

**“TO STUDY THE PREVALENCE, SOCIODEMOGRAPHIC,
CLINICO-RADIOLOGICAL PROFILE AND ALSO
CORRELATION OF COPD ASSESSMENT TEST (CAT) AND
DEGREE OF AIRFLOW OBSTRUCTION IN CHRONIC
OBSTRUCTIVE PULMONARY DISEASE IN FEMALES
ATTENDING TERTIARY CARE HOSPITAL”**

**Dissertation submitted to The Tamil Nadu Dr.M.G.R. Medical
University in partial fulfilment of the requirements for the degree of**

**Doctor of Medicine (M.D) in
Tuberculosis and Respiratory Diseases
Branch – XVII**

**Institute of Thoracic Medicine,
Madras Medical College &
Rajiv Gandhi Government General Hospital**



**The Tamil Nadu Dr. M.G.R. Medical University
Chennai – 600032
Tamil Nadu
India**

April 2017

DECLARATION

This is to certify that the dissertation titled **“TO STUDY THE PREVALENCE, SOCIODEMOGRAPHIC, CLINICO-RADIOLOGICAL PROFILE AND ALSO CORRELATION OF COPD ASSESSMENT TEST (CAT) AND DEGREE OF AIRFLOW OBSTRUCTION IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE IN FEMALES ATTENDING TERTIARY CARE HOSPITAL”** is the bonafide work done by **Dr. RAJESWARL.P** during her **M.D (Tuberculosis and Respiratory Diseases)** course in the academic years 2015-2017, at the Institute of Thoracic Medicine and Rajiv Gandhi Government General Hospital – Madras Medical College, Chennai. This work has not previously formed the basis for the award of any degree.

**Prof. Dr. A.MAHILMARAN M.D.,
D.T.C.D**
Director, Institute of
Thoracic Medicine,
Professor and Head, Department of
Thoracic Medicine,
Rajiv Gandhi Government General
Hospital and Madras Medical College,
Chennai.

**Prof. Dr. M.K.MURALIDHARAN
M.S., M.Ch (Neurosurgery)**
The Dean,
Rajiv Gandhi Government General
Hospital and Madras Medical College,
Chennai.

DECLARATION BY THE GUIDE

This is to certify that the dissertation titled “**TO STUDY THE PREVALENCE, SOCIODEMOGRAPHIC, CLINICO-RADIOLOGICAL PROFILE AND ALSO CORRELATION OF COPD ASSESSMENT TEST (CAT) AND DEGREE OF AIRFLOW OBSTRUCTION IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE IN FEMALES ATTENDING TERTIARY CARE HOSPITAL**” is the Bonafide work done by **Dr. RAJESWARI** during her **M.D (Tuberculosis and Respiratory Diseases)** course in the academic years 2015-2017, at the Institute of Thoracic Medicine and Rajiv Gandhi Government General Hospital – Madras Medical College, Chennai, **under my guidance.**

Signature of the Guide,

Name and Designation of the Guide:

Prof. Dr.A.MAHILMARAN M.D., D.T.C.D.,

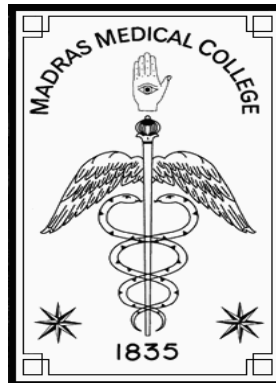
Director, Institute of Thoracic Medicine,

Professor and Head, Department of Thoracic Medicine,

Rajiv Gandhi Government General Hospital and Madras Medical

College, Chennai.

**MADRAS MEDICAL COLLEGE & RAJIV GANDHI
GOVERNMENT GENERAL HOSPITAL, CHENNAI – 600 003**



DECLARATION BY THE SCHOLAR

I hereby declare that the dissertation titled **“TO STUDY THE PREVALENCE, SOCIODEMOGRAPHIC, CLINICO-RADIOLOGICAL PROFILE AND ALSO CORRELATION OF COPD ASSESSMENT TEST (CAT) AND DEGREE OF AIRFLOW OBSTRUCTION IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE IN FEMALES ATTENDING TERTIARY CARE HOSPITAL”** submitted for the degree of **Doctor of Medicine (M.D) in Tuberculosis and Respiratory Diseases, Branch XVII** is my original work and the dissertation has not formed the basis for the award of any degree, diploma, associate ship, fellowship or other similar titles.

Place : Chennai

[DR.RAJESWARI.P]

Date :

ACKNOWLEDGEMENT

First and foremost I would like to thank the almighty for giving me the strength and courage to complete the task successfully.

My sincere thanks to **Prof. Dr.MURALIDHARAN M.D.**, Dean, Rajiv Gandhi Government General Hospital and Madras Medical College for allowing me to do this dissertation and utilize the Institutional facilities.

I am gratefully indebted to Director, Institute of Thoracic Medicine., Professor and Head, Department of Thoracic Medicine, Rajiv Gandhi Government General Hospital and Madras Medical College **Prof. Dr. A. Mahilmaran, M.D., D.T.C.D.**, for his invaluable guidance, advice and encouragement throughout the study.

I sincerely thank **Prof.Dr.O.R.Krishnarajasekhar, M.D.,D.T.C.D.**, Professor, Department of Thoracic Medicine, Rajiv Gandhi Government General Hospital and Madras Medical College, for sparing his precious time in guiding my dissertation writing and reviewing it.

My sincere gratitude also goes to **Prof. Dr.Kalpana, MD** Department of Radiology for her immense guidance and unwavering support for my study.

I specially thank **Dr. V.Sundar, M.D** and **Dr. N.Murugan, M.D** for guiding me during each and every step of my dissertation from subject selection to writing the dissertation.

I am bound by ties of gratitude to Assistant Professors **Dr.G.S.Vijayachandar, Dr.K.Veena, Dr.T.RangaRajan, Dr.P.ArulKumaran, Dr.Deepa Selvi, Dr.M.Hema, Dr.Anbarasi, Dr.Ammaiyappan and Dr.Arun Babu.**

I thank my husband **Mr.R.C.SENDHILKUMAR**, my sons **S.NITHIN CHELLIYA, S.KRISHNA CHELIAAH**, my mother in law **Mrs.C.SAROJA** and my parents **Mr. R.PARAMASIVAM** and **Mrs. MANONMANI** for motivating and encouraging me during each and every step of my dissertation, in every possible way. Because of their prayers, blessings and constant encouragement I was able to finish my dissertation in time.

I am very thankful to **Dr.Anand and Dr.Jayashree** who did all the statistical work in my study.

I am also grateful to all **Postgraduates** and **Technicians** in the Department of radiology for providing assistance and rendering timely help to complete my study.

I would like to thank my seniors for guiding me in doing my thesis, batch mates **Dr.Palaniappan, Dr.Priya** and **Dr.Manju Sara Oommen** who made do my dissertation and write it up in an interesting and joyful way. I would like to thank my juniors especially **Dr.Ramkumar, Dr.Sivakumar and Dr.Sridhar** for doing whatever help I have asked for, in completing my dissertation.

Last but not the least, I am profoundly grateful to all the patients, who were subjects of my study for their participation and co-operation.

ABBREVIATIONS

COPD	-	Chronic Obstructive Pulmonary DISEASE
WHO	-	World Health Organisation
PLATINO	-	Proyecto Latino Americano De Investigation En Obstruccion Pulmonar
FEV1	-	Forced Expiratory Volume in 1 second
HRT	-	Replacement Therapy
SGRQ	-	St. George's Respiratory Questionnaire
PM	-	Particulate Matter
FVC	-	Forced Vital Capacity
GOLD	-	Global initiative for chronic Obstructive Lung Disease
OR	-	Odds Ratio
CI	-	Confidence Interval
FFMI	-	Fat Free Mass Index
FFM	-	Fat Free Mass
BMI	-	Body Mass Index
CT	-	Computed Tomography
SALIA	-	Study on the influence of Air pollution on Lung function , Inflammation and Ageing SHS Second Hand Smoke
ACOS	-	Asthma COPD Overlap Syndrome
EPI-SCAN	-	Epidemiologic Study of COPD in Spain
CAT COPD	-	Assessment Test
LLN	-	Lower Limit of Normal
PH	-	Hypertension
ECLIPSE	-	Evaluation of COPD Longitudinally to Identify Predictive Surrogate End-points mMRC Modified Medical Research Council
PHQ 9	-	Patient Health Questionnaire 9
DSM	-	Diagnostic and Statistical Manual of Mental Disorders
FRC	-	Functional Residual Capacity
ATS	-	American Thoracic Society
6MWT	-	6 Minutes Walk Test
6MWD	-	6 Minutes Walk Distance
HRCT	-	High Resolution Computed Tomography

LAA	-	Low Attenuation Area
A Phenotype	-	Airway Predominant Phenotype
M Phenotype	-	Mixed Phenotype
E Phenotype	-	Emphysematous predominant phenotype
MS	-	Microsoft
SPSS	-	Statistical Package for Social Sciences
COMCOLD	-	COMorbidities in Chronic Obstructive Lung Disease

1. CONTENTS

Sl.No.	TITLE	Page No.
1.	INTRODUCTION	1
2.	REVIEW OF LITERATURE	3
3.	AIMS AND OBJECTIVES	25
4.	MATERIALS AND METHODS	27
5.	OBSERVATIONS AND RESULTS	45
6.	DISCUSSION	81
7.	CONCLUSION	96
BIBLIOGRAPHY		
ANNEXURES		
ABBREVIATIONS		
TURNITIN-PLAGIARISM SCREEN SHOT		
DIGITAL RECEIPT		
ETHICAL COMMITTEE APPROVAL ORDER		
CONSENT FORM		
PROFORMA		
MASTER CHART		

**TO STUDY THE PREVALENCE, SOCIODEMOGRAPHIC,
CLINICO-RADIOLOGICAL PROFILE AND ALSO
CORRELATION OF COPD ASSESSMENT TEST (CAT) AND
DEGREE OF AIRFLOW OBSTRUCTION IN CHRONIC
OBSTRUCTIVE PULMONARY DISEASE IN FEMALES
ATTENDING TERTIARY CARE HOSPITAL**

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a common preventable and treatable disease, is characterized by persistent airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases. Exacerbations and comorbidities contribute to the overall severity in individual patients. [1]

According to *WHO 2012*, Chronic Obstructive Pulmonary Disease is now the third leading cause of death next to ischemic heart diseases and stroke.[2] Several studies have shown that the prevalence of COPD is increasing faster among women. In developed and industrialised nations active smoking seems to play a major role and in developing countries exposure to biomass seems to be a major factor.

Non smokers with COPD are more often women proving genetic predisposition, environmental influences and greater vulnerability of women's lungs. Their lungs are more susceptible to damage from air pollution and smoking. Sexual dimorphism of the human immune response may be responsible for the gender differences in the disease.

Little is known about the biomass related phenotype because pathophysiology related to biomass smoke is less understood. *Rivera et al* in his autopsy found that lung of individuals exposed to biomass displayed more

bronchiolitis and emphysema.[3] Do living patients with COPD due to biomass will have similar findings is not known.

Women are more likely to be diagnosed with asthma instead of COPD. So, greater chances of underdiagnosis and receiving inadequate treatment for their condition. Women also report poor quality of life when compared with men, hence the urgent need for identification of COPD in women, the probable risk factors associated, most common presenting symptoms and their impact on life. Women report greater anxiety , more depressive symptoms, more dyspnea and chronic cough. They also have a poor quality of life, poor nutritional status, higher frequency of exacerbation. Osteoporosis, anxiety and depression were common in women with COPD. Women seem to be more sensitive to the adverse effects of beta 2 agonists.

Hence, we plan to study the epidemiology and presentation of COPD , its impact on physiologic and pathologic impairments ,the probable risk factors associated in the female patients presenting with chronic obstructive pulmonary disease.

REVIEW OF LITERATURE

“IS FEMALE GENDER DIFFERENT ?”

Underdiagnosis of COPD in women

Chapman k et al found that when hypothetical case of both man and a woman presented to the primary care physician with cough, dyspnea and smoking history , COPD was the most likely diagnosis for the male case scenario than the female. But with the use of spirometry the likelihood of diagnosis narrowed between men and women [4]. The primary care physicians are less likely to use spirometry. Women may not be referred to a higher centre for further diagnosis and less likely to receive spirometry. *Ancochea et al.* reveals the significant underdiagnosis that exists in Spain in women with COPD, and leads us to reflect on how to improve its diagnosis in the female population. [5]In most of these studies where there was underdiagnosis like the PLATINO study, the women tend to be younger and non smokers .[6]

Physiology of women COPD

We see increased incidence of asthma in boys when less than 15 years, but steadily increasing in women thereafter till perimenopausal period and there is also premenstrual aggravation of symptoms in airway diseases. All these point to the fact that sex hormones may play a role in airway disorder .[7]

Histologically , female patients had bronchioles with thick airway walls with increased epithelial and adventitial component and also a smaller lumen. We also see increased prevalence of chronic asthmatic bronchitis in females due to that.

There is an increased responsiveness of the innate and adaptive inflammatory leukocytes of the female immune system leading to progressive infiltration of the lungs progressing to chronic inflammation. Emphysema may be an autoimmune disease and certain females with end stage emphysema have anti endothelial cell antibodies. In general autoimmune diseases are more prevalent in women which contributes to the above factor.[8]

Increased susceptibility to smoking and indoor air pollution

Women are at greater risk of lung function impairment than men for the same level of tobacco exposure. Female current smokers have a faster annual decline in lung function compared to their male counterparts.[9] The reasons may be

- 1 . First degree female relatives of probands who were current or ex smokers had greater reduced FEV1 than the male first degree relatives all indicating a genetic predisposition that is gender specific
- 2 . Women tend to underreport tobacco consumption [10]
- 3 . A dose dependant effect as the smaller airway of females have proportionately greater exposure.

- 4 . Hormonal effects on lung and airway size leading to increased sensitivity of airway receptors and increased heavy metal absorption due to menstruation induced anemia. [7]
- 5 . The women do the cooking and spend most of their day near the wood stove because most of the households do not even have separate kitchen. The smaller lung volume and smaller calibre of airways are more susceptible to the damaging effects of biomass exposure.
- 6 . Girls have an earlier and accelerated lung growth than boys , but as adults have a smaller lung volume than men .The child tends to spend time with the mother , eating and sleeping near the wood stove. Thus their lungs are exposed to the harmful effects of biomass particulate matter even during the early stages of its development and later in life leading to airway diseases .
- 7 . Hormone replacement therapy (HRT) users 25% less likely to have decline in lung function than their counterparts not using HRT. [11]

Clinical presentation and symptoms of COPD in females

Women were significantly younger, reported more cough and less phlegm probably be a cultural or societal artefact as they tend to swallow and less likely admit of having it. Dyspnea in women occurs earlier in life and at earlier stages of the disease. They also reported higher level of dyspnea measured by the modified medical research

council scale than men with same degree of airway impairment. Women also have worse scores in all domains of the St. George's Respiratory Questionnaire (SGRQ) thus have a lower quality of life. This may be due to.

1. The experience of dyspnea has cognitive, affective and physical dimensions, meaning impact of dyspnea not only related to the degree of lung impairment but to the patients emotional response to and higher interpretation of sensation. For example, limitation in exercise performance causes more distress than the sensation of dyspnea itself.
2. Women have higher intrinsic sensitivity to noxious stimuli including dyspnea. Neuroimaging studies reveal gender differences in the laterality of prefrontal cortical processing of noxious stimuli.
3. Women have greater awareness and attention to somatic sensations than men, making them more likely to sense dyspnea.
4. They tend to have lower inspiratory muscle strength
5. Women have higher rate of bronchial hyperreactivity, it is also an indicator of disease severity and progression.

Biomass exposure - a significant risk factor in women

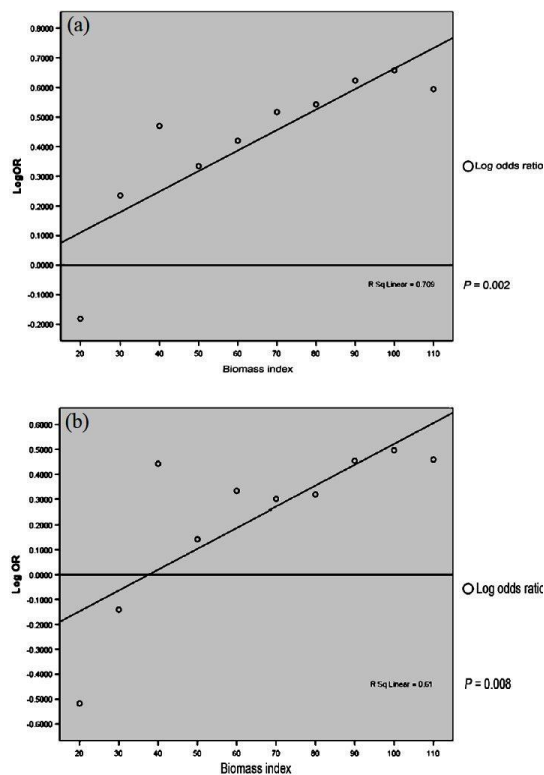
About two third of rural India use biomass fuel for cooking. Combustion of biomass fuels is the most important source of indoor air pollution in developing countries. A recent meta-analysis has shown that biomass fuel exposure is associated with a significant risk of chronic bronchitis and COPD and the strength of association similar to that of cigarette smoking [12] Those exposed to wood smoke having the greatest effect (relative risk 4.3) compared with animal dung / crop residue (relative risk 2.5) and coal / charcoal (relative risk 1.5-1.8).

Indoor air pollution due to biomass is ranked 10th among most preventable risk factor for global disease burden. Biomass smoke is a complex mixture of hundreds of volatile and particulate matter including organic and inorganic compounds and more than 90% are in the respirable range of less than 10 microns. The most important toxic constituents are solid particulate matter (PM₁₀ and PM_{2.5}), carbon monoxide, nitrogen and sulphur monoxides, aldehydes such as formaldehyde, polycyclic aromatic hydrocarbons such as benzopyrene, volatile organic compounds and free radicals.

