray shows Chronic bronchitis in 15 patients , Chronic bronchitis with Emphysema in 16 patients , Emphysema in 24 patients and was Normal in 5 patients
7. Among the Patients 57% were smokers. 71% of male patients had a history of smoking for 5yrs or more. Among smokers 73% had anemia and in Non-smokers 61% had anemia.

8. In our study 56% of Patients had anemia.Among them 70% had Normocytic Normochromic Anemia and the remaining 30% had Microcytic Hypochromic Anemia.
9. The Prevalence of anemia was more common with increased duration of illness.Duration of illness 6-10 yrs had 57% risk for anemia and > 10 yrs had 87% risk of anemia.
10. The prevalence of anemia was more common in Cottton mill workers with 71% and 64% in non –mill workers.
11. The prevalence of anemia was more common in Patients with m MRC grade 3 and 4 dyspnoea.
12. In our study the fall in FEV1 was associated with fall in Haemoglobin percentage and vice versa.

SUMMARY

This study was conducted to detect the haematological manifestations in relation to FEVI in COPD Patients.60 Patients were included in this study.

Results from the study showed Patients with decreased FEV1, having prolonged duration of illness and increasing grades of dyspnea had high risk for anemia. Patients with exposure to Cotton Mill dust and with the history of smoking had increased risk for anemia, but its association was not statistically significant.

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PROFORMA

NAME OF THE PATIENT:

AGE: **SEX:**

OCCUPATION:

ADDRESS:

EDUCATION:

MARITAL STATUS:

INCOME:

DATE OF ADMISSION:

DATE OF EXAMINATION:

IP NUMBER: **OP NUMBER:**

PRESENTING COMPLAINTS: **DURATION**

1. Cough

2. Breathlessness

3. Sputum

4. Hemoptysis

5. Weight loss

6. Weakness and fatigue

9. Wheeze

10. Fever

11. Chest pain

12. Swelling of lower limbs

A) HISTORY OF PRESENTING COMPLAINTS:

1. Cough :

Duration

Dry/productive

Persistent/paroxysmal

Postural variation- Present/Absent

Diurnal variation - Present/Absent

Seasonal variation- Present/Absent

Progression- Increasing/decreasing/persisting

Any triggering factors- Smoke/cold/pollen/infection/dust

Relation to occupation- Increased at work/not

2. Breathlessness :

Duration

Onset- Sudden/insidious

Progression- increasing/deteriorating/static

Persistent/paroxysmal

Relation to exertion- present/absent

Seasonal variation- present/absent

Precipitating factors- present/absent

Any triggering factors- Smoke/cold/pollen/infection/dust

Orthopnoea/trepopnoea/platypnoea/PND

3. Sputum:

Colour

Quantity minimal/ moderate/ copious

Consistency

Postural variation Yes/ No

Diurnal variation Yes/ No

Foul smell-present/absent

4. Weight loss: Percentage of loss of weight:

Time period:

5. Chest pain :

Duration

Onset- Sudden/insidious

Location.

Nature of pain- pleuritic/anginal/other types

Radiationf.

Aggravating factors- deep inspiration/exertion/cough/any other

Relieving factors.

Duration of each episode

6. Wheezing :

Duration.

Precipitating factors.

Duration of attack

Seasonal variation- Present/Absent

7. Hemoptysis: Present/Absent

If present- Streaky/frank hemoptysis/pink frothy

8. Fever :

Onset- sudden/insidious

Type- continuous /intermittent/remittent

Accompanied by chills / rigors / sweating

9. Swelling of lower limbs:

Onset- sudden/insidious

Duration.

Pain- present/absent

Diurnal variation- present/absent

Postural variation- present/absent

10. Any other symptoms; Details:

B) Past history:

History of similar complaints

H/O tuberculosis/ asthma/ /allergy/epilepsy/nasal polyps/sinusitis/cardiac illness/ diabetes mellitus/hypertension/ repeated respiratory infections in childhood

History of exacerbations:

Number of exacerbations:

Hospitalization was required or not: Yes/ No

Duration of hospitalization:

C) Family history:

History of Asthma/tuberculosis/allergic disease.

No. of family members smoking.

Exposure to smoke (passive smoking) during infancy and childhood.

D) Personal history:

Diet: veg/ nonveg/ mixed

Sleep: Sound/ disturbed

Snoring- present/absent

Early morning headache- present/ absent

Appetite: Normal/decreased/increased

Bowel habits: regular habits/ constipated/loose stools

Habits:

Smoking: Yes/No

ETS or Passive Smoking exposure: Yes/ No

If Yes:

At Home/ work place

Duration of exposure: <3hrs/ >3hrs per day

Years of exposure:

Exposure to bio mass fuels at home or work place: yes/no

Duration of exposure: nature of fuel:

No. of years exposure:

Tobacco chewing: No. of times/day-

Duration

Snuff inhalation: yes/no

Alcohol: Quantity-

Duration- Type-

Occupational exposure to dust/fumes/vapors/smoke/chemicals.

Nature of exposure

Duration of exposure per day: hours per week

Place of living: urban/ rural

Exposure to atmospheric air pollution: present/ absent

E) Menstrual history: regular/irregular.

F) Treatment history: h/o taking any steroids, any beta 2 agonist or any theophylline derivative.

2. A) GENERAL PHYSICAL EXAMINATION:

Comfortable/dyspnoeic

Built: well built/moderate built/poorly built

Nourishment: well nourished/moderate/poorly

Weight:

Height:

Body mass index:

B) Vital signs

Pulse:

Rate- / min

Rhythm-

Volume-

Condition of the Vessel wall:

Blood Pressure: mm/Hg

Respiration: Rate-

Rhythm

Type

Temperature: °C

Pallor: Present/absent

Cyanosis: Present/absent

Central/ peripheral/mixed

Clubbing: present/absent degree

Koilonychia/platynychia: Present/absent

Icterus: Present/absent

Lymphadenopathy: Present/absent

Oedema: Present/absent

Any other significant signs-

3. SYSTEMIC EXAMINATION:

RESPIRATORY SYSTEM

A) Upper respiratory system:

B) Lower respiratory tract

INSPECTION:

a. Position of the trachea: Central / left / right.

b. Shape of the chest: Normal/ barrel chest/ pigeon chest/ pectus excavatum / kyphoscoliosis / Harrison's sulcus /any other chest deformity.

c. Asymmetry: Bulging/ retraction.

d. Respiratory movements: Normal

Diminished- bilateral / unilateral / localized

e. Respiratory sounds heard: respiratory stridor/ respiratory wheeze

f. Position of the apical impulse:

g.Visible veins over the chest: present/absent

h.Special features to be noted

i.Tracheal descent with inspiration

j.Excessive use of scalene and sternocleidomastoid muscle

PALPATION:

a. Position of the trachea: central/right/left.

b. Position of the apical impulse:

c. Movements of the chest wall- normal / decreased

d. Measurements: during Deep inspiration - cms

Deep expiration - cms

A P diameter - cms

Transverse diameter- cms

Chest expansion- cms

Hemithorax Right- cms

Left- cms

e. Any palpable rhonchi or rub –

f. Vocal fremitus- Normal / increased / decreased

Areasg.