Cooking results in significant emissions the mean 24h PM₁₀ may range from 300 to 3000mcg/m³ and may reach upto 30,000mcg/m³ . The mean 24 h carbonmonoxide ranged from 2 – 50ppm and may reach upto 500ppm . A study from Colombia found that biomass stove use for 10 or more years was associated with a greater risk of COPD as defined by a postbronchodilator

FEV1/FVC ratio less than 0.70 (GOLD stage 1 or greater; OR, 1.5; 95% CI, 1.22–1.86)[13] . Behera et al developed a simple , easily applicable ‘biomass exposure index ‘ that was calculated by multiplying the hours spent on cooking per day and number of years of cooking [14] to calculate the intensity of exposure. P.A.Mahesh et al demonstrated that a minimum threshold of biomass exposure index of 60 necessary to have a significant risk of developing chronic bronchitis.[14]

FIG; 1(a) *Linear regression plot of increasing biomass smoke exposure*



and log odds of chronic bronchitis among women in rural Mysore district, (b) Mysore district Taluk

The type of housing and ventilation play a role in level of exposure to particulate matter. In rural India most of the houses are mud houses with no separate kitchen and poor ventilation with no windows let the combustion particles settle in the household leading to high exposure. And also due to lack of space / rooms sleep and spend time in the same room increasing the hours of exposure. An interventional trial in Mexico showed significant attenuation in "FEV1 and fewer respiratory symptoms with the use of improved cooking stoves.[16]

Childhood exposure to biomass is an added risk factor for the development of COPD. The developing lung is highly susceptible to damage from exposure to PM particulate matter from biomass fuel combustion. School children in rural India, where use of biomass is common have a significant reduction in lung function and increased bronchial hyper reactivity. *Grigg et al* 2009, showed a direct link between childhood exposure to PM of biomass and development of COPD in non cigarette smoking women.[17]

Nutritional status of women

Nutritional status is mainly evaluated by BMI. *Charlotte Landbo et al* in his analysis over 17 years showed an independent effect of BMI on survival, with significantly higher mortality seen in underweight subjects than in those of normal weight. In subjects with mild or moderate COPD, the associations between BMI and mortality did not reach significance, but the relation tended to be U-shaped. The impact of BMI on COPD mortality was stronger than that

on all-cause mortality, with RRs between the lowest and highest BMI of 5.56 (range: 2.47 to 12.54) and 7.17 (range: 2.45 to 21.00) in men and women, respectively. [18]

The body mass is divided into two compartments, one called fat mass and the other fat free mass with the latter to contain the main metabolically active organs particularly skeletal muscle mass. This might be attributed to the fact “that loss of skeletal muscle mass is the main cause of weight loss in COPD, whereas loss of fat mass contributes to a lesser extent, leading to the plausible theory that FFMI reflects better the muscle mass than BMI. Low FFMI is significantly correlated with severity of COPD. Thus, an important issue is to explain why the skeletal muscle mass diminishes as the disease progress while it remains stable in early stages. This might be attributed to the high rest energy expenditure due to increased work of breathing in combination with inadequate dietary intake, to physical inactivity due to exercise intolerance, to excessive apoptosis of skeletal muscle due to increased systemic inflammation, and/or to the presence of hypoxia and the more frequent use of systemic corticosteroids.[19]

Smoking status of Indian women

Cigarette consumption in India is falling steadily even as the number of women smokers is rising, making it home to the second largest number of female smokers after the United States. According to the latest data on cigarette consumption given by the health ministry in Parliament, showed that the

number of women smokers in India went up from 5.3 million in 1980 to 12.7 million in 2012. According to a 2009-10 survey by the health ministry on tobacco smoking among Indian men and women were 15% and 2% respectively. About 9% of men and 1% of women both chew tobacco and smoke it. [20]

Long standing asthma and the risk of COPD

Chronic airway obstruction is persistence of airway obstruction(low FEV1) inspite of pharmacological attempts at reversal. Longer duration of asthma may lead to severe airway obstruction. Asthma is associated with additional decline in FEV1 than normal. Low baseline lung function, less response to beta agonists, severe bronchial hyperresponsiveness, male sex, mucus production and frequent exacerbations all lead to persistent airflow limitation.

In childhood asthma, wheezing between the ages of 3 and 6 (Tucson study), severe bronchial hyperresponsiveness, early onset of respiratory symotoms, severe respiratory symptoms, female sex, persistent wheezing all lead to low FEV1 in adulthood.[21]

CT scan revealed bronchial wall thickening in asthmatic patients with irreversible airway obstruction. When emphysema features are seen in asthmatics with irreversible airway obstruction, have longer duration of disease and increased asthma severity. It still remains unclear whether asthmatics with

airway obstruction meeting GOLD spirometric criteria are pathologically and phenotypically similar or different from COPD.

Outdoor air pollution as a risk factor

Outdoor air pollution is a mixture of hundreds of pollutants that originate from traffic, industries and other sources. There are evidences that living near busy roads leads to exposure to particulate matter, O₂ and NO₂ lead to deleterious effects on the airway due to airway oxidative stress, systemic.” and “pulmonary inflammation. Reduced ciliary activity, amplification of viral infections and increase in bronchial hyper reactivity. Biological plausibility is provided by the observation between black carbon content in respiratory macrophages and decreased pulmonary function growth. It occurs during our entire life span and may lead to irreversible airway obstruction and COPD. There is evidence that daily variation in outdoor air pollution can lead to exacerbation of COPD. The German SALIA study showed higher PM₁₀ in the environment lead to increase in COPD prevalence [22]. Many studies present strong evidence of an association between outdoor air pollution and decreased pulmonary function during childhood and adolescence.

Second hand smoke as a risk factor for COPD

Exposure to secondhand smoke (SHS), which contains potent respiratory irritants, may lead to chronic airway inflammation and obstruction s. Biological plausibility is supported by the presence of numerous airway

irritants contained in tobacco smoke and the strong relationship between direct smoking and COPD. *Rachel E Jordan et al* showed that never smokers having clinically significant COPD, where never smokers exposed to between 1 and 19h of passive smoking in a week had a 52% excess risk and those exposed to ≥ 20 h had an excess risk of 98%.[23] A study from China found that self-reported cumulative lifetime SHS exposure at home and work was related to a greater risk of COPD, as defined by spirometry (GOLD stage 1 or greater).[24]“Another study showed that living with a smoker was associated with a greater risk of a physician diagnosis of COPD.[25]

Occupational exposure as a risk factor for COPD

A diagnosis of “occupational COPD” is rarely made by clinicians; this situation is in sharp contrast to occupational asthma, which is more frequently recognized. The demonstration of an association between occupational exposures and COPD in epidemiological studies can be difficult because of several factors. First, COPD is multifactorial in etiology, with critical (and mostly unknown) host as well as nonoccupational environmental determinants of risk. Second, unlike workers with pneumoconioses, individuals with COPD due to occupational exposures cannot be distinguished from those with the disease due to other causes. Third, many workers with COPD have concurrent exposure to cigarette smoke (direct and/or secondhand smoke) and workplace irritants. Fourth, exposed workers at baseline tend to have better overall health and pulmonary function than the general population, the so-called healthy

worker effect. Fifth, workforce studies are often limited to a “survivor” population because of inability to assess or monitor workers who leave their jobs, thereby underestimating the chronic effects of occupational exposures.

It has been estimated that 15% of COPD is attributable to occupational exposure. Vapors, gas, dust, and fume exposures have been shown to be associated with COPD among workers in various occupations and industries. Exposure to organic, inorganic dust, and sensitizing agents in agricultural and food workers shows higher prevalences of respiratory conditions including COPD morbidity and mortality. Chronic exposure to coal and silica dust increases risk of COPD among miners. The dusts from coal, stone quarries, wood, cereals and agricultural work, animal stables, textiles, and paper production that can arise in occupational environments have been regulated by the International Labor Organization and considered possible as contributors to COPD.

Among construction and extraction workers, the odds of having chronic bronchitis were 1.4 times that of workers in management occupations, indicating that there are factors associated with construction and extraction work.[26]. *Doney et al* in the National Health Interview Survey Data 2004 to 2011, showed that females had a higher prevalence than males, which is consistent with findings reported by *Ford et al.*[27][28] Various factors have been associated with higher COPD among women, some of which are

environmental tobacco smoke, biological differences, occupational exposure, or a combination of all these factors.

Low socioeconomic status as a risk factor

Socioeconomic status is a total measure of an individual's or family's economic and social position in relation to others. Poor housing conditions and home dampness with increased house dust mites and biomass usage are all associated with respiratory symptoms, reduced lung function, and lower socioeconomic status. Household crowding has been hypothesised to cause increased instances of respiratory infections and thus increased rates of respiratory disorders, although this was not confirmed in the Tucson study. Bakke et al. have showed that low educational level is an independent determinant for COPD.[29] Individuals of the lowest socioeconomic strata were at least twice as likely to have poor outcomes as those of the highest (range from no difference to 10-fold difference). *Gershon et al* showed evidence that social and economic disadvantage appears to have a significant consistent impact on COPD mortality and morbidity.[30]

Asthma COPD Overlap Syndrome (ACOS)

Asthma COPD overlap syndrome is a heterogenous disease characterised by persistent airflow limitation showing several features which has association with both asthma and COPD. They are usually above 40 years of age with respiratory symptoms that may or may not present in childhood

with persistent airflow limitation showing partial reversibility. *Joan B Soriano et al.*[31] studied data from a very extensive population and reported that 19% patients with obstructive lung disease had a concomitant diagnosis of asthma, chronic bronchitis or emphysema. Similarly, *S E March et al.*[32] in a total of 469 patients reported that 55% of the population studied had asthma as the predominant COPD phenotype.

There are two well-known hypotheses, the “Dutch hypothesis” suggests that COPD and asthma are the same basic disease process and that long standing asthma predisposes to COPD and the “British hypothesis” that “proposes COPD and asthma are distinct entities and that both diseases coexist separately within the same individual.

Marco R et al.[86] in a survey of Italian patients revealed that in those diagnosed with asthma, 16-61% also had ACOS and those diagnosed with COPD, 25-33% also had ACOS. Soriano et al.[26] reported that an estimated 23% COPD subjects between ages 50 and 59 possibly has a mixed phenotype. With an increase in age to 70-79.4, the percentage increased to 52%. In the EPISCAN epidemiological study where bronchodilator test was used as a reference, 31.5% of the COPD patients had a positive test. Hence, from the above data, it can be concluded that between 20-50% of COPD patients may have a mixed phenotype

Radiological presentation of female COPD patients

Pat G.Camp et al found that participants exposed to biomass smoke had less emphysema, based on both qualitative and quantitative CT measures, than those exposed to tobacco smoke. He also noted that predominance of airway wall thickening with predominant airway phenotype in biomass exposed women. The radiologists' rating also showed worse air trapping in women exposed to biomass smoke.[34] Several mechanisms may be responsible for the phenotypic differences between exposure groups observed in this study.

First, differences in chemical composition of biomass and tobacco smoke could lead to different pathophysiological processes. Secondly, biomass exposure in rural villages can begin *in utero*, and individuals are often exposed throughout their lives, with women receiving the largest cumulative exposures may predispose to a different COPD phenotype as adults, compared to tobacco smokers who may begin smoking at an older age. Thirdly, there are possible differences in the inhalation pattern individuals inhaling biomass smoke would use a consistent tidal breathing pattern. Conversely, cigarette smokers usually smoke in a two-phase pattern: first the smoke is drawn into the mouth without direct inhalation into the lungs, then the smoke is inhaled into the lungs with an additional volume of air. This leads to deposition of particles in airways in biomass exposed individuals producing airway predominant phenotype as opposed to tobacco smoke exposed individuals where it gets deposited more peripherally and producing emphysema predominant phenotype. In addition,

the radiologists indicated the presence of bronchiectasis in the scans of 14% of the participants exposed to biomass smoke compared to 0% of those exposed to tobacco smoke ($p=0.009$). [34]

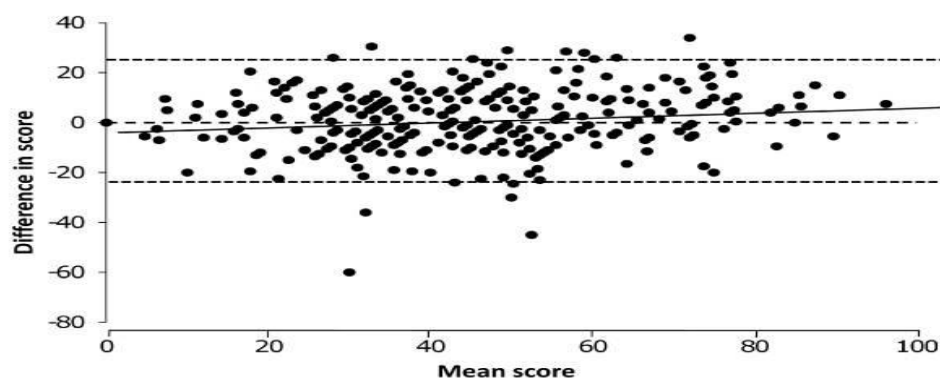
Severity of symptoms by CAT score.

CAT scores in women

The COPD assessment test is a short and simple (set of 8 questions) completed by the patient which aim to assess the impact of cough, sputum, dyspnea, chest tightness on the health status of the patient due to COPD. It has a high correlation with SGRQ ($r=0.84$) which is a validated score to assess the quality of life. Higher scores denote a more severe impact of COPD on a patient's life. The difference between stable and exacerbation patients was five units.

As women tend to have a lower quality of life, they have a higher CAT score. The CAT questionnaire in spite of having small number of components, cover broad range of health effects of COPD on health. ,

FIG 2 : *The Bland and Altman plot showed a very stable relationship across the scaling range between CAT score and SGRQ score[35]*



At the mild end of the CAT scale the score slightly over-estimated severity by a small amount (SGRQ = 0, adjCAT = 5, equivalent to 2 CAT units) and at the severe end it slightly under-estimated severity (SGRQ = 100, adjCAT = 92.5, equivalent to 37 CAT units). This level of agreement was sufficient to permit direct mapping between SGRQ and CAT scores. [35]

Impact of CAT score on degree of airflow limitation.

The relationship between CAT score and FEV1% predicted suggests that CATscore is linked to severity of airflow limitation and GOLD classification in stable COPD patients. There was a significant association between the FEV1% predicted and total CAT score ($r = -0.55$, $p < 0.001$). Health status as measured by CAT worsens with severity of airflow limitation. [36]

Paul W Jones et al(21) found patient centered assessment successfully graded COPD severity clinically and appeared to have greater discriminative power for assessing severity in COPD than FEV1 based staging. [37]. Sumer et al in his study had a significant correlation between CAT score and the stage of the disease ($p = 0.004$).[38]The number of exacerbations in the preceding year and FEV1 were independent predictors of the CAT score in the general linear model. During exacerbation, rises in CAT score were significantly associated with falls in FEV₁(6) [39]

Factors affecting severity of airflow limitation

The impact of age on FEV1 was proven when *We Johannes et al* studied the incidence and significance of airflow limitation in a population-based geriatric sample using both an age-dependent predicted lower limit of normal (LLN) value and a fixed-ratio of $< .70$ spirometric criterion. The incidence

increased dramatically with age when using a fixed ratio, but less so when using LLN. In addition, a sex effect was observed with the LLN criterion. He found that female sex may be a risk factor for developing airflow limitation and consequently COPD. [40]

Kurmi et al observed ventilatory function (forced expiratory volume in 1 s (FEV1), forced vital capacity (FVC) and forced expiratory flow at 25–75% of FVC) was significantly reduced in the population using biomass across all age groups compared to the non-biomass-using population, even in the youngest (16–25 yrs) age group (mean FEV1 (95% CI) 2.65 (2.57–2.73) versus 2.83 (2.74–2.91) L; $p=0.004$).[41]

Mitra et al showed that mean BMI of stage 1 COPD subjects was 26.21, stage 2 was 22.91, stage 3 was 20.78, and stage 4 was 15.71. One-way ANOVA showed that BMI of the patients were decreasing with increasing severity of the disease (GOLD) and it was statistically significant ($P < 0.05$). Thus found a positive correlation between severity of obstruction and BMI in COPD. [42]

Banurekha B et al noted that mMRC dyspnoea scale correlated well with FEV1 post spirometric indices. $D(P=0.0001)$ [43]

Pulmonary Hypertension in female COPD patients

Pulmonary hypertension (PH) is a common and well established complication of chronic obstructive pulmonary disease (COPD). Its presence is associated with decreased survival. The pathophysiology of the development of PH in COPD is poorly understood and is likely multifactorial. Morphologic changes in the pulmonary arteries are initiated by the toxic effects of tobacco

and biomass smoke and progress in parallel with the parenchymal changes of COPD. An endothelium-derived vasoconstrictor-dilator imbalance, mainly from a decreased endothelial nitric oxide expression, increased vascular endothelial growth factor and serotonin transporter expressions have also been implicated in the pathogenesis of pulmonary hypertension. *Sertogullarindan et al* in their study sex had highest value of OR for PH in moderate and very severe COPD cases and posed that biomass smoke has a greater risk for PH in women and also suggested a predisposition to PH in females. [105]

Common co-morbidities in female COPD

It has been observed in the ECLIPSE study that comorbidities were significantly higher in patients with COPD than in smokers and never smokers [44]*Dal Negro RW et al* assessed that the overall prevalence of co morbidities was 2.6 per patient with 2.5 in males and 3.0 in females.($p < 0.05$).[45]The important comorbidities associated with COPD are cardiovascular disorders (coronary artery disease and chronic heart failure, hypertension), metabolic diseases (diabetes mellitus, metabolic syndrome and obesity), bone disease (osteoporosis and osteopenia), stroke, lung cancer, cachexia, skeletal muscle weakness, anaemia, depression and cognitive decline. [46] Cardiovascular disorder was more common in males while metabolic, digestive and osteoarticular disorder more frequent in females.

Diabetes in female COPD patients.

Diabetes affects 2–37 % of patients with COPD. women with pre-existing asthma or COPD had a higher risk of developing type 2 diabetes,

independently of traditional diabetes risk factors including cigarette smoking chronic airway inflammation may increase the risk of type 2 diabetes through underlying proinflammatory mechanisms. Elevated circulating levels of certain inflammatory cytokines caused by chronic airway inflammation may also contribute to the development of insulin resistance in the liver, skeletal muscle, and vascular endothelium, ultimately leading to the clinical expression of type 2 diabetes.[47]

The average rate of decline in lung function in diabetes mellitus patients with history of no lung disease, as measured by FEV1 was 71 ml/year compared to an expected decline in healthy non-smokers of 25–30 ml/year, The lung function decline in patients with diabetes may be a consequence of diabetes itself and diabetic patients seem to have an increased risk of several non-neoplastic lung conditions such as COPD [48]

Hypertension in female COPD patients.

Augusti A et al and *Sin DD et al* noticed that Reduced FEV1 nearly doubles the risk for cardiovascular mortality independent of age, sex and cigarette smoking.[44, 49] *Anthonisen NR et al* reported that a 10 per cent decrease in FEV1 among COPD patients increases the cardiovascular event rate 28 per cent. [50]

Curkendall SM et al showed COPD patients were 1.76 times more likely to have arrhythmias, 1.61 times more likely to have angina, 1.61 times more likely to develop acute myocardial infarction and 3.84 times more likely to develop congestive heart failure. [51] *DeLucas Ramos et al* in their multivariate

analysis adjusted for the remaining factors, COPD was still an independent risk factor suggesting that COPD patients had a high prevalence of cardiovascular disease, higher than expected given their age and the co-existence of classic cardiovascular risk factors [52]

Depression in female COPD patients

Patients with COPD, particularly severe COPD, were at an increased risk of developing a diagnosis of incident depression. A wide range of depression prevalences in patients with COPD from 7% with an $FEV_1 < 80\%$, up to 79.1% in patients with COPD with chronic respiratory failure. This is in accordance with *Van Mannen et al* study.[53]*Schneider et al* in his study noticed that the relative risk estimate (odds ratio [OR]) of developing an incident depression diagnosis for patients with COPD was 1.44 (95% CI, 1.30–1.60), The cumulative incidence has recently been reported to be 6.1%.[54]

The assessment of the impact of depression in COPD is complicated by a two-sided association; depression is believed to Patients with COPD, particularly severe COPD, were at an increased risk of developing a diagnosis of incident depression.

Depression contribute indirectly to the development of COPD as depressed people are less likely to quit smoking, but depression can also develop as a direct or indirect consequence of a COPD diagnosis. Anxiety and depressive symptoms are common in patients affected by COPD, even when their disease is mild in terms of FEV_1 and respiratory symptoms.

Female patients appear to be more susceptible to psychological impairment, which correlates with some specific symptomatic aspects of the disease, such as dyspnea. These aspects have greater importance in view of the rising prevalence of COPD in females. Psychological aspects need to be carefully assessed in COPD patients, particularly in females. Dyspnea more strongly correlated with depression more in women than men.

AIMS AND OBJECTIVES

Primary Objectives

1. To study the prevalence of severity of airway obstruction, sociodemographic, clinico-radiological profile in female Chronic Obstructive Pulmonary Disease.
2. To assess the correlation of COPD assessment test (CAT) and degree of airflow obstruction in Chronic Obstructive Pulmonary Disease in females.

Secondary Objectives

1. To also assess the correlation between sociodemographic and clinicoradiological factors affecting the severity of airflow obstruction.