Tenderness over the chest- areas

PERCUSSION:

a. Percussion note: Normal / impaired / hyper resonant / woody dullness/ stony dullness

b. Liver dullness:

c. Cardiac dullness- Normal/ obliterated.

d. Tidal percussion - Normal / diminished

AUSCULTATION:

a. Breath sound: type: NVBS / bronchial / vesicular breath sounds with prolonged expiration.

b. Air entry: Normal / diminished / absent

c.Rhonchi: Present / absent

d.Crepitation: Present / absent

Inspiratory/expiratory

Fine/coarse

e.Pleural rub: Present / absent

f.Vocal resonance: Normal / increased /decreased

CARDIOVASCULAR SYSTEM:

ABDOMEN:

NERVOUS SYSTEM:

4. INVESTIGATION

BLOOD: Hb%-

TC DC

ESR FBS

PPBS Blood Urea

Serum creatinine

URINE: Albumin- Sugar-

Microscopy-

SPUTUM: Quantity per day-

Colour- Odour-

Blood: present/ absent

Microscopy: Gram stain

AFB stain

CHEST RADIOGRAPH: Report –

SPIROMETRIC PARAMETERS: Pre bronchodilator Post bronchodilator

FVC:

FEV1:

FEV1/FVC:

STATISTICAL METHODS

The collected data was analysed with SPSS 16.0 version. To describe about the data descriptive statistics frequency analysis, percentage analysis, mean and S.D were used. To associate the various clinical variables Chi-Square test was done.In the above statistical tool the probability value .05 is considered as significant level.

KEY TO MASTER CHART

S.NO-SERIAL NUMBER

ETS-ENVIRONMENTAL TOBACCO EXPOSURE

BMI-BODY MASS INDEX

JVP-JUGULAR VENOUS PULSE

m MRC-MODIFIED MEDICAL RESEARCH COUNCIL

HB –HAEMOGLOBIN

MCV-MEAN CORPUSCULAR VOLUME

MCH-MEAN CORPUSCULAR HAEMOGLOBIN

MCHC- MEAN CORPUSCULAR HAEMOGLOBIN

CONCENTRATION

SEX

1 = MALE

2= FEMALE

AGE

1= 21-30 YRS

2= 31- 40 YRS

3= 41-50YRS

4= 51-60 YRS

PLACE OF LIVING

1= URBAN

2= RURAL

BMI

1= < 18 kg/sqcm

2= 18-23 kg/sqcm

3= 23-24.9 kg/sqcm

4= > 25 kg/sqcm

GOLD CRITERIA SEVERITY OF COPD

1= mild

2= moderate

3= severe

4= very severe

SMOKING

1= yes

2= no

PACK YEARS

1= nil

2= 20-30 pack years

3= 31-40 pack years

4= 41-50 pack years

5= 51-60 pack years

MILL WORKER /ETS /BIOMASS FUEL

1= yes

2= no

PRESENTING SYMPTOMS

COUGH	EXPECTORATION	DYSPNOEA	WHEEZE	FEVER	FATIGUE	WEIGHT LOSS

1= yes

2= no

PRESENTING SIGNS

TACHYPNOEA	CYANOSIS	CLUBBING	ACCESSARY MUSCLE	PEDEL EDEMA	JVP	LOUD P2	PARASTERNAL HEAVE	HEPATOMEGALY	CREPITATIONS	RHONCHI