ALGORITHM SHOWING SAMPLING METHOD ADOPTED

113 female patient above 40 years with respiratory symptoms with chronic cough,sputum,breathlessness and with post-bronchodilator ratio FEV1/FVC <0.70(according to GOLD) presenting to thoracic medicine OPD in RGGGH,Chennai and ITM,chennai

EXCLUSION CRITERIA

- Presence of active tuberculosis / Treated pulmonary tuberculosis
- Relative Contraindications for Spirometry like recent history of myocardial infarction, history of recent upper abdominal/thoracic surgery/cataract surgery, history of hemoptysis
- Psychiatric diseases other than depression
- Patients with significant cognitive impairment
- Those with disabling loco motor diseases and bed ridden patients were not included in the study
- Patients not consenting for the study

n=22

91 female patients
were finally included in the study

MATERIALS AND METHODS

Study design:

- The study was a cross sectional observational study.
- No specific intervention was done.
- Patients were included in the study through random selection.
- No specific method of randomisation was used.
- No controls were used in the study

Study period: January 2016 to August 2016

Study centre: Thoracic medicine outpatient department at,

1. Rajiv Gandhi Government General Hospital.
2. Institute of Thoracic Medicine, Chetpet.

Subject selection

Inclusion criteria:

(As per GOLD Guidelines 2016, Female COPD suspects criteria)[56]

Female patients more than 40 years of age with clinical history and symptoms suggestive of COPD for more than 8 weeks

- Dyspnoea: Progressive (worsens over time), Characteristically worse with exercise
- Chronic cough: Intermittent and productive or unproductive

- Chronic sputum production

AND

- Spirometric diagnosis of COPD according to GOLD guidelines 2016 with post bronchodilator FEV1/FVC under 0.70 [56]

Exclusion criteria

- Presence of active tuberculosis / Treated pulmonary tuberculosis
- Relative Contraindications for Spirometry like recent history of myocardial infarction, history of recent upper abdominal/thoracic surgery/cataract surgery, history of hemoptysis
- Psychiatric diseases other than depression
- Patients with significant cognitive impairment were also not included as we needed our study patients to fill questionnaires.
- Besides since we needed our study patients to undergo six minute walk test, so those with disabling loco motor diseases and bed ridden patients were not included in the study.
- Patients not consenting for the study.

Sample size:

- 91 female patients who satisfied the inclusion and exclusion criteria were enrolled in the study

Data collection

The following were assessed in our study in patients with Chronic Obstructive Pulmonary Disease (COPD)

- History of presenting symptoms
- Severity of symptoms
- Socioeconomic status
- Risk factor assessment
- Nutritional status
- Presence and severity of depression
- Degree of airflow obstruction and reversibility
- Exercise capacity
- Radiological presentation

History of presenting symptoms

- History of cough, sputum production, breathlessness and wheeze their duration taken.
- Previous history of exacerbations and hospitalization in the past 1 year (exacerbations when patient needed to attend a health care unit because of symptoms which lead to increase in dose or addition of an antibiotic)

Severity of symptoms

Severity of symptoms as perceived by the patient was measured using Modified Medical Research Council grades (mMRC) and COPD Assessment Test (CAT).

COPD Assessment Test (CAT) :

The COPD Assessment Test (CAT) is a Patient-completed questionnaire assessing the impact of COPD on health status .Validation studies show that CAT has propertiesvery similar to much more complex health status questionnaires such as the St George’s Respiratory Questionnaire (SGRQ).[57] It consists of 8 questions each presented as a six-point (0-5) differential scale with a total score out of 40.It is available in 66 different languages. Tamil version of the document was used in our study.

The clinical impact of the disease is graded as follows: **Table 1**

CAT score	Impact level of symptoms
0-5	None
5-10	Low
10-20	Medium
20-30	High
30-40	Very high

Modified Medical Research Council grades (mMRC)

We used the modified medical research council scale of dyspnea to assess the severity of dyspnea. It quantifies the disability associated with breathlessness by identifying when breathlessness occurs (Grades 0 and 1) or by quantifying the associated exercise impairment (Grades 2–4). [58]The grades correlate well with other dyspnoea scales, lung function measurements[59] and with objective measures of disability such as six minute walking distance.[60]

The mMRC grades were self-administered asking patients to choose the

description that best suited their condition

Table: 2

The Modified Medical Research Council (MMRC) Dyspnoea Scale

Grade of dyspnoea	Description
0	Not troubled by breathlessness except on strenuous exercise
1	Shortness of breath when hurrying on the level <i>or</i> walking up a slight hill
2	Walks slower than people of the same age on the level because of breathlessness <i>or</i> has to stop for breath when walking at own pace on the level
3	Stops for breath after walking about 100 m <i>or</i> after a few minutes on the level
4	Too breathless to leave the house <i>or</i> breathless when dressing or undressing

Socioeconomic status

The most widely accepted scale for urban populations has been proposed by Kuppuswamy in 1976 . We used Modified Kuppuswamy's Socioeconomic Scale.[61]

Table 3 Kuppuswamy's Socioeconomic Scale

EDUCATION OF HEAD	SCORE
1. Profession or Honours	7
2. Graduate or post graduate	6
3. Intermediate or post high school diploma	5
4. High school certificate	4
5. Middle school certificate	3
6. Primary school certificate	2
7. Illiterate	1
OCCUPATION OF HEAD	SCORE
1. Profession	10
2. Semi-Profession	6
3. Clerical, Shop-owner, Farmer	5
4. Skilled worker	4
5. Semi-skilled worker	3
6. Unskilled worker	2
7. Unemployed	1

FAMILY INCOME PER MONTH [In Rs/month]	SCORE
1. \geq 41,796	12
2. 20,898 - 41,795	10
3. 15,674 - 20,897	6
4. 10,449 - 15,673	4
5. 6,269 - 10,448	3
6. 2,111 - 6,268	2
7. \leq 2,110	1
SCORING Socioeconomic Class	Total score
Upper	26-29
Upper middle	16- 25
Lower middle	11-15
Upper lower	5-10
Lower	< 5

Risk factor assessment

- Severity of smoking

If history of smoking was present, the severity was graded with smoking index for number of beedis/cigarettes smoked.

Smoking index is calculated as the product of number of cigarettes or bidis smoked per day and the duration of smoking habit in years.

Table :4 Severity of smoking based on Smoking Index [63]

SMOKING INDEX	SEVERITY OF SMOKING
< 100	Light smokers
100 – 300	Moderate smokers
> 300	Heavy smokers

- Biomass exposure

Information was collected regarding type of Biomass fuel exposure and duration of exposure. Number of years of exposure / Number of hours spent on cooking everyday personally. Then Biomass exposure index was calculated as the average number of hours spent on cooking daily for cooking multiplied by the total number of years spent in cooking personally.[14]As minimum threshold of biomass exposure index of 60 was necessary to have a significant risk of developing COPD was identified by Mahesh P et al we divided the study group as follows [15]

Table: 5 Severity of biomass exposure

Severity of biomass exposure	Biomass exposure index
No exposure	0
Exposure less than threshold	< 60
Exposure above the threshold	> 60

- History of Biomass Exposure during childhood less than 15 yrs was also obtained.[64]
- History of lower respiratory infections in childhood [65]
- History of physician diagnosed asthma and duration- the patients were divided into 3 categories as no asthma, history of asthma for more than 10 years and history of asthma for less than 10 years [66, 67]
- Family history of COPD- This history was not reliable as most of them were not sure about the diagnosis in the relatives and could not provide proof regarding the diagnosis[68]

- Occupational history- Information was collected on Occupational exposure to dust/fumes and duration of exposure.[69,70,71,72]
- Type of housing and living conditions - Type of housing – Thatched/ Tiled/ Concrete/ others, and whether it has separate kitchen or not/ whether it was the only room in the house/ regarding ventilation with windows and vent for the escape of biomass combustion products.[41]
- Whether the locality of the house is near to busy road with heavy traffic of motor vehicles including buses and lorries.[22]

Presence and severity of depression:

The presence and severity of depression in our study subjects was assessed using Patient Health Questionnaire – 9. Patient Health Questionnaire is a tool used worldwide for screening, diagnosing, monitoring and measuring the severity of depression. It is a simple tool that can be filled by the patients within minutes. The questionnaire has been shown to have good reproducibility. The questionnaire has 9 questions. Each question is scored from 0 to 3. Thus the total score of the tool ranges from 0 to 27. The 9 questions actually represent the 9 criteria used to diagnose depression as per DSM-IV.[73] PHQ-9 score of greater than 10 has the sensitivity and specificity of 88 % in diagnosing major depression. The Patient Health Questionnaire has been internationally validated for the purpose of identifying and grading the severity of depression. [74] PHQ-9 score of 4 is considered to be the upper limit of normal. Severity of depression is graded as given in Table 6 [75]

PHQ-9 score	Level of depression
0-4	None
5-9	Mild
10-14	Moderate
15-19	Moderately severe
20-27	Severe

Table 6: Interpretation of PHQ-9 score in grading severity of depression

Nutritional Status of the patient:

The nutritional status of the patient was measured using Body Mass Index and Fat Free Mass.

Body Mass Index:

Body Mass Index (BMI) was calculated from the height and weight of the patient using the formula $BMI = \text{Weight in kg} / (\text{Height in m})^2$.

The patients were classified based on the BMI as given in Table 8 as per WHO recommendations

BMI	Nutritional status
< 18.5	Underweight
18.5 – 24.9	Normal
25 – 29.9	Overweight
> 25	Obese

Table 7: Interpretation of nutritional status of patients using BMI[76]

Fat Free Mass (FFM):

Fat Free Mass has been shown to be a better indicator of nutritional status of an individual than Body Mass Index particularly in patients with COPD. [76]

Though there are different methods to measure Fat Free Mass like

We used the method of skin fold thickness in our study. The skin fold thickness was measured using adult skin callipers 4 different sites

- Front of arm
- Back of arm
- Below the scapula
- At the level of hips

Body density was calculated from skin fold thickness using

*Durnin and Womersley equations as shown in the **table: 7***

Age (years)	Equations for males	Equations for females
40 -49	$D = 1.1620 - (0.0700 \times L)$	$D = 1.1333 - (0.0612 \times L)$
> 50	$D = 1.1715 - (0.0779 \times L)$	$D = 1.1339 - (0.0645 \times L)$

Table 8: *Body density equations of Durnin and Womersley (D = Body density, L = Skin Fold Thickness)*

Body Fat percentage was then calculated using Siri Equation and Fat Free Mass was derived from it [78]

$$\% \text{ Body Fat} = (495 / \text{Body Density}) - 450$$

$$\text{Fat Free Mass} = (100 - \text{Body Fat } \%) \times \text{Body weight} / 100$$

Pulmonary Function Test:

Spirometry was done for all patients who satisfied the inclusion criteria. It was done as per the American Thoracic Society recommendations using Easyone Spirometer. [79]The instrument was calibrated daily. The procedure was explained to all patients before the test. Any recent history of illness, medication were enquired and the height and weight were recorded. The appropriate technique of spirometry was demonstrated to each patient individually before the start of procedure. The patients were asked to inhale rapidly and completely from Functional Residual Capacity (FRC). The patients were instructed to hold the mouth piece in their mouth, sealed tightly by their lips. Patients were asked to blast out air without any hesitation and were asked to completely exhale. Throughout the procedure, patients were coached using body languages and phrases. The testing was done in sitting position and nose clips were used.[79]

Within-manoeuvre criteria

Individual spiromgrams are “acceptable” if

They are free from artefacts [3]

Cough during the first second of exhalation

Glottis closure that influences the measurement

Early termination or cut-off

Effort that is not maximal throughout

Leak

Obstructed mouthpiece

They have good starts

Extrapolated volume <5% of FVC or 0.15 L, whichever is greater

They show satisfactory exhalation

Duration of ≥ 6 s (3 s for children) or a plateau in the volume–time curve or

If the subject cannot or should not continue to exhale

Between-manoeuvre criteria

After three acceptable spiromgrams have been obtained, apply the following tests

The two largest values of FVC must be within 0.150 L of each other

The two largest values of FEV₁ must be within 0.150 L of each other

If both of these criteria are met, the test session may be concluded

If both of these criteria are not met, continue testing until

Both of the criteria are met with analysis of additional acceptable spiromgrams

or

A total of eight tests have been performed (optional) or

The patient/subject cannot or should not continue

Save, as a minimum, the three satisfactory manoeuvres

Fig 3: *Acceptability and repeatability criteria as per ATS guidelines*

Measurements were made before and after at least 15 minutes of two puffs of salbutamol (200µg) administered using metered dose inhaler with a spacer. The degree of airflow obstruction was assessed as per GOLD guidelines³¹

GOLD classification	Severity	FEV₁/FVC	FEV₁
1	Mild	<0.70	FEV ₁ ≥80% predicted
2	Moderate	<0.70	50% ≤FEV ₁ <80% predicted
3	Severe	<0.70	30% ≤FEV ₁ <50% predicted
4	Very severe	<0.70	FEV ₁ <30% predicted

Table:9 Severity of airflow limitation as per GOLD guidelines

Exercise capacity:

Exercise capacity was measured using six minute walk test (6MWT). The six minute walk test was done as per the American Thoracic Society recommendations.[80] The test was performed indoors in a 100 ft hallway (30 m length). The length of the hallway was marked every 3m as well as the starting and ending point of each 60m lap. The turnaround points were marked with two small cones. The patients were asked to wear comfortable dresses and foot wear. They were asked to continue their usual medications. They were asked to avoid heavy meals before the test or indulge in any strenuous physical activity before 2 hours of beginning the test. The patients were instructed to rest for 10 minutes in a comfortable chair before beginning the test. During this period their vitals were measured. The patients were then instructed to walk as far as possible in six minutes. They were allowed to slow down or rest and then continue the test if needed. A visual

demonstration of the test was given before starting the tests. Post testing, the patients were made to relax and vitals monitored again. The distance covered by them in 6 minutes was measured in metres. The expected six minute walk distance was calculated for each patient using the following regression equations⁴² based on their sex, based on their sex, age (in yrs), height (in metres), weight (in Kgs).

The reference equation in females [81]

$$256.8 + [2.4 \times \text{Height}] - [1.5 \times \text{Age}] - [0.3 \times \text{weight}]$$

The six minute walk distance of each patient was expressed as percentage of the expected, calculated as

$$\text{Observed value} / \text{expected value} \times 100$$

The severity of impairment of exercise capacity was arbitrarily graded as in

Table 10: Interpretation of impairment of exercise capacity using 6MWT distance

6MWD%	Impairment of exercise capacity
100-75	No impairment
75-50	Mild impairment
50-25	Moderate impairment
25-0	Severe impairment

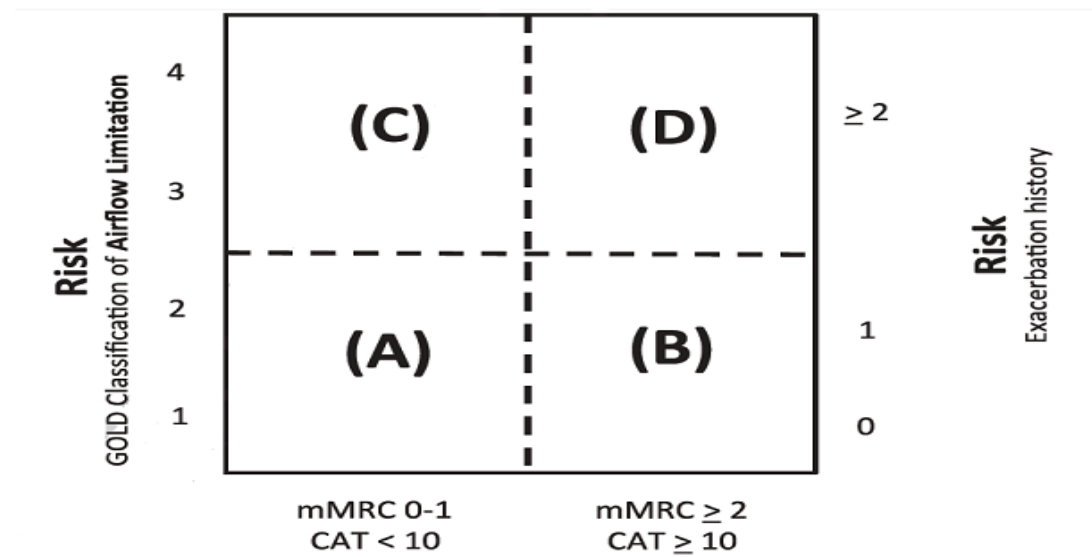
GOLD combined assessment of patients with COPD:

A new method for classification of COPD patients has been proposed by GOLD. [56] It takes into account 2 factors

- Level of symptoms by either of
 - o MMRC grade

- o COPD Assessment Test score
- Risk of exacerbations by either of
 - o GOLD classification of airflow limitation
 - o No of exacerbations in the preceding one year

The GOLD combined assessment of patients reflects the complexity of COPD better than the unidimensional analysis of airflow limitation previously used for staging the disease is given in **Fig 4**



GOLD Combined Assessment classification of patients with COPD [56]

- Group A - Less symptomatic, Low risk
- Group B - More symptomatic, Low risk
- Group C - Less symptomatic, High risk
- Group D - More symptomatic, High risk

HRCT CHEST:

An HRCT chest was taken for all patients included in the study. Following an initial conventional helical scanning for screening, an HRCT was done in full inspiration at 1mm slices. Four slices of 1 mm thickness were obtained at the following levels:

1. Superior margin of aortic arch (level of upper lung)
2. Level of carina (level of middle lung)
3. Level of inferior pulmonary veins (level of lower lung)

The window levels were set from -700 to -900HU which was appropriate for the lungs.[83]**Table 11**

SCORE	LAA PERCENTAGE
0	LAA <5%
1	$5\% \leq \text{LAA} < 25\%$
2	$25\% \leq \text{LAA} < 50\%$
3	$50\% \leq \text{LAA} < 75\%$
4	$75\% \geq \text{LAA}$

The low attenuation area (LAA) was measured by the visual assessment in bilateral lung fields according to the method of Goddard.[84] The total scores and grade of emphysema was calculated as follows: **table 12.**

TOTAL SCORE	GRADING
0	0
1-6	1
7-12	2
13-18	3
19-24	4

Bronchial wall thickness was assessed visually as follows: **Table 13**

GRADE	BRONCHIAL WALL THICKNESS
1	NONE
2	<50% adjacent pulmonary artery diameter
3	>50% adjacent pulmonary artery diameter

Based on the visual HRCT assessment, patients were classified into three phenotypes as follows:

1. Absence of emphysema, which showed little emphysema and LAA \leq grade 1 with and without BWT (**A phenotype**)
2. Apparent emphysema \geq grade 2 without BWT (**E phenotype**)
3. A combination of apparent emphysema = grade 2 and BWT of more than grade 1 (**M phenotype**)

Routine investigations including:

1. Chest X ray PA view
2. Hemogram
3. Plasma absolute eosinophil count
4. Random Blood Sugar
5. HIV antibody testing were done for all patients.
6. Sputum examination for acid fast bacilli
7. Electrocardiogram
8. Echocardiogram

STATISTICAL ANALYSIS:

The data collected was entered in to Microsoft (MS) excel worksheet and analyzed using Statistical Package for Social Sciences (SPSS) software version 16.0. Results were subjected for appropriate statistical analysis. All quantitative variables were expressed as mean & standard deviation for normally distributed data and median & inter quartile range for skewed data. All qualitative variables expressed as percentages & proportions. The statistical significance of association was tested using independent sample t – test or ANOVA for quantitative variables and Pearson Chi - square test for qualitative variables. The strength of association was expressed using Odds ratio, wherever applicable. Fisher's exact test was used when the expected value of more than 25% of the cells were less than five. Mann Whitney U test or Kruskal-Wallis Test was applied when the variable is ordinal or when the quantitative data was not normally distributed. All hypotheses were tested at a significance level of 95% and power of 80%.

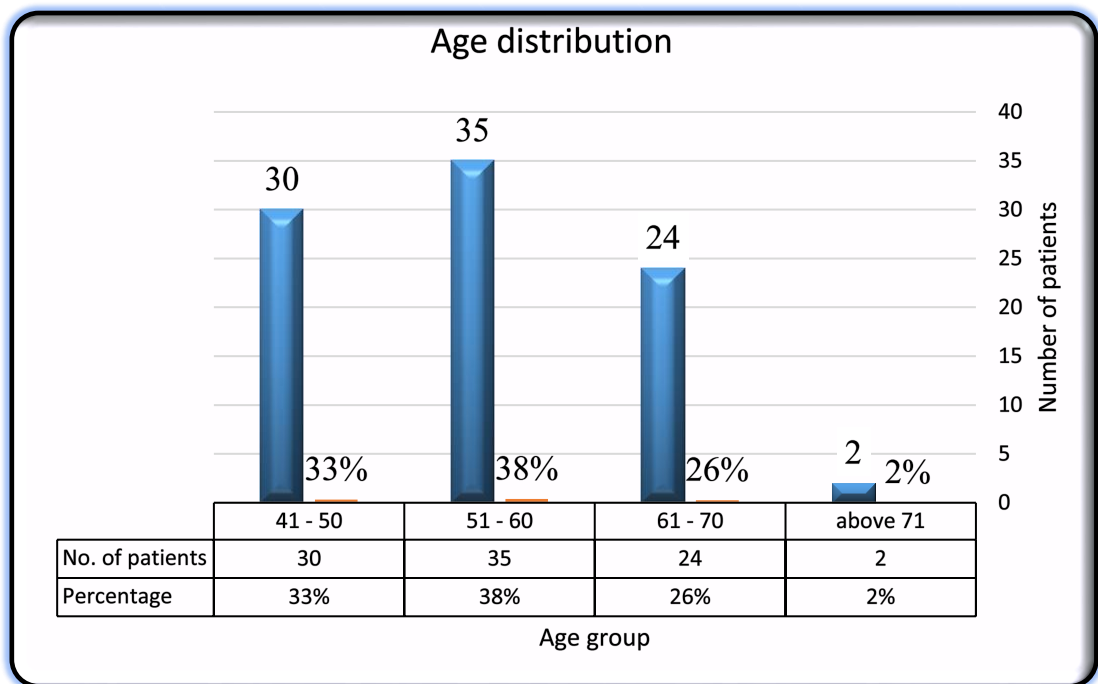
PATIENT CHARACTERISTICS

In the given study period, a total of 91 study subjects who satisfied the inclusion criteria were interviewed and their results are presented as follows.

Age distribution

The age group of patients in this study ranged from 41 to 73. The mean age of the study population was 56.63 with a standard deviation of 8.074. The number of patients in the age groups 41-50, 51-60, 61-70, and above 71 were 30 (33.3%), 35 (38.8%), 24 (26.6%) and 2 (2.2%) respectively.

Fig 5: Age distribution of patients



Symptom analysis

Combination of cough, sputum and breathlessness reported by 32 patients, presence of cough and sputum in 30 patients while combination of cough, breathlessness and wheeze least reported only in 2 patients. Cough is the most common symptom in 70 patients, while 67 reported sputum production too.

Fig 6: Count of patients by symptoms

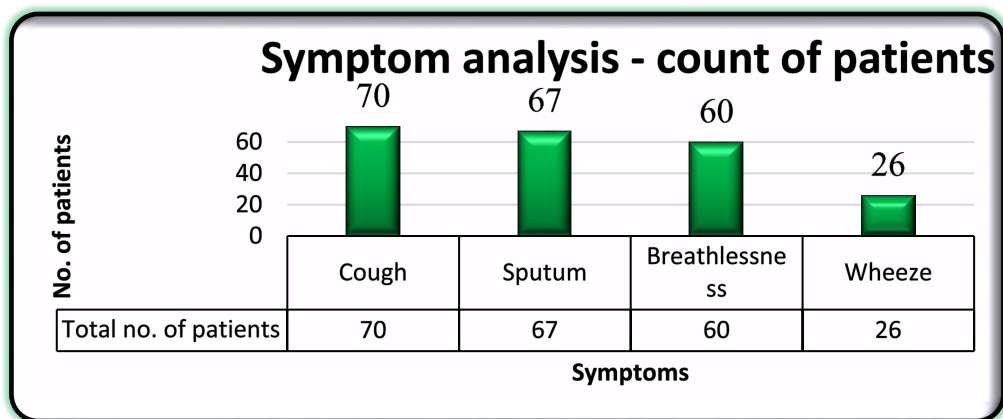
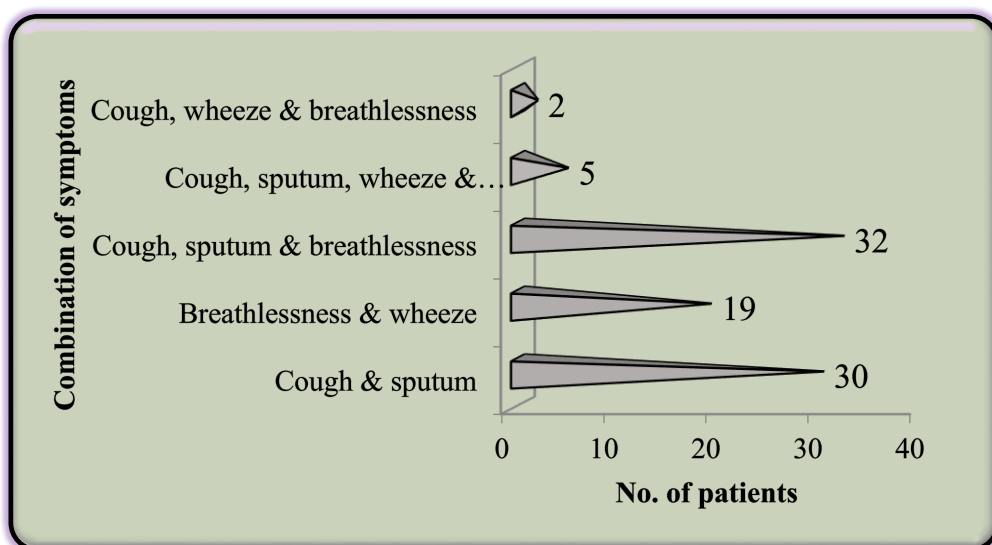


Fig 7: Distribution of patients by combination of symptoms (n=91)

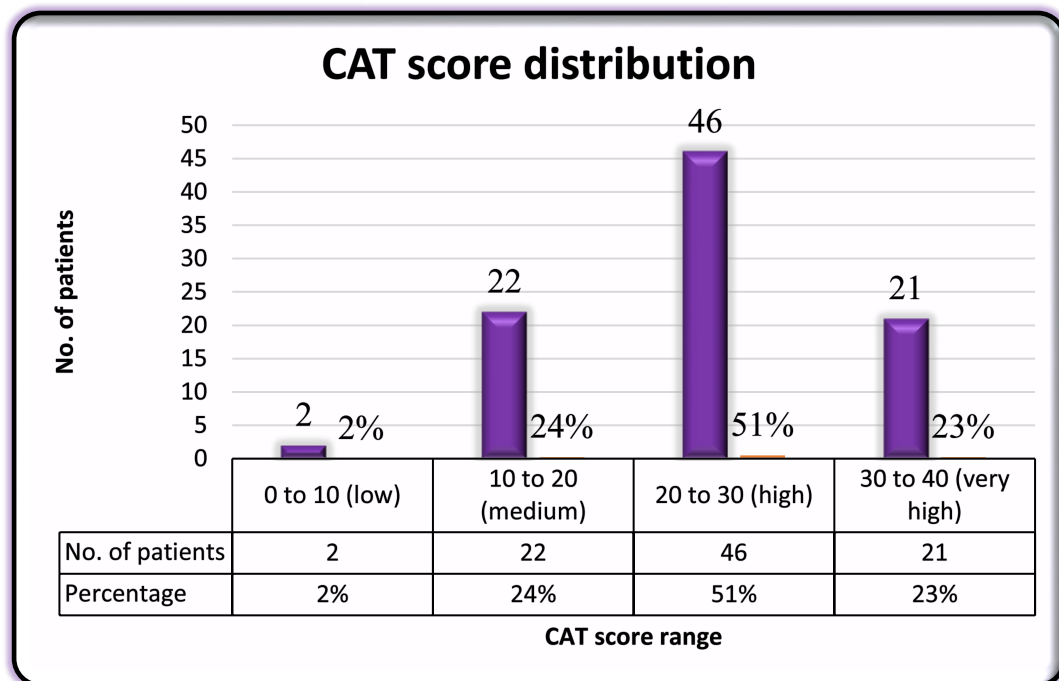


Severity of symptoms of the study population

CAT Score of the study population

When the severity of symptoms assessed with COPD Assessment Test (CAT), it ranged from 7 to 33 with a mean of 24.43 ± 6.55 . Almost 97.8% of patients were in the more symptomatic group according to GOLD guidelines.

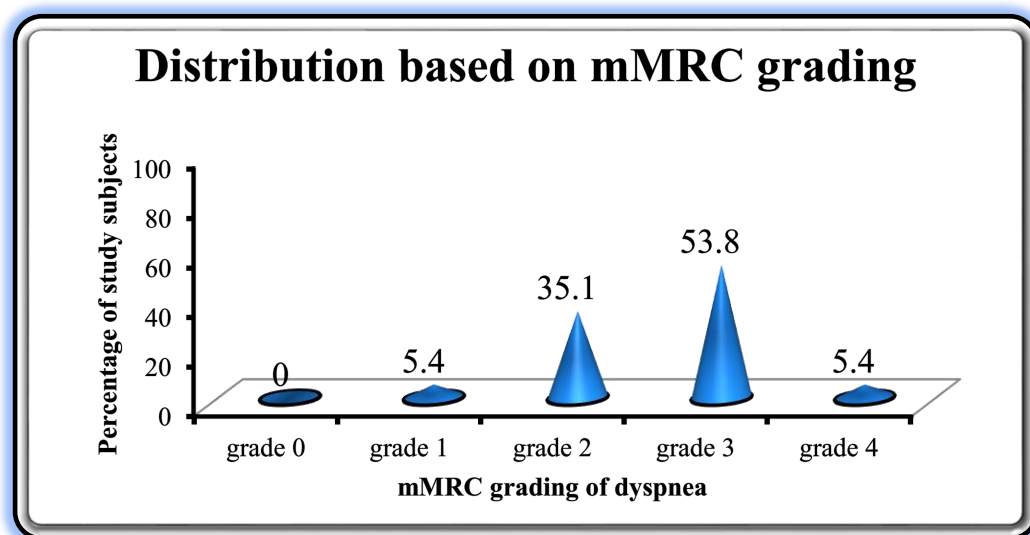
Fig 8: Distribution of patients by CAT score



Severity of symptoms based on mMRC grading of dyspnea

The severity of symptoms such as breathlessness was assessed based on mMRC grading of dyspnea. Of the 91 patients, no one reported grade 0, while the majority had grade 2 or 3 of mMRC of dyspnea. The number of patients with grade 0, 1, 2, 3 and 4 are 0, 5.4%, 35.1%, 53.8% and 5.4% respectively.

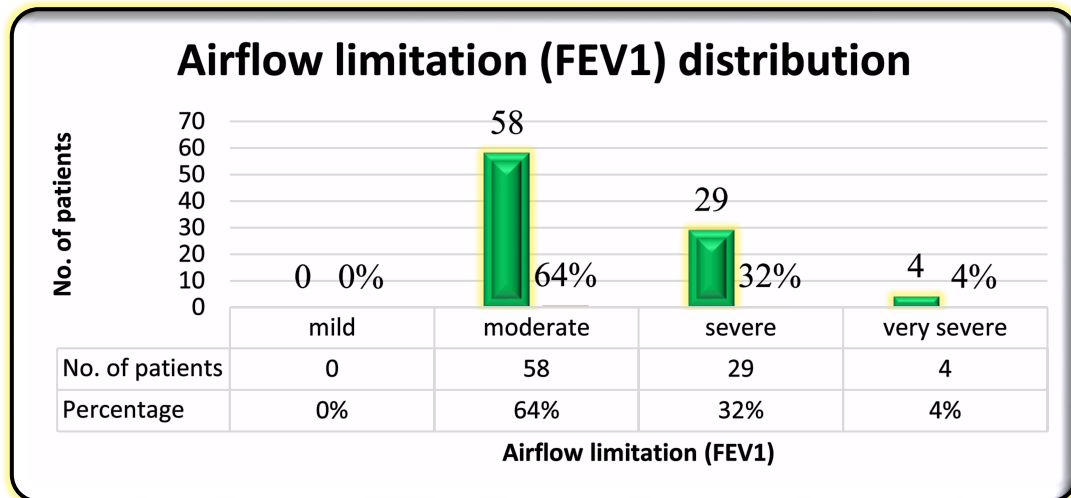
Fig 9: Distribution of patients based on mMRC grading of dyspnea



Degree of airflow limitation in study population

Airflow limitation assessed based on post bronchodilator FEV1% predicted as in GOLD guidelines. In our study population no patient was in the mild category (> 80% predicted). 4 of them were in the very severe category (< 30% predicted), 29 were in the severe airflow limitation category (30 to 50% predicted) and majority i.e., 58 were found to have moderate degree of airflow limitation(50 to 80% predicted). The mean FEV1 of the study population was 53.29% of predicted.

Fig 10: Distribution of patients by airflow limitations (FEV1)



Exacerbations and hospitalisation in the study population

In our study population 53 of them reported exacerbations in the last one year and 15 of them also had h/o hospitalisation in the last one year. Of the 53 patients, 30 (32.9%) of them had more than one episode of exacerbation. We can see that 35 (38.1%) of the patients are in the high risk category based on hospitalisation and frequency of exacerbations.

Fig 11: Distribution of patients by exacerbations

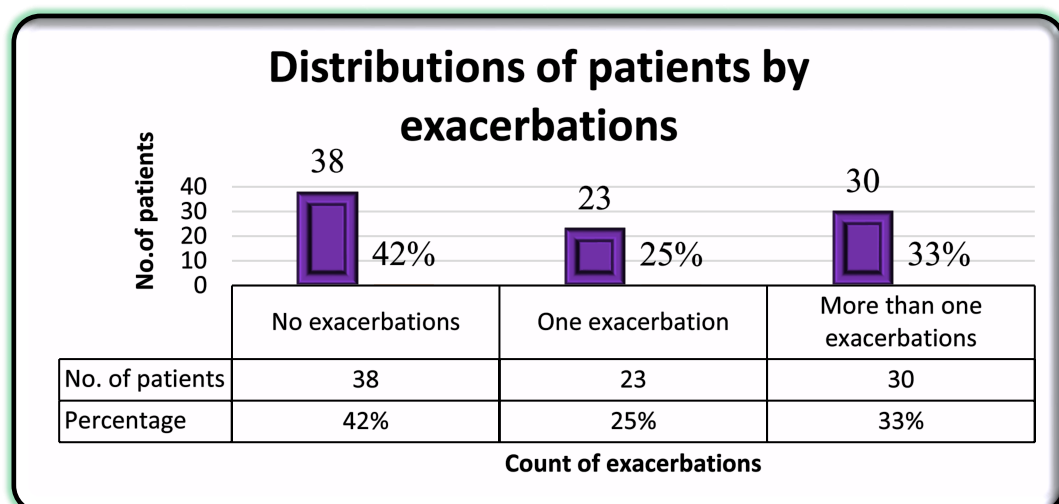
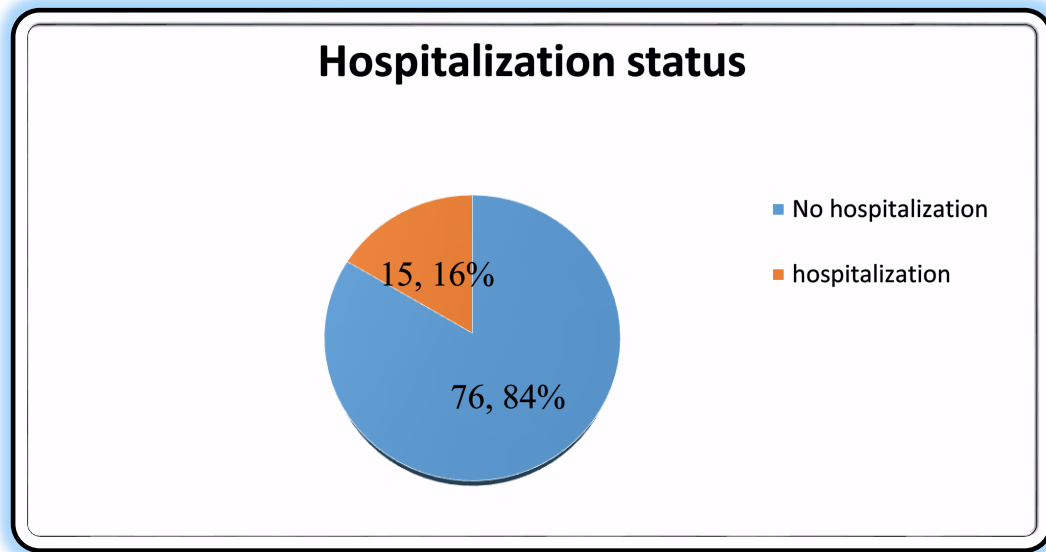


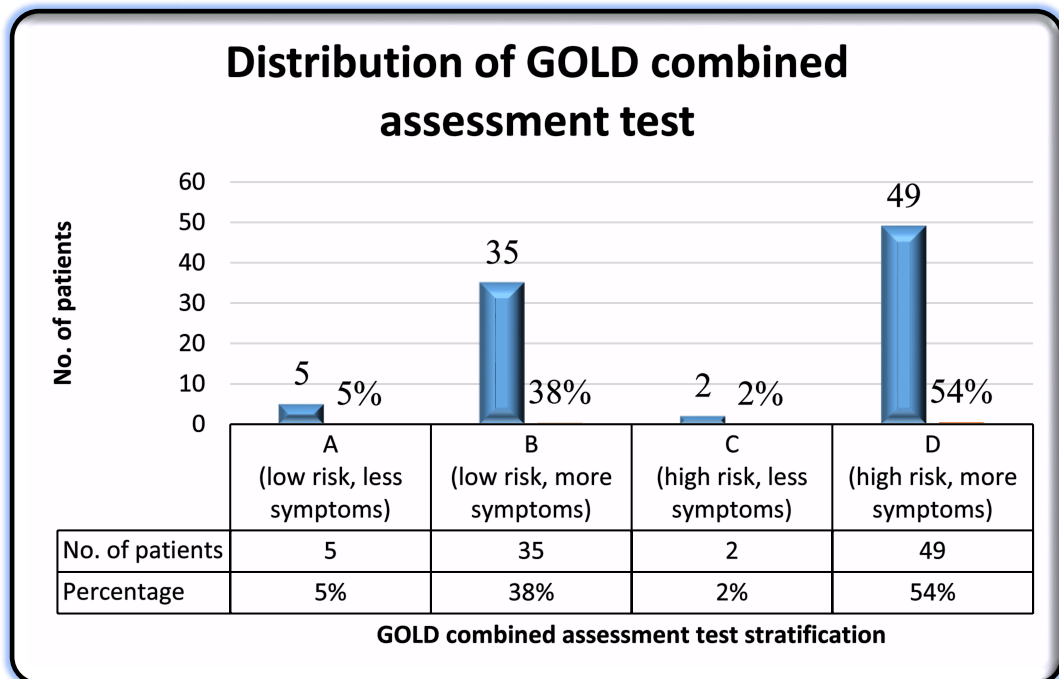
Fig 12: Hospitalization status



GOLD Combined Assessment Test stratification of study population

Our study group was classified based on GOLD combined assessment test as it is able to stratify whether our population is at high risk or low risk and also whether they were less symptomatic or more symptomatic. As the female population had high CAT score / higher mMRC grading of dyspnea , most of them almost 92.3% belonged to either B or D . Since most patients had more than one exacerbations, 49 (53.8%) belonged to the more symptoms and high risk group D. 35(38.4%) of them belonged to the more symptoms and less risk group B. We can see that 51 (56.04%) belong to the high risk group C and D. Only 5 (5.4%) belonged to the less symptoms and low risk group A.