1= yes

2= no

CHEST EXPANSION

1= >5cm

2= 3-5cm

3= <3cm

m MRC GRADING

1= 1

2= 2

3= 3

4= 4

Fev1

1 = mild

2= moderate

3= severe

4= very severe

Chest x ray

1= Normal

2= Bronchitis

3= Emphysema

4= Emphysema +Bronchitis

PERIPHERAL SMEAR

1= Normal

2= Normocytic normochromic

3= Microcytic hypochromic

BIO DATA								RISK FACTORS					PRESENTATION OF SYMPTOMS					
S.NO	NAME	SEX	AGE	PLACE OF LIVING	DURATION OF ILLNESS	BMI	GOLD SEVERITY OF COPD	SMOKING	PACK YEARS	MILL WORKERS	ETS	BIOMASS FUEL	COUGH	EXPECTORATION	DYSPNOEA	WHEEZE	FEVER	FATIGUE
1	GANESAN	1	3	1	1	2	1	2	1	1	2	2	1	1	1	1	1	2
2	ARUMUGHAM	1	3	1	1	4	1	1	2	1	2	2	1	1	2	2	2	2
3	KARUPPAN	1	3	1	1	4	1	2	1	1	1	2	1	1	1	1	2	2
4	PAYCHI	2	3	2	2	2	1	2	1	1	2	1	1	1	2	2	2	2
5	KALIAMMA	1	2	1	2	2	1	2	1	2	1	2	1	1	1	1	2	2
6	MURUGAN	1	3	1	1	3	1	1	3	2	2	2	1	1	2	2	2	2
7	LAKSHMI	2	3	2	1	3	1	2	1	2	1	1	2	2	1	2	2	2
8	SUNDARAMORTHY	1	5	1	2	3	1	2	1	2	2	2	1	1	2	2	2	2
9	VAIRAMUTHU	1	4	1	1	2	1	1	3	2	2	2	1	1	1	1	1	2
10	KANNAN	1	4	1	1	4	1	2	1	1	1	2	1	1	1	1	2	2
11	PASUMPON	1	5	1	1	2	1	1	3	2	2	2	2	2	1	1	1	1
12	PAYCHIMUTHU	1	5	1	1	2	1	2	1	2	1	2	2	2	1	2	2	2
13	BHARATHIRAJAN	1	4	1	2	2	1	1	3	1	2	2	1	1	2	2	1	2
14	VENKATACHALAM	1	4	1	2	2	1	1	2	1	2	2	2	2	1	2	2	2
15	ARIVAZHAN	1	3	1	1	2	1	1	2	2	2	2	1	1	2	2	1	2
16	MANIYAN	1	4	1	2	2	1	2	1	2	1	2	2	2	1	2	2	1
17	MAHALI	1	5	1	1	2	1	2	1	1	2	2	1	1	1	1	2	2
18	NATARAJAN	1	3	1	2	2	1	1	3	1	1	2	1	1	1	2	1	1
19	PANDIYARAJAN	1	4	1	2	2	1	2	1	1	2	2	1	1	1	1	1	1
20	KUPPUSAMY	1	5	1	2	2	1	1	3	1	2	2	1	1	1	2	2	1
21	GUNAVATHI	2	3	2	3	2	1	2	1	2	1	2	1	1	1	2	2	1
22	ILANGO	1	3	1	3	2	2	1	2	1	2	2	2	2	1	2	2	2
23	MARIMUTHU	1	3	1	1	2	2	2	1	1	1	2	1	1	1	2	2	2
24	MURUGESAN	1	4	1	2	3	2	1	4	1	2	2	2	2	1	2	2	2