Fig 13: Distribution of patients by GOLD combined assessment test

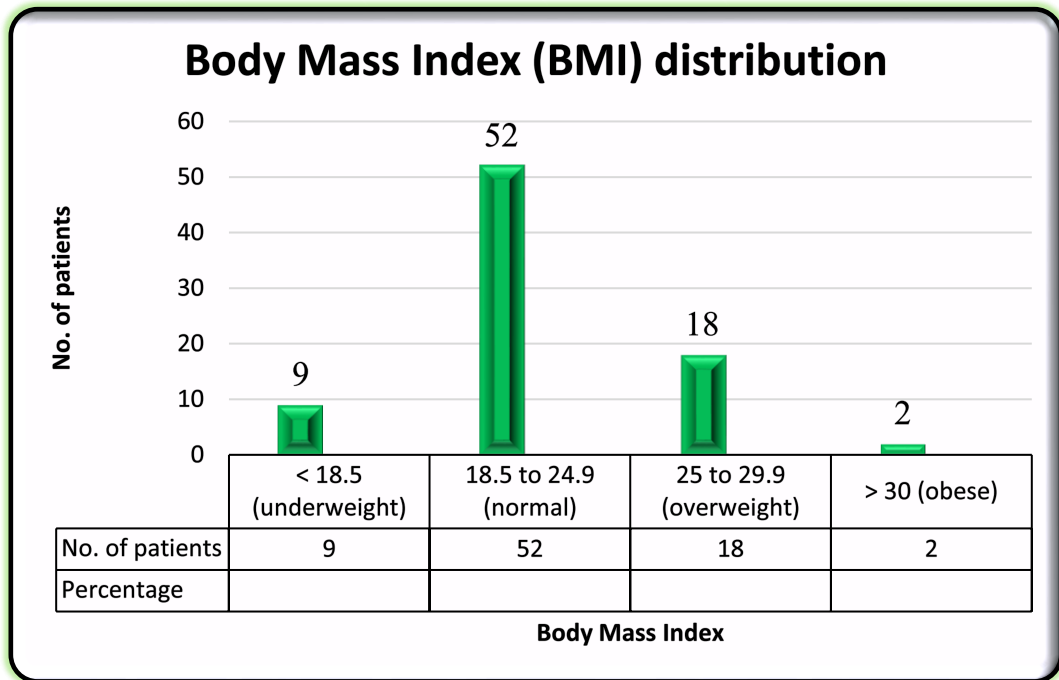


Nutritional status of the study population

Body mass index of the population

The body mass index of the study population was calculated by measuring the height, weight and using the Quetlet index. In this index the mean value was 22.37 kg/m² and the standard deviation observed was 3.307. In the study population 62 of them were in the normal healthy BMI range and only 2 of them were in the obese range. 9 were underweight range in our study. The mean fat free mass of the study population was 31.87 kg with a standard deviation of 4.87kg.

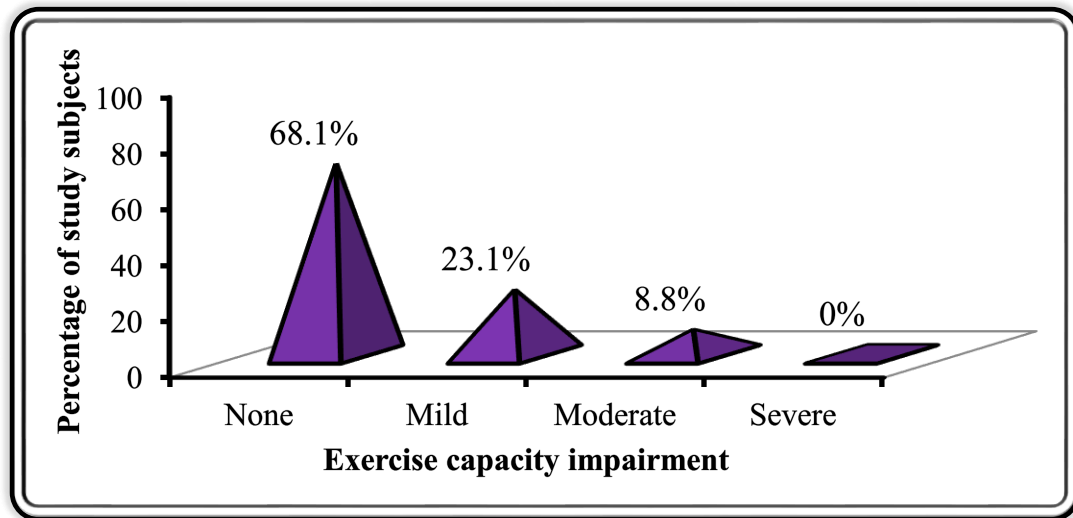
Fig 14: Distribution of patients by Body Mass Index using Quetlet index



Exercise capacity of the study population

The exercise capacity of the study population was studied based on 6 minute walk distance as recommended by ATS. As we divided the impairment based on 6MWD% arbitrarily into nil, mild, moderate and severe, no patient was in the severe group. Patients with nil impairment were 62, while 21 of them had mild impairment and 8 had moderate impairment of exercise capacity.

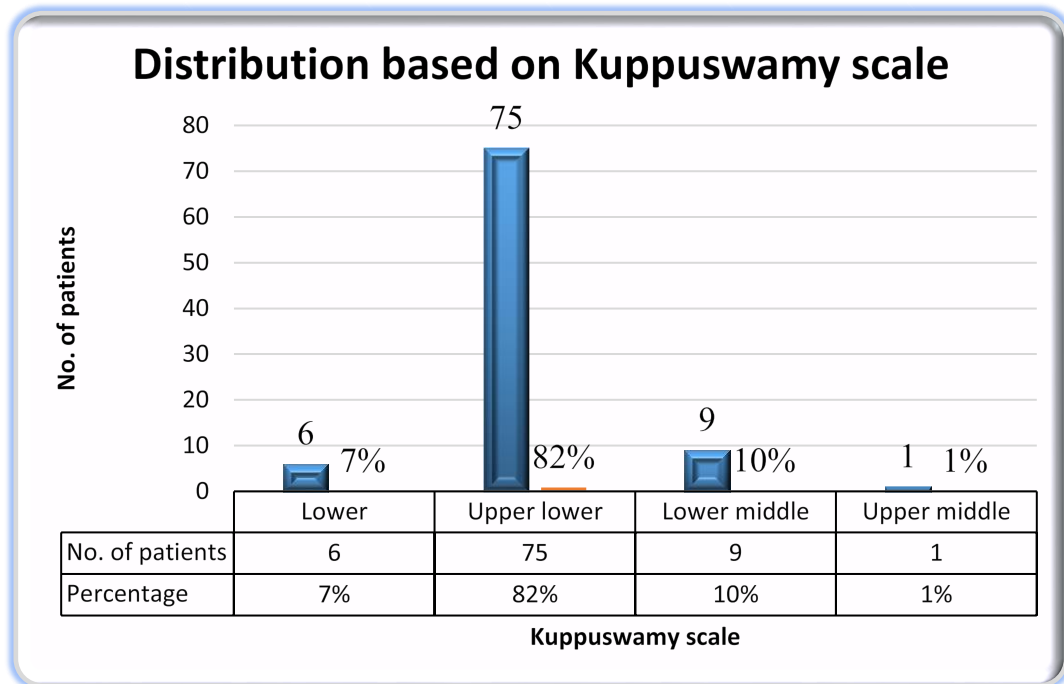
Fig :15 Distribution of patients based on exercise impairment



Socioeconomic status of the study population

The socioeconomic status of the study population was assessed based on Kuppuswamy scale of socioeconomic status. Most of the study population was in the upper lower class, only 1 patient was in the upper middle class, 6 in the lower class and 9 in the lower middle class. The percentage of patients belonging to lower, upper lower, lower middle and upper middle are 6.5%, 82.4%, 9.8% and 1.0% respectively. The mean income of the group was Rs. 5365.9 with a standard deviation of 2727.65. The lowest income was Rs. 2000 and the maximum income was Rs.15000.

Fig 16: Distribution of patients by Kuppuswamy scale



Educational status of the study population

In our study population 57 did not know to read or write. Of the remaining 34 patients only 17 had completed primary education, 6 had completed SSLC and only 2 had done their PUC.

Fig 17: Distribution of patients by educational status

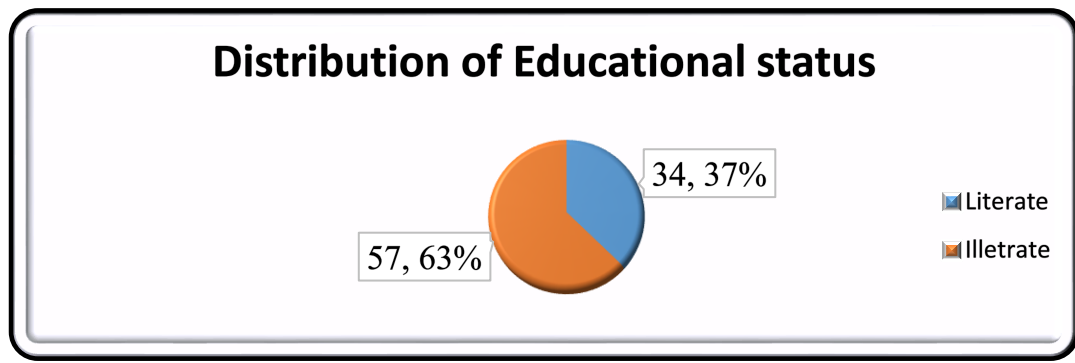
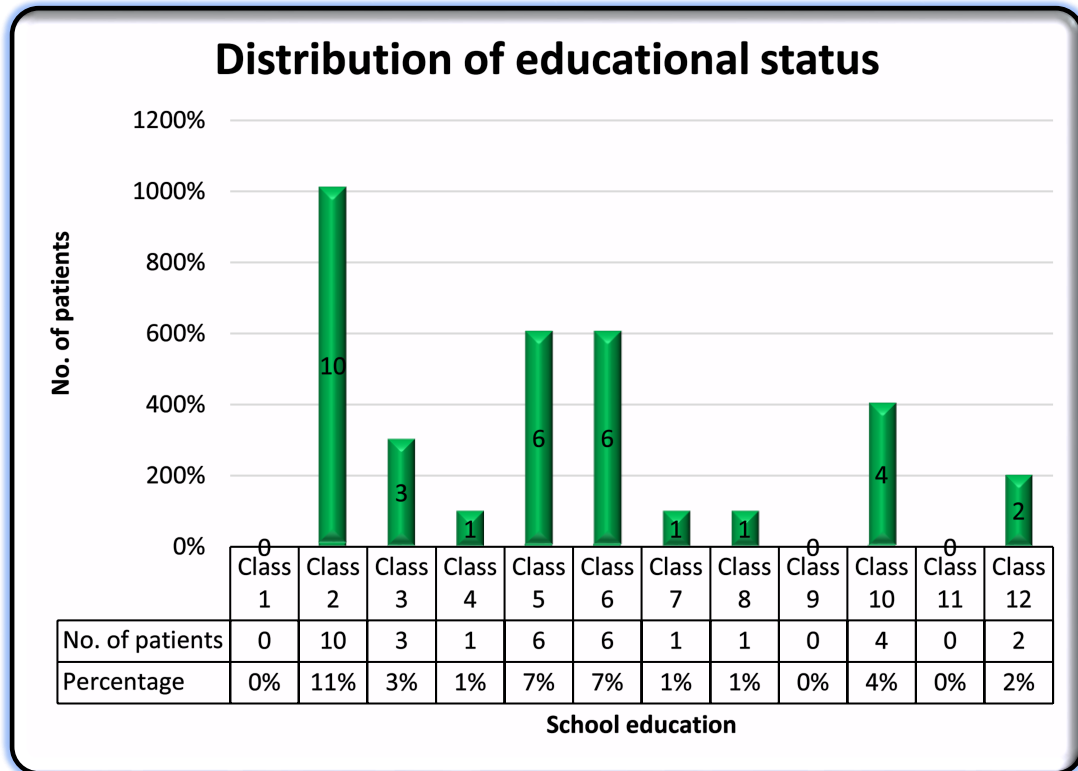


Fig 18: Distribution of patients by school education

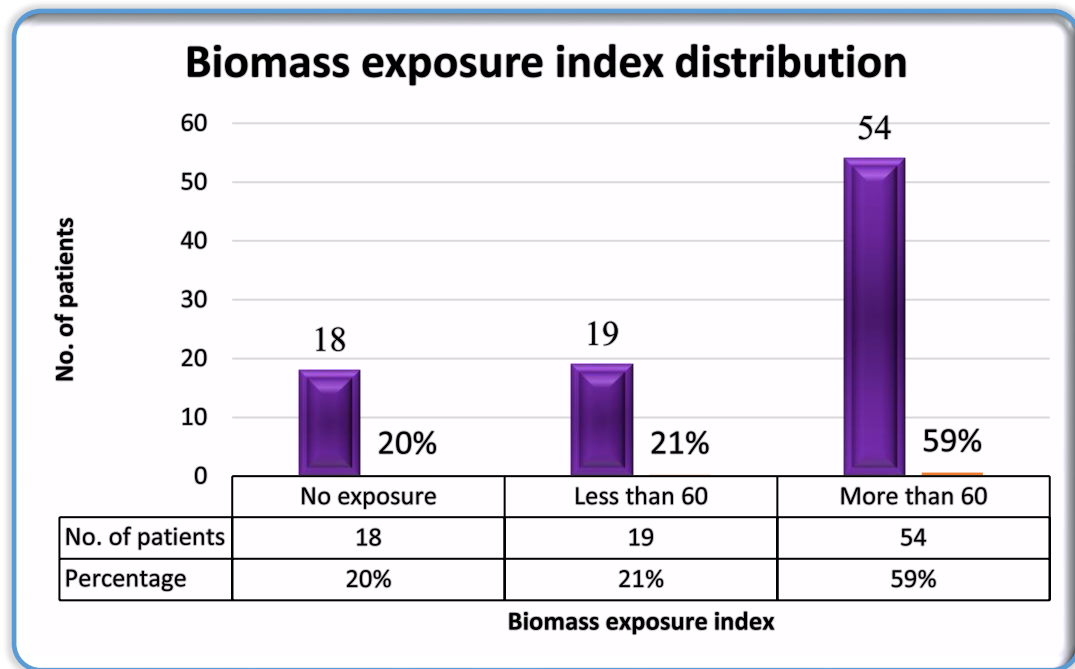


Biomass Exposure of the study population

In my study population of the 91 females, 73 had exposure to biomass fuel during cooking. Number of years of biomass exposure ranged from a minimum of 5 years to a maximum of 45 years with a mean value of 27.2 years in biomass exposed individuals. We also measured the biomass exposure index as suggested by Behera et al which is a more precise index of the intensity of exposure. It ranged from 0 to 161. As indicated by P. Mahesh et al (threshold for development of chronic bronchitis) we divided the biomass exposure group into less than 60 and more than 60. 54 (59.3%) of our patients were in the high index group (>60) and only 19 patients in the low index group (<60), suggesting longer years / hours spent in cooking. We also saw a decrease in the

trend in the usage of biomass and shift to LPG usage due to free schemes by the government.

Fig 19: Distribution of patients by Biomass exposure index



Prevalence of smoking and second hand smoke exposure in our study group

When we questioned about smoking only 3 females had accepted to smoking and all were in the moderate smoking index (100 - 300) group.. They were smoking around 8 to 15 cigarettes per day. In these 3 patients 1 had presented with lung cancer.

On the other hand we found that 40 (44%) of our patients had exposure to second hand smoke. In these 40, for 32 patients their spouses were heavy smokers with smoking index of greater than 300.

Fig 20: Distribution in patients by prevalence of smoking

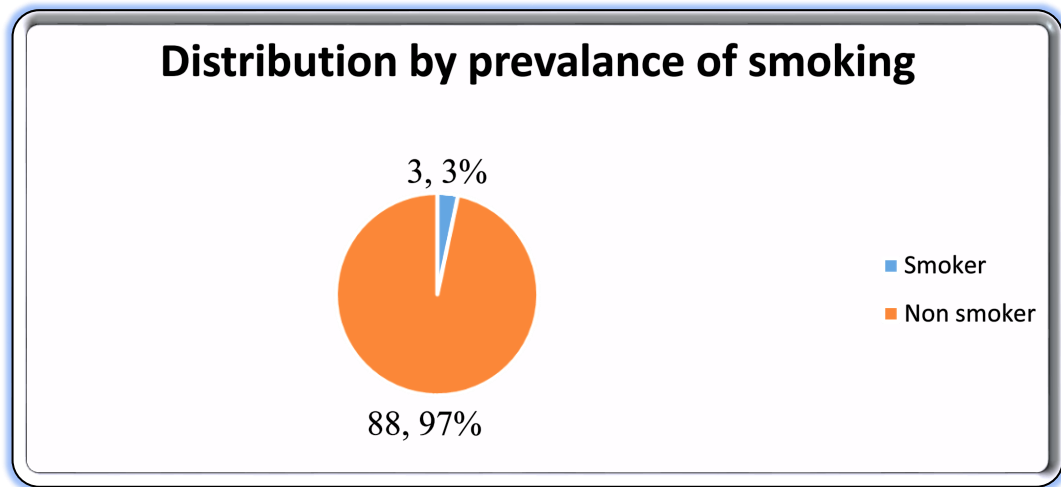
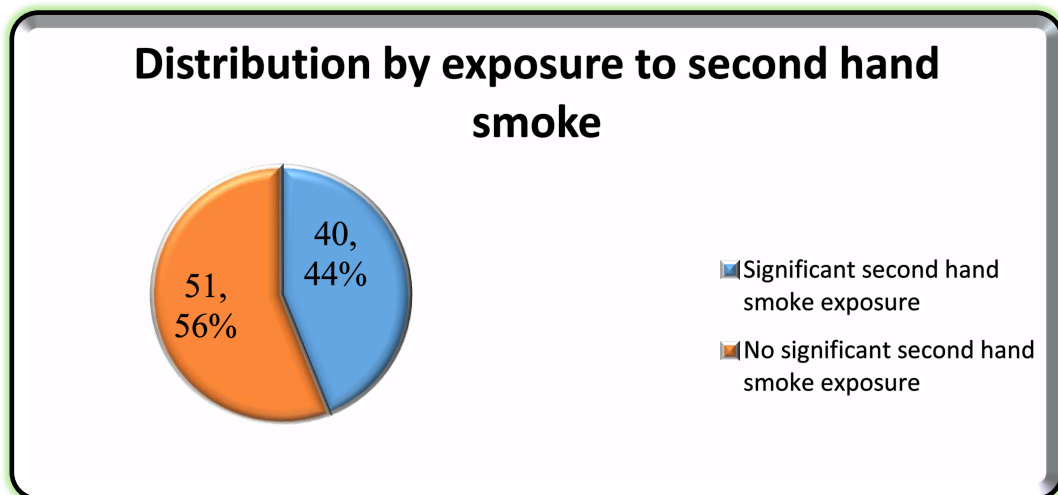


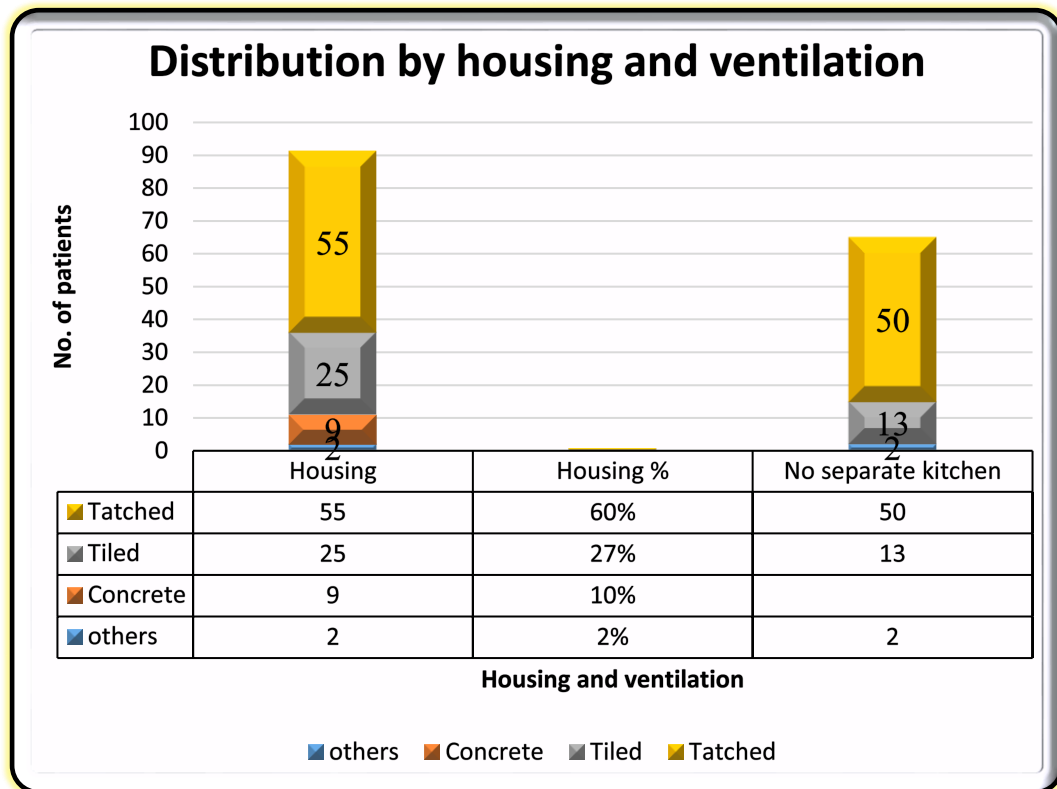
Fig 21: Distribution in patients by exposure to second hand smoke



Housing and ventilation

In our study population 55 (60.4%) of them lived in thatched roof with no separate kitchen, 25 (27.5%) of them lived in tiled houses and 9 (9.9%) lived in concrete houses. Other 4 lived in sheet houses. In this group 65 (70.3%) of them had no separate kitchen and with no windows and ventilation. They cooked, ate and slept in the same room increasing their exposure to biomass

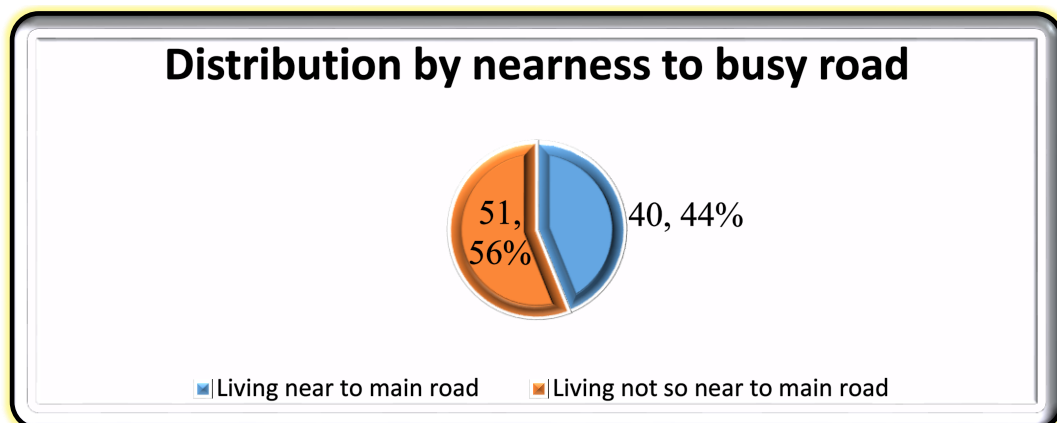
Fig 22: Distribution of patients by housing and ventilation



Environmental exposure due to nearness to busy road

In our study population 40 (44%) of them lived on the main road with heavy traffic including buses.

Fig 23: Distribution of patients by environmental exposure due to nearness to busy road

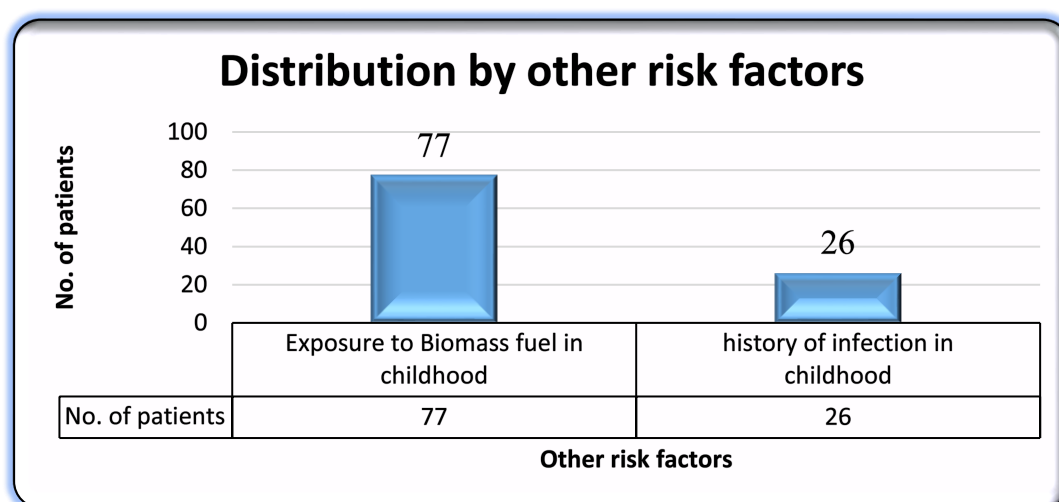


Prevalence of other risk factors in our study group

Since exposure in biomass in childhood plays a role in airway remodelling and later might lead to respiratory diseases we found that 77 (84.6%) had exposure to biomass fuel in less than 12 years.

In our study group 26 (28.5%) of our patients gave history of infections in their childhood, had several hospital visits in their childhood.

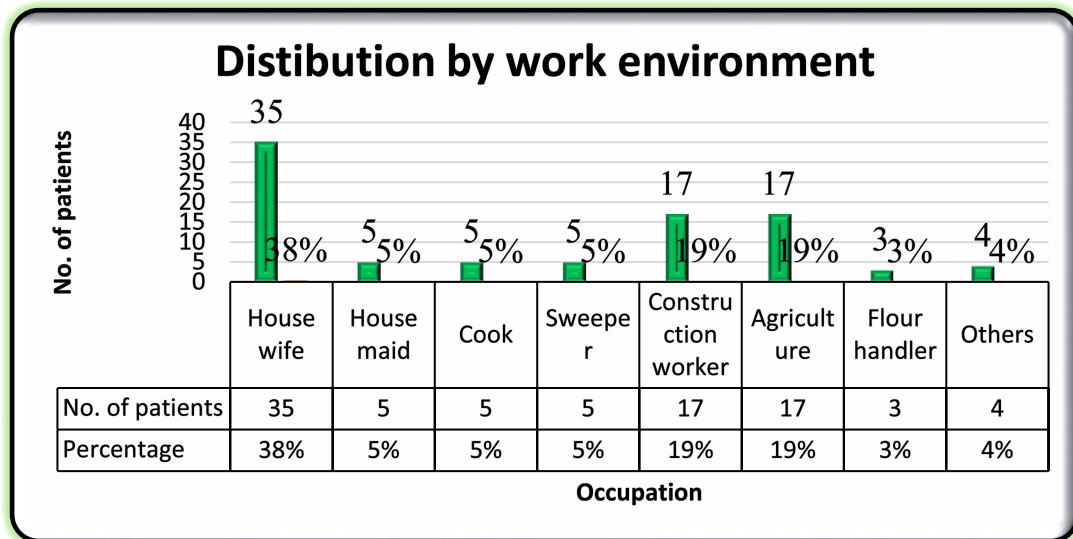
Fig 24: Distribution in patients by prevalence of other risk factors



Work environment of the study population

In our study group 35 (38.5%) of them were home makers, while the most common occupations were construction work and agricultural work, 17 (18.6%) of them doing each work. 3 of them were in occupations involving handling flour, either packing or sieving. 5 (5.4%) were cooks, another 5(5.4%) were sweepers and another 5(5.4%) were housemaids.

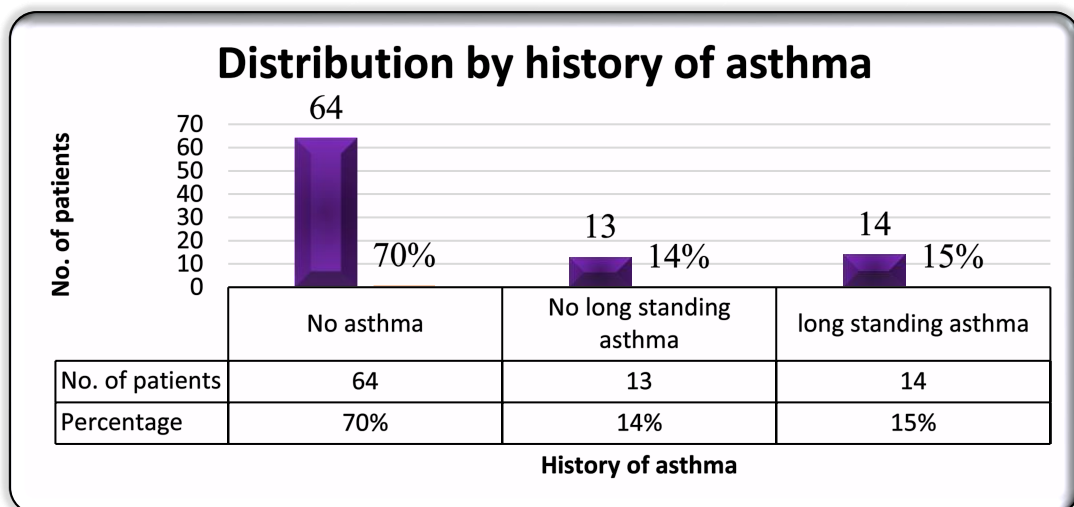
Fig 25: Distribution of patients by work environment



Asthma prevalence in the population

History of physician diagnosed asthma was studied in our group. 64 (70.3%) of them had no history of asthma. 14 (15.4%) of our patients gave history of asthma for more than 10 years and 13 (14.3%) gave history of asthma of less than 10 years.

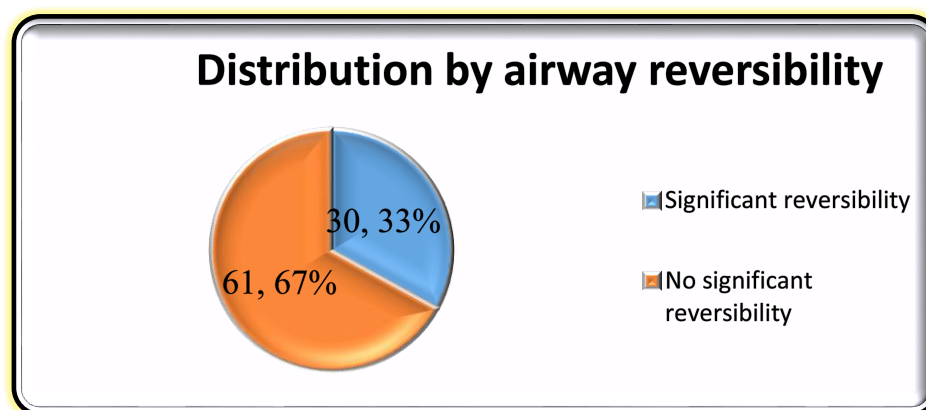
Fig 26: Distribution of patients by history of asthma



Significant reversibility in the population

In our group 91 patients, 30 had a significant reversibility of 12% and 200 ml in FEV1 after a bronchodilator indicating an asthma overlap. In these 30 patients we further observed that 21 belonged to airway predominant ct phenotype and the remaining 9 were mixed phenotype.

Fig 27: Distribution in patients by airway reversibility



EOSINOPHILS REVERSIBILITY ON BRONCHODILATOR:

The mean eosinophil % as well as AEC were higher in those subjects who demonstrated a reversibility on bronchodilators than those whose FEV1 values were not reversible post bronchodilators. This association of increased peripheral eosinophils with patients who had reversibility on bronchodilators was found to be statistically significant suggesting a presence of 36.2% asthma overlap in our total number of 91 cases.

Table 14: Association of eosinophils with reversibility on bronchodilators (n=91).

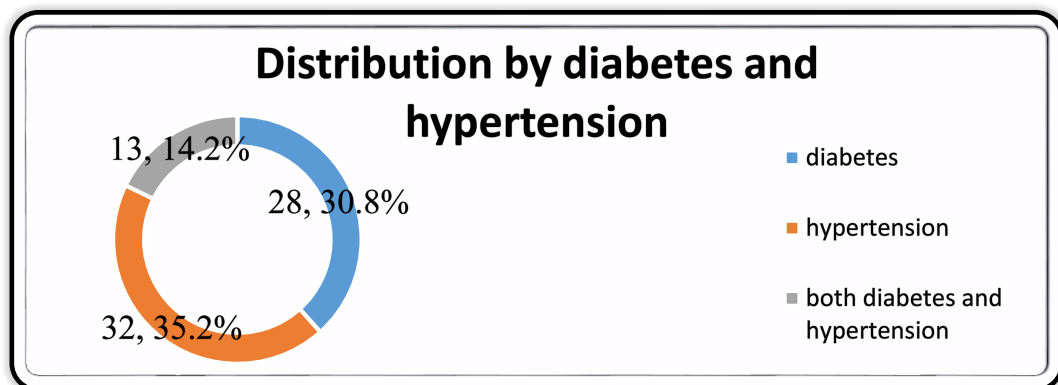
Eosinophils	Reversibility on bronchodilators		independent T test	
	reversible Mean (SE)	irreversible Mean (SE)	t value	<i>p</i> value
Mean eosinophil %	9.85 (0.86)	7.16 (0.46)	2.752	0.008
Mean AEC	786.83 (88.68)	473.42 (51.26)	3.289	0.001

Co morbid illnesses

Diabetes and Systemic Hypertension

More than one third of our patients had been diagnosed with diabetes or hypertension or both. In our study group 28 (30.8%) had diabetes and 32 (35.2%) had systemic hypertension. In this group, 13 patients had both diabetes and hypertension. In the female COPD patients 9.8% gave history of ischemic heart diseases.

Fig 28: Distribution of patients by diabetes and hypertension



Presence of Pulmonary Hypertension in our patients

Presence and severity of pulmonary hypertension correlated with severity of airflow obstruction. ECG and Echocardiogram done and showed the following results

Table 15 : Prevalence of Pulmonary Hypertension

Pulmonary Hypertension	No. of patients	% of patients
Mild	5	5.4
moderate	2	2.1
With right heart failure	1	1

Table 16: Association of Pulmonary hypertension with severity of airflow obstruction

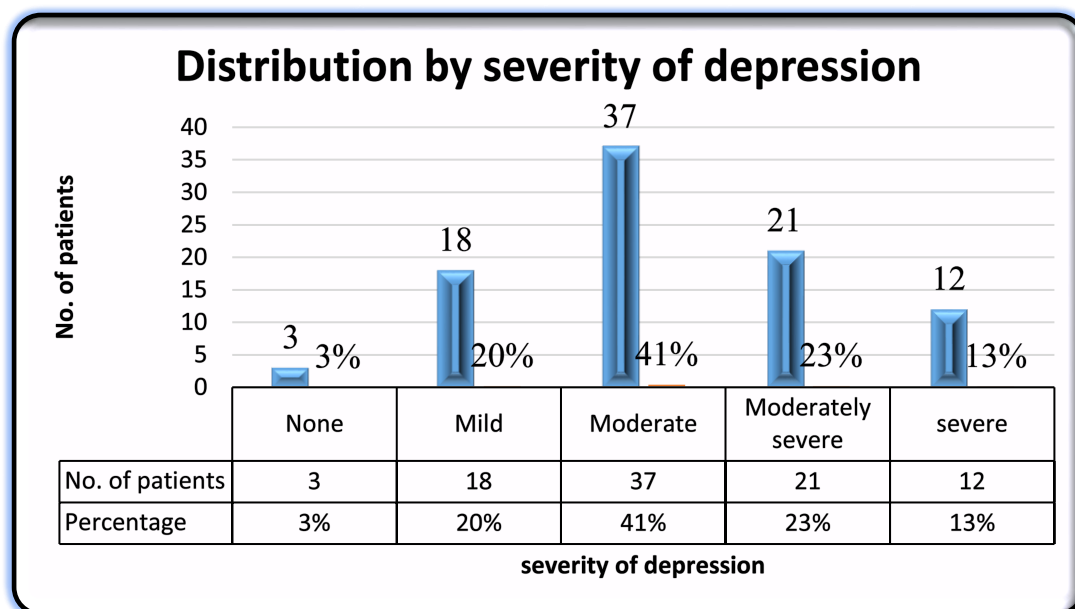
Co-morbid conditions		Degree of airflow obstruction			Fisher's Exact test
		moderate n (%)	severe n (%)	very severe n (%)	
Pulmonary hypertension	present	2 (3.4)	4 (13.8)	2 (50.0)	$\chi^2 = 8.844$, $df = 2$, $p = \mathbf{0.008}$

Presence and severity of depression in our patients

Astonishingly only 3 patients had no symptoms based on PHQ9 questionnaire indicating that 96.7% of patients had some forms of depression. Majority of the patients belong to the moderate symptoms of depression, almost 37 (40.7%). 21 (23.1%) patients had moderately severe symptoms of

depression .In our study group, 18 (19.1%) patients had mild symptoms of depression. Only 12 patients had very severe symptoms of depression.