25	SIVARAJ	1	3	1	3	3	2	1	3	2	2	2	1	1	1	2	2	2
26	PARTHIBAN	1	5	1	3	3	2	2	1	2	2	2	2	2	1	2	2	2
27	SUMANGALI	2	3	2	3	2	2	1	3	2	1	2	1	1	2	2	2	2
28	THANGAVEL	1	3	1	2	2	2	1	3	2	2	2	2	2	1	2	2	2
29	VELLINGIRI	1	3	1	2	2	2	1	3	2	1	2	1	1	2	1	2	2
30	RUBAN	1	4	1	2	2	2	2	1	1	2	2	2	2	1	1	2	2
31	PADMANABAN	1	4	1	2	2	2	1	2	1	2	2	1	1	2	1	2	2
32	SUNDARAM	1	3	1	3	2	2	2	1	1	1	2	1	1	1	2	2	2
33	MUTHUCHELVI	2	3	2	2	1	2	2	1	2	1	1	1	1	2	2	2	2
34	ALAGUSMY	1	3	1	2	1	2	1	4	1	2	2	1	1	1	2	2	2
35	RANGARJ	1	3	1	2	1	2	1	5	1	2	2	2	2	1	2	2	2
36	YUSUF	1	4	1	1	1	2	1	5	1	2	2	1	1	1	2	2	2
37	SELVI	2	4	2	2	2	2	2	1	1	2	1	1	1	2	2	2	2
38	KARTHIKEYAN	1	3	1	1	1	2	1	3	2	2	2	1	1	1	1	2	2
39	THULASIMANI	2	4	2	2	2	2	2	1	2	1	1	1	1	2	2	1	2
40	SURESHBABU	1	3	1	1	2	2	1	3	1	2	2	1	1	2	2	2	2
41	CHINAMMA	2	4	2	2	2	2	2	1	2	2	1	1	1	2	2	2	2
42	KANMANI	2	3	2	1	1	2	2	1	1	1	2	1	1	2	2	1	2
43	GURUSAMY	1	4	1	2	2	2	1	5	1	2	2	2	2	1	2	1	2
44	PECHIYAMMAL	2	5	2	2	1	2	2	1	2	1	1	1	1	2	2	1	2
45	GEORGE	1	4	1	2	2	3	1	3	1	2	2	2	2	1	2	1	2
46	PAULRAJ	1	5	1	3	1	3	1	3	1	1	2	1	1	2	1	1	1
47	MANICKAM	1	3	1	3	2	3	1	5	1	2	2	2	2	1	2	1	1
48	DHIVAGARAN	1	3	1	2	2	3	1	3	1	1	2	1	1	2	2	1	1
49	CHELLADURAI	1	3	1	3	2	3	1	3	1	2	2	2	2	1	1	1	2
50	PALANI	1	3	1	3	2	3	2	1	2	2	2	2	2	1	1	2	2
51	RANGASAMY	1	4	1	3	1	3	1	4	2	1	2	1	1	2	1	2	2
52	JAYAKUMAR	1	3	1	1	1	3	1	4	2	2	2	2	2	1	1	2	1
53	SENTHILVEL	1	3	1	2	2	3	2	1	2	2	2	1	1	2	2	2	2
54	VENKATACHALAM	1	5	1	2	2	4	1	4	1	1	2	2	2	1	2	2	1
55	SHANMUGAVEL	1	5	1	2	1	4	1	4	1	1	2	1	1	2	2	1	1
56	KANNIAMMA	2	4	2	2	2	4	2	1	1	1	2	1	1	1	2	1	1
57	SUBRAMANI	1	4	1	3	1	4	1	3	2	1	2	2	2	1	2	1	2
58	VIMALA	2	4	2	3	1	4	2	1	1	2	2	1	1	2	2	1	2
59	KANAGARAJ	1	4	1	3	1	4	1	2	1	2	2	1	1	1	2	1	2
60	PARAMAN	1	4	1	3	1	4	1	3	1	2		1	1	1	2	1	2