Fig 29: Distribution of patients by severity of depression



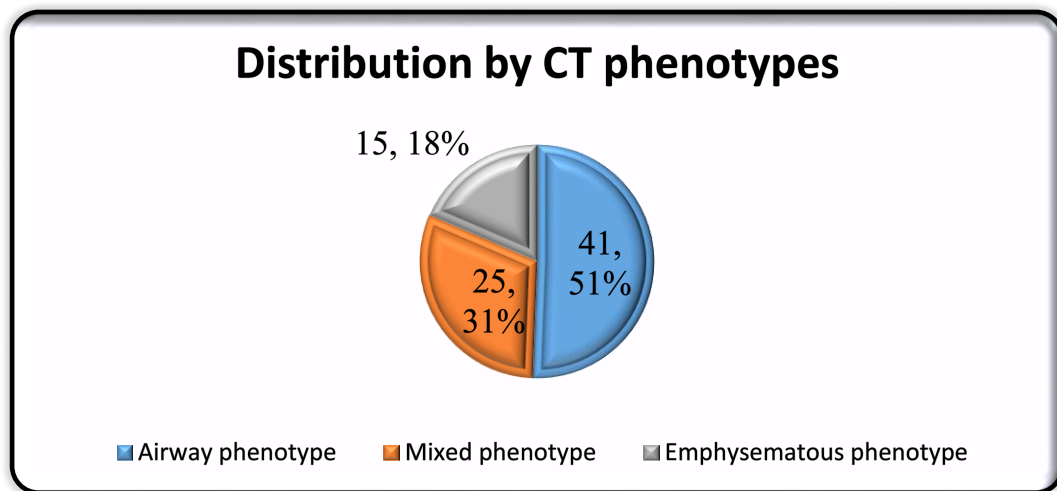
CT phenotypes in our population

In our group of 91 patients,HRCT could not be taken in 10 patients as 7 of them were not willing to do it and 3 of them did not come back with the report.Phenotyping was done by experienced radiologists based on % of low attenuation areas, bronchial wall thickening and airtrapping. Of the remaining 81 patients, 41 (45.1%) had airway predominant phenotype, 15 (16.5%) had emphysematous predominant phenotype and 25(27.5%) had mixed phenotype. 14 patients who had bronchiectasis have been included in the airway predominant phenotype.

Table 17: Distribution Of CT Phenotypes in our patients

CT Phenotype	Frequency (n = 91)	% of patients
Airway	41	45.1
Emphysematous	15	16.5
Mixed	25	27.5

Fig 30: Distribution in patients by CT phenotypes



UNIVARIATE ANALYSIS TO FIND FACTORS ASSOCIATED WITH

DEGREE OF AIRFLOW OBSTRUCTION:

SOCIO DEMOGRAPHIC FACTORS & DEGREE OF AIRFLOW OBSTRUCTION:

Among the socio demographic variables, age of the study subjects was found to have significant association ($p=0.012$) with degree of airflow obstruction. Other factors like education, occupation, socioeconomic status, type of housing, separate kitchen and nearness to busy road were not significantly associated with degree of airflow obstruction. These results are shown in the following Table .

Table 18 : Association of socio demographic factors with degree of airflow obstruction (n=91).

Socio-demographic profile		Degree of airflow obstruction			Fisher's Exact test
		moderate n (%)	severe n (%)	very severe n (%)	
Age (in years)	41 - 50	22 (37.9)	5 (17.2)	0 (0)	$\chi^2 = 14.091$, df = 6, $p = 0.012$
	51 - 60	23 (39.7)	10 (34.5)	1 (25.0)	
	61 - 70	13 (22.4)	13 (44.8)	2 (50.0)	
	>70	0 (0)	1 (3.4)	1 (25.0)	
Education	Illiterate	39 (67.2)	15 (51.7)	3 (75.0)	$\chi^2 = 2.224$, df = 2, $p = 0.382$
	Literate	19 (32.8)	14 (48.3)	1 (25.0)	
Occupations with possible exposure to allergens	Minimal exposure	25 (43.1)	12 (41.4)	2 (50.0)	$\chi^2 = 0.267$, df = 2, $p = 1.000$
	Considerable exposure	33 (56.9)	17 (58.6)	2 (50.0)	
Socio economic status	Upper middle	1 (1.7)	0 (0)	0 (0)	$\chi^2 = 2.554$, df = 6, $p = 1.000$
	Lower middle	6 (10.3)	3 (10.3)	0 (0)	
	Upper lower	47 (81.0)	24 (82.8)	4 (100)	
	Lower	4 (6.9)	2 (6.9)	0 (0)	
Type of housing	katcha	35 (60.3)	17 (58.6)	3 (75.0)	$\chi^2 = 0.387$, df = 2, $p = 1.000$
	non katcha	23 (39.7)	12 (41.4)	1 (25.0)	
Separate kitchen	present	18 (31.0)	8 (27.6)	1 (25.0)	$\chi^2 = 0.233$, df = 2, $p = 0.919$
	absent	40 (69.0)	21 (72.4)	3 (75.0)	
Nearness to busy road	present	24 (41.4)	13 (44.8)	3 (75.0)	$\chi^2 = 1.670$, df = 2, $p = 0.424$
	absent	34 (58.6)	16 (55.2)	1 (25.0)	

FACTORS AFFECTING THE SEVERITY OF THE DISEASE

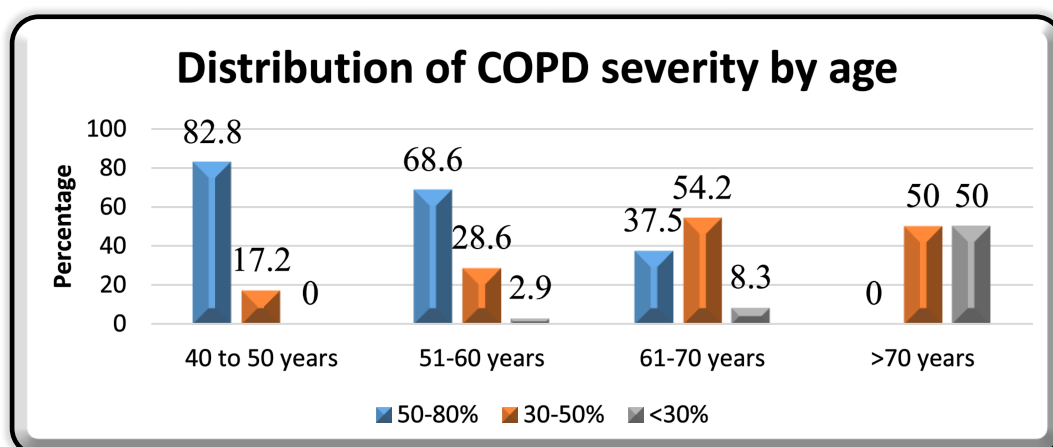
AGE AND SEVERITY OF AIRFLOW OBSTRUCTION:

The mean age (in years) among study subjects with moderate, severe and very severe degree of obstruction was found to be 54.60, 59.48 and 65.25 respectively. It was observed that as the age of the study subjects increased, the degree of airflow obstruction also increased. This difference in mean was found to be statistically significant ($p=0.002$) as depicted in Table .

Table 19 : Association of age with degree of airflow obstruction (n=91).

Variable	Degree of airflow obstruction			one way ANOVA	
	moderate	severe	very severe	F value	<i>p</i> value
	Mean (SE)	Mean (SE)	Mean (SE)		
Age (in years)	54.60 (0.96)	59.48 (1.53)	65.25 (2.68)	6.661	0.002

Figure 31: Bar graph showing distribution of COPD severity across age groups- Age group wise



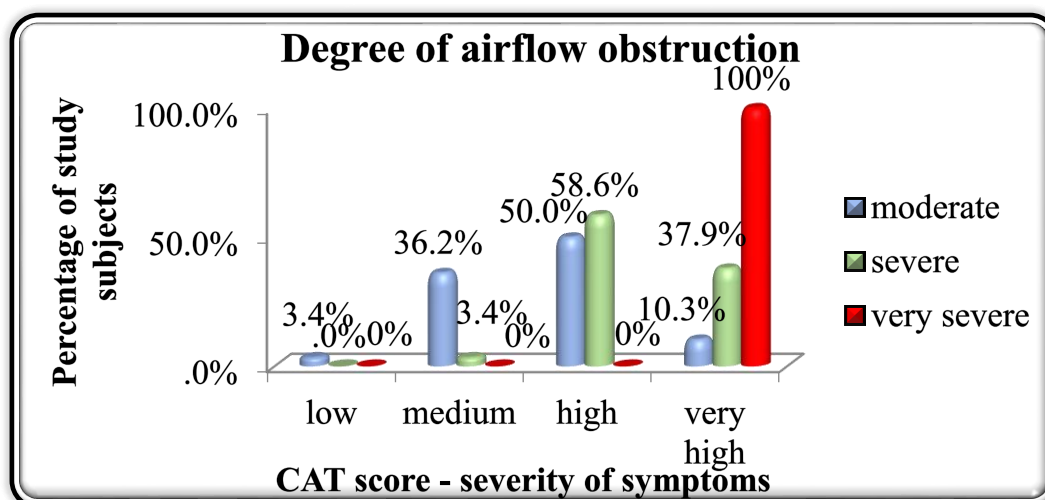
CORRELATION OF CAT SCORE & DEGREE OF AIRFLOW OBSTRUCTION:

When the severity of symptoms was assessed with COPD Assessment Test (CAT), it was observed to be significantly associated with the degree of airflow obstruction ($p=0.000$). This can be studied in Table .

Table 20 : Association of CAT scoring of symptom severity with degree of airflow obstruction (n=91).

CAT scoring of symptom severity	Degree of airflow obstruction			Fisher's Exact test
	moderate n (%)	severe n (%)	very severe n (%)	
low	2 (3.4)	0 (0)	0 (0)	$\chi^2 = 27.576,$ $df = 6, p = \mathbf{0.000}$
medium	21 (36.2)	1 (3.4)	0 (0)	
high	29 (50.0)	17 (58.6)	0 (0)	
very high	6 (10.3)	11 (37.9)	4 (100.0)	

Fig 32: CAT score grading and degree of airflow obstruction



The median CAT score for symptom severity was found to be increasing as the degree of airflow obstruction increases. Difference in this median was observed to be statistically significant ($p=0.000$) as depicted in Table .

Table 21 : Association of CAT scoring of symptom severity with degree of airflow obstruction (n=91).

Variable	Degree of airflow obstruction			Kruskal Wallis Test
	moderate	severe	very severe	
	Median (IQR)	Median (IQR)	Median (IQR)	
CAT score	21 (11)	29 (5)	31 (2)	$p = 0.000$

Figure 33: Box plot showing distribution of CAT score among FEV ranges

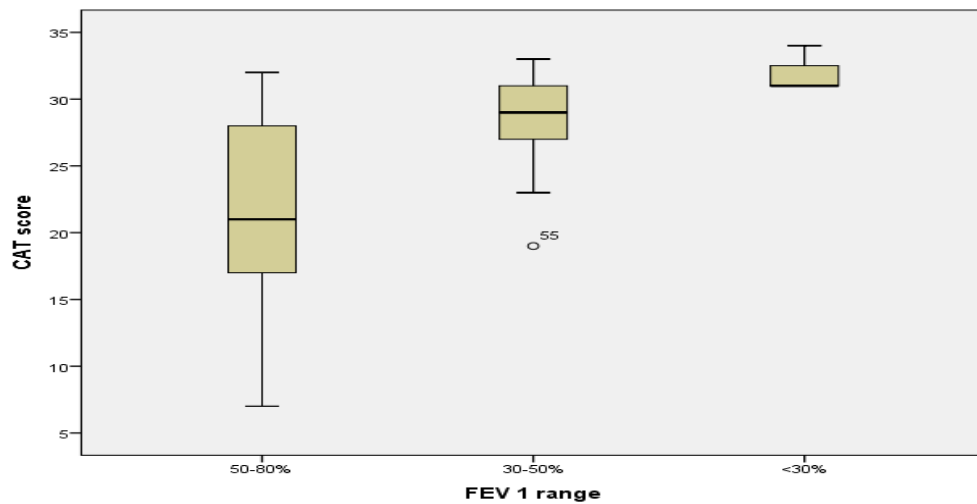
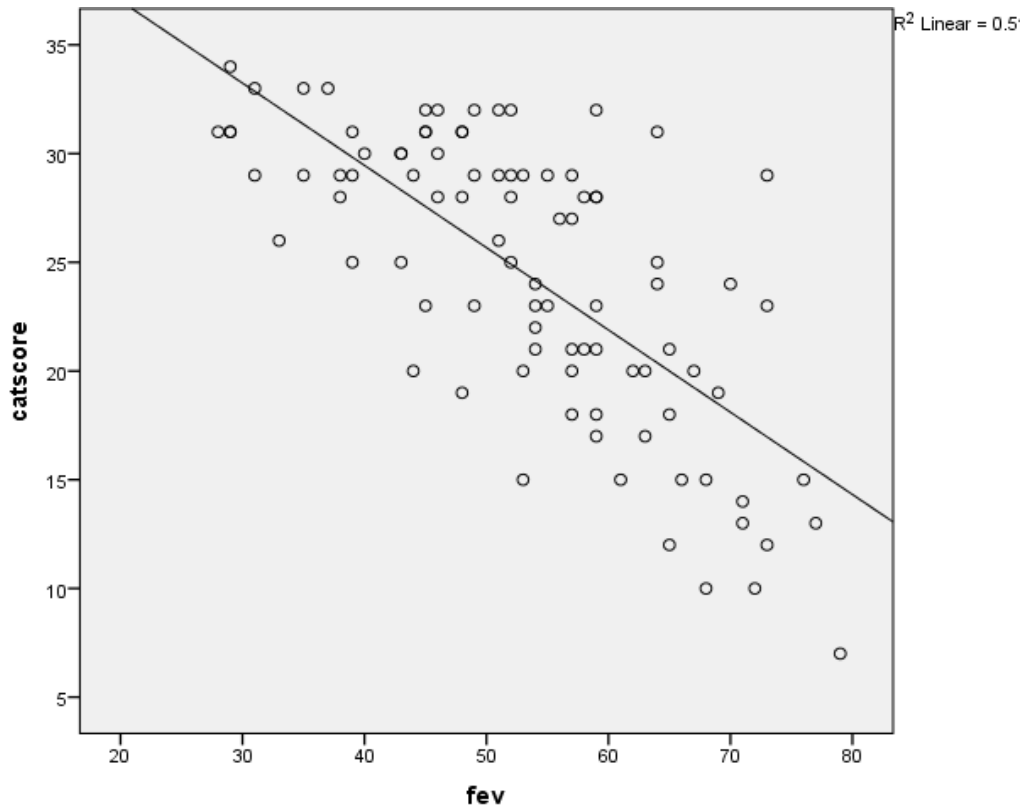


Figure 34: Scatter plot showing distribution CAT score and FEV1



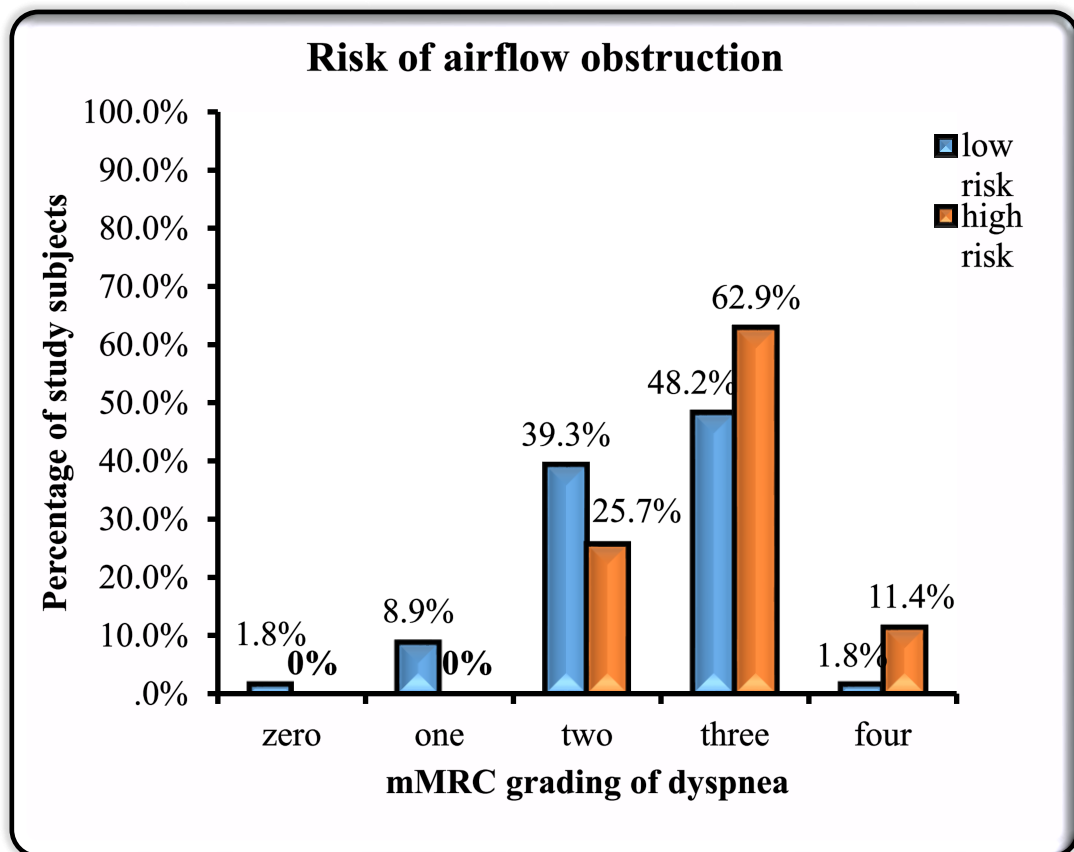
CORRELATION OF mMRC GRADING OF DYSPNEA AND SEVERITY OF AIRFLOW OBSTRUCTION

Breathlessness of the study subjects was assessed by mMRC grading of dyspnea. Percentage of predicted FEV1 values were classified as low risk (<50%) and high risk (>50%) of airflow obstruction. There was a significant association ($p=0.039$) noted between mMRC grading of dyspnea and the risk of airflow obstruction, which can be observed from Table .

Table 22 : Association of mMRC grading of dyspnea with risk of airflow obstruction (n=91).

mMRC grading of dyspnea	Risk of airflow obstruction		Fisher's Exact test
	low risk n (%)	high risk n (%)	
Grade – 0	1 (1.8)	0 (0)	$\chi^2 = 8.796, df = 4,$ $p = 0.039$
Grade – 1	5 (8.9)	0 (0)	
Grade – 2	22 (39.3)	9 (25.7)	
Grade – 3	27 (48.2)	22 (62.9)	
Grade – 4	1 (1.8)	4 (11.4)	

Fig 35 : mMRC grading and risk of airflow obstruction



The median mMRC grade of dyspnea was observed to be higher in patients with high risk of airflow obstruction (3.0) when compared to patients with low risk of airflow obstruction (2.5). This difference in median was found to be statistically significant ($p=0.005$) as depicted in Table .

Table 23 : Association of mMRC grading of dyspnea with risk of airflow obstruction (n=91).

Variable	Risk of airflow obstruction		Mann-Whitney Test
	low risk Median (IQR)	high risk Median (IQR)	
mMRC grading of dyspnea	2.5 (1.0)	3.0 (1.0)	$p = 0.005$

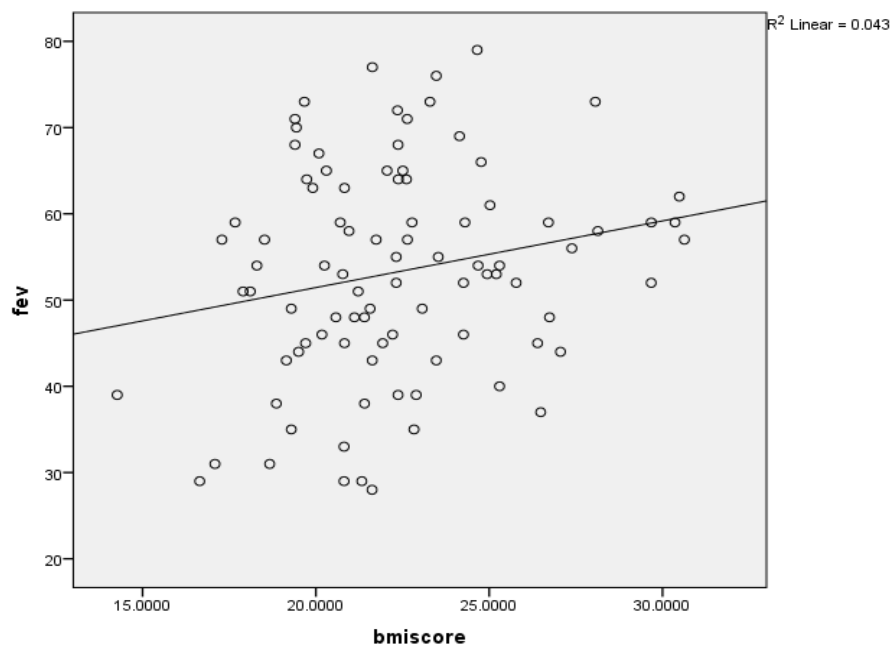
NUTRITIONAL STATUS & DEGREE OF AIRFLOW OBSTRUCTION:

Height and weight of the study subjects were measured and their nutritional status was assessed by calculating the body mass index (BMI). It was observed that subjects at higher risk of airflow obstruction had a significantly ($p=0.020$) lower BMI (21.45 kg/m²) as compared to subjects at lower risk of airflow obstruction (22.95 kg/m²). This observation is depicted in the Table below.