BIO DATA			PRESENTATION OF SIGNS														HAEMATOLOGICAL						
S.NO	NAME	WEIGHT LOSS	TACHYPNOEA	CYANOSIS	CLUBBING	ACCESSARY MUSCLE	PEDEL EDEMA	JVP	LOUD P2	PARASTERNAL HEAVE	HEPATO MEGALY	CREPITATIONS	RHONCHI	CHEST EXPANSION	mMRC GRADE	FEV1	CHEST X-RAY	HB %	HCT	MCV	MCH	MCHC	PERIPHERAL SMEAR
1	**GANESAN**	1	1	2	2	2	2	2	2	2	2	1	1	1	1	2.02	2	12.4	45.9	81	28	32.4	1
2	**ARUMUGHAM**	2	1	2	2	2	2	2	2	2	2	1	1	1	3	1.98	2	13.4	43.5	86	28.6	34.4	1
3	**KARUPPAN**	2	1	2	2	2	2	2	2	2	2	1	1	1	3	1.66	2	12.8	40.6	95	30	34.6	1
4	**PAYCHI**	2	1	2	2	2	2	2	2	2	2	1	1	1	2	1.6	2	14.4	43.6	88	31	35.4	1
5	**KALIAMMA**	2	2	2	2	2	2	2	2	2	2	1	1	1	1	2	2	15	42.2	96	32	32	1
6	**MURUGAN**	2	1	2	2	2	2	2	2	2	2	1	1	1	1	1.7	1	8.6	43	56	25	28.8	3
7	**LAKSHMI**	2	1	2	2	2	2	2	2	2	2	1	1	1	1	1.86	2	11.2	44	67	24.6	27.8	3
8	**SUNDARAMORTHY**	2	2	2	2	2	2	2	2	2	2	1	1	1	1	1.72	3	15.2	45.2	90	30	36	1
9	**VAIRAMUTHU**	2	2	2	2	2	2	2	2	2	2	1	2	1	1	1.66	2	16.6	43.6	89	31	32.4	1
10	**KANNAN**	2	2	2	2	2	2	2	2	2	2	2	2	1	2	1.8	2	11	40.2	90.4	29.6	32.8	1
11	**PASUMPON**	2	2	2	2	2	2	2	2	2	2	2	2	1	3	1.52	2	10.8	40.4	70	23	27.6	3
12	**PAYCHIMUTHU**	1	2	2	2	2	2	2	2	2	2	2	2	1	2	2	3	13	40.6	89	29.4	32	1
13	**BHARATHIRAJAN**	1	2	2	2	2	2	2	2	2	2	2	1	1	2	2.02	2	12.4	40.8	89	30.4	33	1
14	**VENKATACHALAM**	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	1	13	43.4	88	30.2	34	1
15	**ARIVAZHAN**	2	1	2	2	2	2	2	2	2	2	2	2	2	1	2	1	15	43.8	92.4	31.8	35.4	1
16	**MANIYAN**	2	1	2	2	1	2	2	2	2	2	2	2	2	3	1.98	2	14	44	93	28.8	35.6	1
17	**MAHALI**	2	1	2	2	1	2	2	2	2	2	1	2	2	3	1.78	3	13	40	90.2	29.2	35.8	1
18	**NATARAJAN**	2	1	2	2	2	2	2	2	2	2	1	1	2	2	1.9	1	14	40.6	93	32	34.2	1
19	**PANDIYARAJAN**	2	1	2	2	2	2	2	2	2	2	1	1	2	3	1.88	3	13	40.7	87.4	31.6	34.6	1
20	**KUPPUSAMY**	2	1	2	2	2	2	2	2	2	2	2	1	2	2	1.68	1	11.2	40.2	82.7	32	34.8	2
21	**GUNAVATHI**	2	1	2	2	1	2	2	2	2	2	2	1	2	2	1.58	4	9	42.4	69	22.8	30.2	3
22	**ILANGO**	2	2	2	2	1	2	2	2	2	2	2	1	1	3	0.9	3	13	43.8	88.8	32	34.8	1
23	**MARIMUTHU**	2	2	2	2	1	2	2	2	2	2	2	1	2	3	1.22	3	8.6	44.4	74	24.2	31.2	3
24	**MURUGESAN**	2	2	1	2	1	2	2	2	2	2	2	1	2	2	1.34	2	12	43.2	92.6	31.4	32	1
25	**SIVARAJ**	2	2	1	2	1	2	2	2	2	2	2	1	2	2	1.22	2	16	42.5	88	32	32.8	1
26	**PARTHIBAN**	2	2	2	2	1	2	2	2	2	2	1	1	2	2	1.4	2	11.4	42.6	90	32	32.8	2
27	**SUMANGALI**	2	1	2	2	1	2	2	2	2	2	1	1	2	2	1.36	4	8	42.8	76.8	20.8	28.8	3
28	**THANGAVEL**	2	1	2	2	1	2	2	2	2	2	1	2	2	2	1.38	3	11.2	42.6	80.4	32.4	32.4	2