Table 24 : Association of BMI with risk of airflow obstruction (n=91).

Nutritional status	Risk of airflow obstruction		independent T test	
	low risk	high risk	t value	p value
	Mean (SE)	Mean (SE)		

Fig 36 : Scatterplot showing distribution of BMI and FEV1



BIOMASS EXPOSURE & DEGREE OF AIRFLOW OBSTRUCTION:

Biomass exposure index was calculated by multiplying the number of years of cooking with the number of hours spent in cooking on a whole day. Percentage of predicted FEV1 values were classified as low risk (<50%) and high risk (>50%) of airflow obstruction. From table , it can be observed that as the number of years of exposure to biomass as well as the biomass exposure index increased, the risk of airflow obstruction also increased. This association of biomass exposure and risk of airflow obstruction was found to be statistically significant.

Table 25 : Association of biomass exposure with risk of airflow obstruction (n=91).

Biomass exposure	Risk of airflow obstruction		independent T test	
	low risk Mean (SE)	high risk Mean (SE)	t value	p value
	Years of exposure to biomass	18.89 (1.81)		
Biomass exposure index	48.66 (5.13)	68.60 (6.18)	2.484	0.015

FIG : 37 Bar Graph showing association Biomass exposure and Severity of airflow limitation

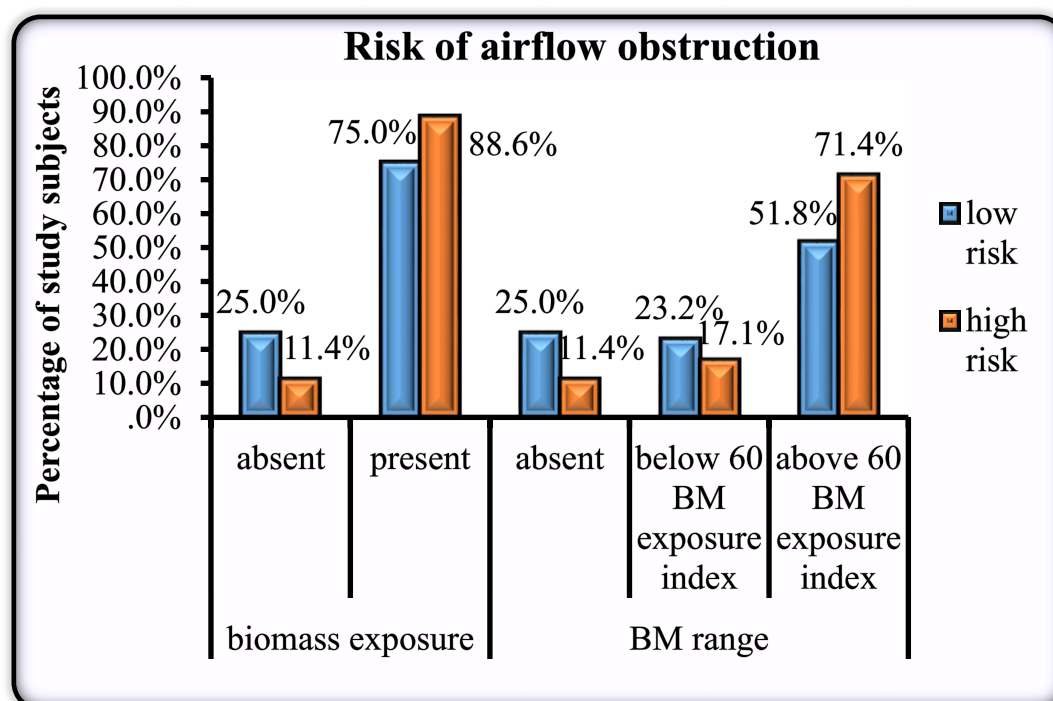
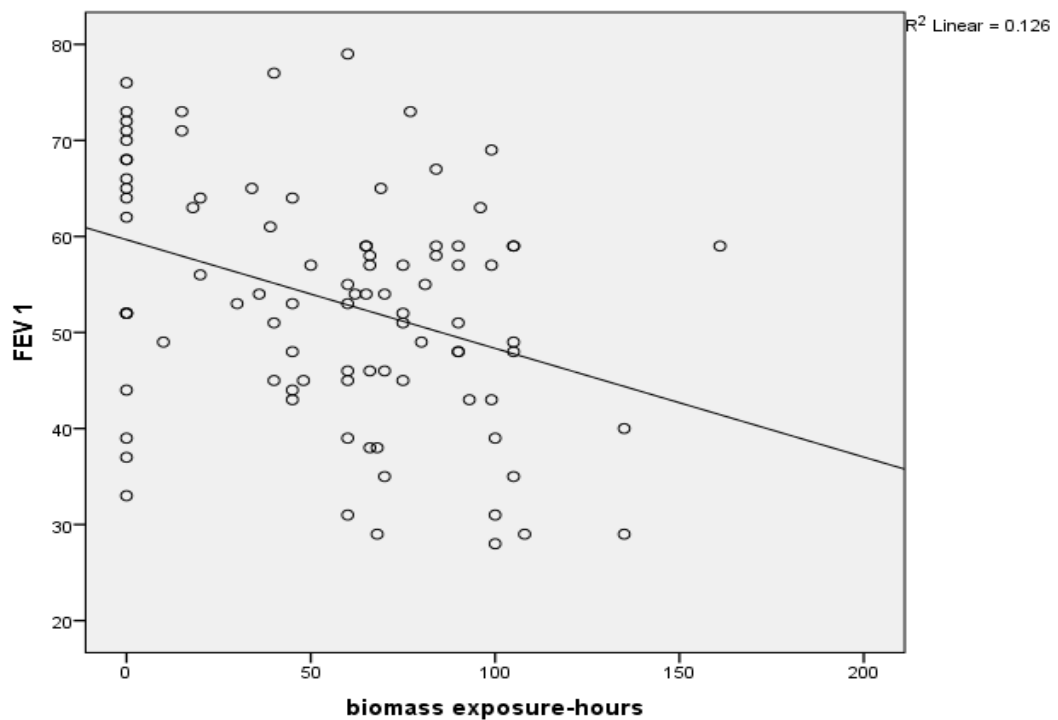


Table 26: Association of biomass exposure index with airflow limitation

Variable	moderate Mean (SE)	severe Mean (SE)	very severe Mean (SE)	one way ANOVA	
				F value	<i>p</i> value
Biomass exposure Index	51.12 (5.00)	60.34 (6.95)	102.75 (13.79)	3.776	0.027

FIG 38: Scatter plot showing distribution of biomass exposure index and

FEV1



EXERCISE CAPACITY & DEGREE OF AIRFLOW OBSTRUCTION:

The exercise capacity of the study subjects was calculated based on “6 Minute Walk Distance” as recommended by American Thoracic Society. It was noted that the mean 6MWD observed value decreased as the severity of airflow obstruction increased. This association was statistically significant ($p=0.000$) as depicted in Table .

Table 27: Association of exercise capacity with degree of airflow obstruction (n=91).

Exercise capacity	Degree of airflow obstruction			one way ANOVA	
	moderate	severe	very severe	F value	p value
	Mean (SE)	Mean (SE)	Mean (SE)		
6MWD observed value	253.10 (6.00)	225.34 (12.27)	150.00 (14.72)	8.766	0.000

FIG 39 Correlation between FEV1 and Observed 6MWD – Scatter plot

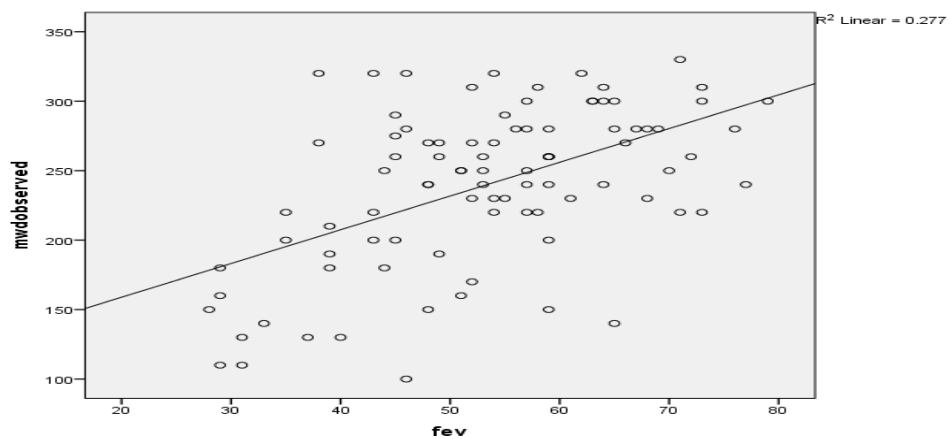
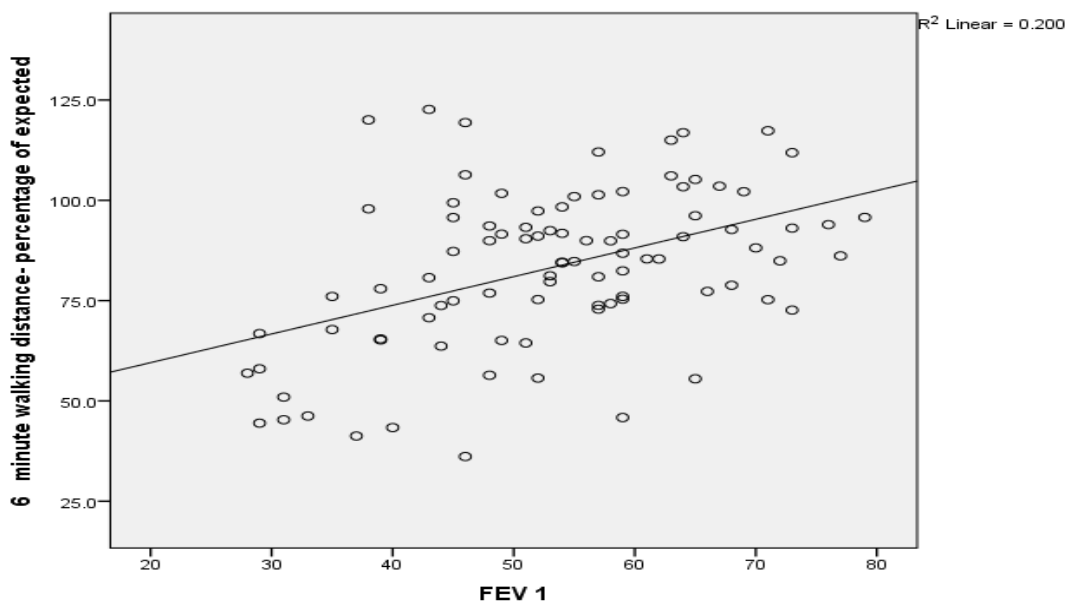


Table 28: Association of 6MWD % with degree of airflow obstruction (n=91).

6MWD RANGE	moderate n (%)	severe n (%)	very severe n (%)	Fisher's Exact test
0 - 50%	2 (3.4)	5 (17.2)	1 (25.0)	$\chi^2 = 15.659$, $df = 4$, $p = \mathbf{0.001}$
51 - 75%	10 (17.2)	8 (27.6)	3 (75.0)	
76 - 100%	46 (79.3)	16 (55.2)	0 (0)	

Figure 40: Correlation between FEV1 and 6 MWD %- Scatter plot



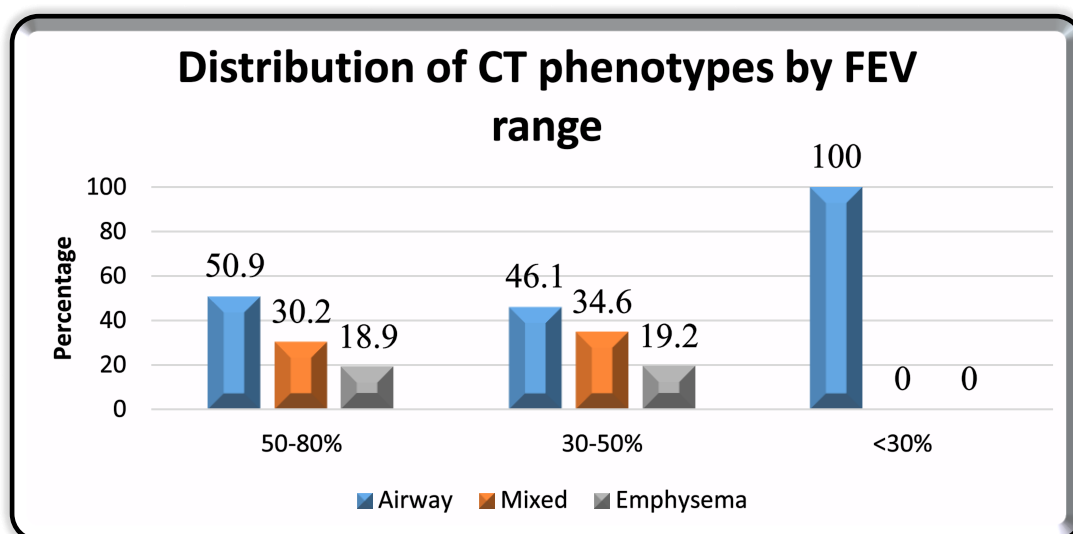
CT PHENOTYPE & SEVERITY OF AIRFLOW OBSTRUCTION:

In the present study, it was observed that the CT phenotype of the study subjects was significantly associated with the risk of airflow obstruction. This is depicted in Table .

Table 29 :Association of CT phenotype with risk of airflow obstruction (n=91).

CT phenotype	Risk of airflow obstruction		Pearson chi-square
	low risk n (%)	high risk n (%)	
Nil	5 (8.9)	5 (14.3)	$\chi^2 = 9.666$, $df = 4$, $p = \mathbf{0.046}$
Airway	20 (35.7)	7 (20.0)	
Bronchiectasis	4 (7.1)	10 (28.6)	
Emphysematous	10 (17.9)	6 (17.1)	
Mixed	17 (30.4)	7 (20.0)	

FIG 41: CT phenotypes across FEV ranges



BIOMASS EXPOSURE & CT PHENOTYPE:

The mean years of exposure to biomass was found to be 20.30, 31.93, 17.00 and 19.67 respectively among those study subjects whose CT phenotype were found to be airway, bronchiectasis, emphysematous and mixed types. This difference in mean years of exposure to biomass among CT phenotype was found to be statistically significant ($p=0.028$), as shown in Table .

Table 30: Association of exposure to biomass and CT phenotype (n=91).

Biomass exposure	Nil Mean (SE)	Airway Mean (SE)	Bronchiectasis Mean (SE)	Emphysematous Mean (SE)	Mixed Mean (SE)	one way ANOVA	
						F value	p value
Exposure to biomass in years	25.00 (5.57)	20.30 (2.66)	31.93 (2.23)	17.00 (3.57)	19.67 (2.62)	2.871	0.028

FIGURE 42: Major Characteristics of the patients

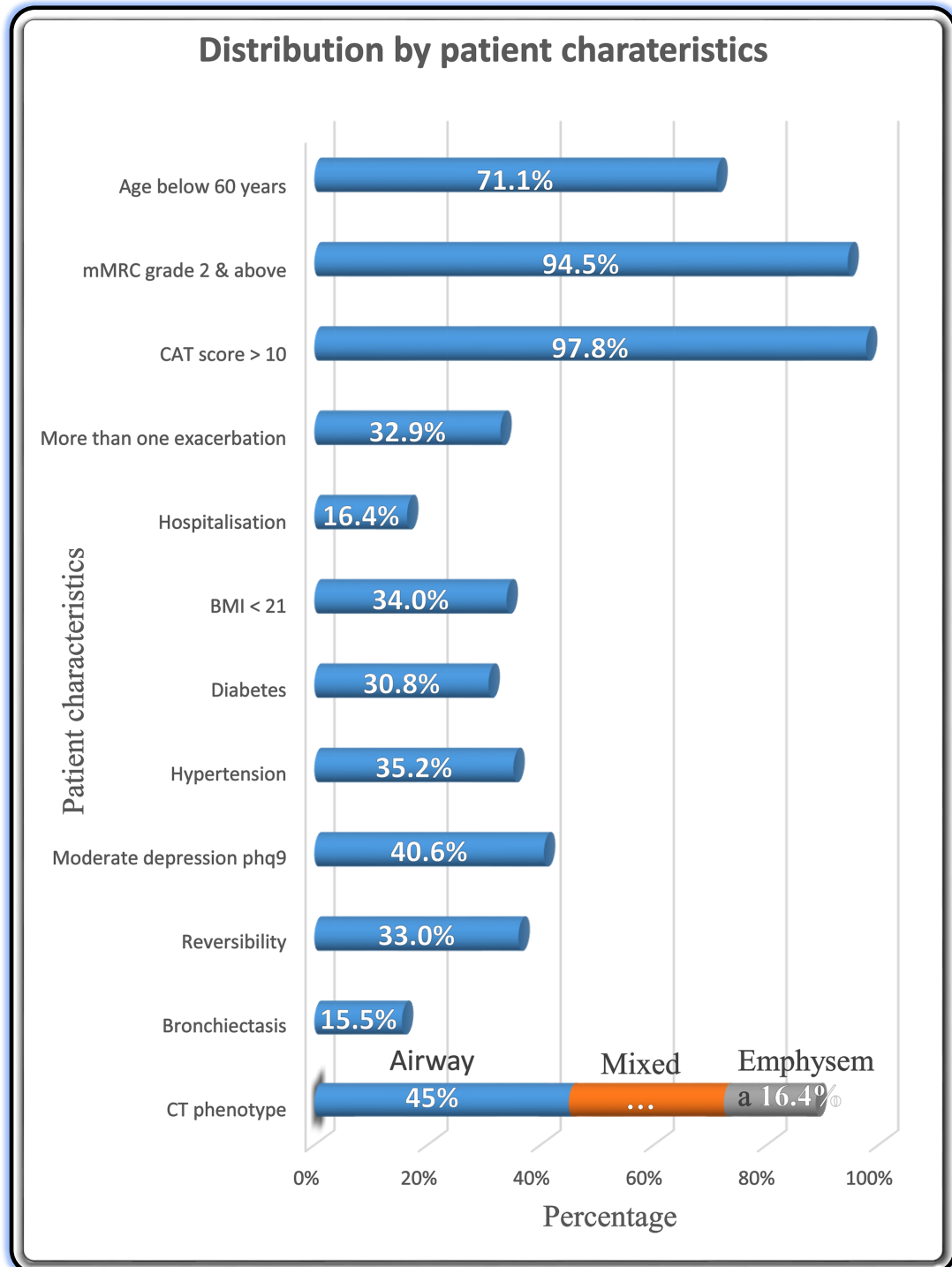