29	**VELLINGIRI**	2	1	2	2	2	2	2	2	2	2	1	2	2	2	1.4	4	10	44	88	32	34.2	2
30	**RUBAN**	1	1	1	2	1	2	2	2	2	2	2	2	2	2	1.48	3	12	43.6	92.5	32	34.8	1
31	**PADMANABAN**	2	2	1	2	2	2	2	2	2	2	2	2	2	2	1.44	3	9.8	42.8	77	2O.4	29.6	3
32	**SUNDARAM**	1	2	1	2	2	2	2	2	2	2	2	2	2	2	1.06	3	8	42.7	88.5	31	36	2
33	**MUTHUCHELVI**	2	2	2	2	1	2	2	2	2	2	1	2	2	2	0.94	3	6.6	44	75	23.8	30	3
34	**ALAGUSMY**	2	1	1	2	1	2	2	2	2	2	1	2	2	2	0.88	4	14	43.4	90.2	28	35.4	1
35	**RANGARJ**	2	1	2	2	1	2	2	2	2	2	1	2	2	2	0.98	3	13	45	91.4	32	36.2	2
36	**YUSUF**	2	1	1	2	2	2	2	2	2	2	1	1	2	2	1.28	3	12	44.2	91	32.4	34.6	1
37	**SELVI**	1	1	2	2	2	2	2	2	2	2	1	1	2	3	1.36	4	16	44.8	93.6	28.8	34.6	1
38	**KARTHIKEYAN**	1	1	2	2	2	2	2	2	2	2	2	1	2	2	1.34	3	9	44.2	86.8	28	31.8	2
39	**THULASIMANI**	2	2	2	2	2	2	2	2	2	2	2	1	2	3	1.12	3	15	42.2	95	28	34.4	1
40	**SURESHBABU**	2	2	2	1	2	2	2	2	2	2	1	1	2	3	1.24	3	12.2	43.6	87.6	29.4	34.2	1
41	**CHINAMMA**	1	1	1	1	2	2	2	2	2	2	1	1	2	2	1.28	4	10	42.6	86.7	30.4	28.8	2
42	**KANMANI**	1	1	2	2	1	2	2	2	1	1	1	1	2	2	1.24	4	11.4	44.4	89.2	32	31.8	2
43	**GURUSAMY**	2	1	2	2	1	2	2	1	1	1	1	1	2	2	1.48	4	12	43	90.6	32	35	1
44	**PECHIYAMMAL**	1	1	1	2	1	2	2	1	1	1	1	1	2	2	1.36	4	10.8	43.6	89.6	31.4	31.4	2
45	**GEORGE**	1	1	1	1	2	2	2	1	2	2	1	2	3	3	1.34	4	12	43.8	89	30.4	34	1
46	**PAULRAJ**	1	1	1	1	2	2	2	1	2	2	1	2	3	4	1.24	3	11	43.3	88.8	30.6	31.4	2
47	**MANICKAM**	1	1	1	1	1	1	2	2	2	2	1	1	3	3	1.02	4	11	43.6	88.4	30.8	32	2
48	**DHIVAGARAN**	2	1	1	1	1	1	1	2	2	2	1	1	2	3	0.94	3	10.8	46.8	84	29.8	32	2
49	**CHELLADURAI**	1	1	1	2	1	1	1	1	2	2	1	1	3	3	0.92	4	7.6	45	82.2	28.4	32.8	2
50	**PALANI**	2	1	1	2	1	1	1	1	2	2	1	1	2	4	0.88	3	9.8	43.8	88	28.4	32.4	2
51	**RANGASAMY**	1	1	1	2	1	1	1	1	1	2	1	1	3	4	0.98	4	7.8	44.8	92	28.8	32.4	2
52	**JAYAKUMAR**	2	1	1	1	2	1	1	2	2	2	1	1	2	3	1	3	8.6	44.2	92.4	28.8	32	2
53	**SENTHILVEL**	1	1	1	1	1	1	1	2	2	2	1	1	3	4	0.65	3	14	42.4	94	29.6	34	I
54	**VENKATACHALAM**	2	1	1	1	1	1	1	2	2	2	1	1	3	4	0.66	4	9.6	43	86.4	29.8	34.4	2
55	**SHANMUGAVEL**	1	1	1	2	1	1	1	2	2	2	1	1	3	3	0.58	3	11	43.4	88	29.4	34.8	2
56	**KANNIAMMA**	1	1	1	2	1	1	1	1	2	2	1	1	3	4	0.62	2	10.4	45.2	86.2	28.4	35	2
57	**SUBRAMANI**	1	1	1	2	1	1	1	1	2	2	1	1	3	3	0.5	3	9	44.2	89.8	28.4	34	2
58	**VIMALA**	1	1	1	2	1	1	1	1	2	2	1	1	3	4	0.46	4	8.4	44.4	94	28.4	32.4	2
59	**KANAGARAJ**	1	1	1	2	1	1	1	1	2	2	1	1	2	4	0.51	4	6.6	43.2	92	29.4	32.2	2
60	**PARAMAN**	1	1	1	2	1	1	1	1	2	2	1	1	3	3	0.48	3	7.8	42.2	86	29.8	32.4	2

INFORMED CONSENT

Department of General Medicine

COIMBATORE MEDICAL COLLEGE, COIMBATORE

Principal Investigator : Dr. M. Gowri Sankar

Research Guide : Dr.S.Usha.M.D

Organization : Department of General Medicine

Informed consent: I have been invited to participate in research project titled "**HAEMATOLOGICAL MANIFESTATIONS IN CHRONIC OBSTRUCTIVE LUNG DISEASE IN RELATION TO FEV1 *IN COIMBATORE MEDICAL COLLEGE HOSPITAL***", I understand, I will be answering a set of questionnaire, undergo physical examination, investigation and appropriate treatment. I also give consent to utilize my personal details for study purpose and can be contacted if necessary.

I am aware that I have the right to withdraw at any time which will not affect my medical care.

Name of the participant:

Signature:

Date:

ஒப்புதல் படிவம்

பெயர் :

பாலினம் : வயது:

முகவரி :

அரசு கோவை மருத்துவக் கல்லூரியில் பொது மருத்துவத் துறையில் பட்ட மேற்படிப்பு பயிலும் மாணவர் மேற்கொள்ளும் **"மூச்சுக்குழல்கள் அடைப்பு காரணமாக முதல் நொடியில் வெளித்தள்ளும் மூச்சின் அளவைப் பொறுத்தும் மற்றும் நுரையீரலின் மாறுதலினாலும் இரத்தத்தில் ஏற்படும் விளைவுகள்"** குறித்த ஆய்வில் செய்முறை மற்றும் அனைத்து விவரங்களையும் கேட்டுக் கொண்டு எனது சந்தேகங்களை தெளிவுபடுத்திக் கொண்டேன் என்பதை தெரிவித்துக் கொள்கிறேன்.

நான் இந்த ஆய்வில் முழு சம்மதத்துடனும், சுய சிந்தனையுடனும் கலந்து கொள்ள சம்மதிக்கிறேன்.

இந்த ஆய்வில் என்னுடைய அனைத்து விபரங்கள் பாதுகாக்கப்படுவதுடன் இதன் முடிவுகள் ஆய்விதழில் வெளியிடப்படுவதில் ஆட்சேபனை இல்லை என்பதை தெரிவித்துக் கொள்கிறேன். எந்த நேரத்திலும் இந்த ஆய்விலிருந்து நான் விலகிக் கொள்ள எனக்கு உரிமை உண்டு என்பதையும் அறிவேன்.

இடம்: கையொப்பம் / ரேகை

நாள்: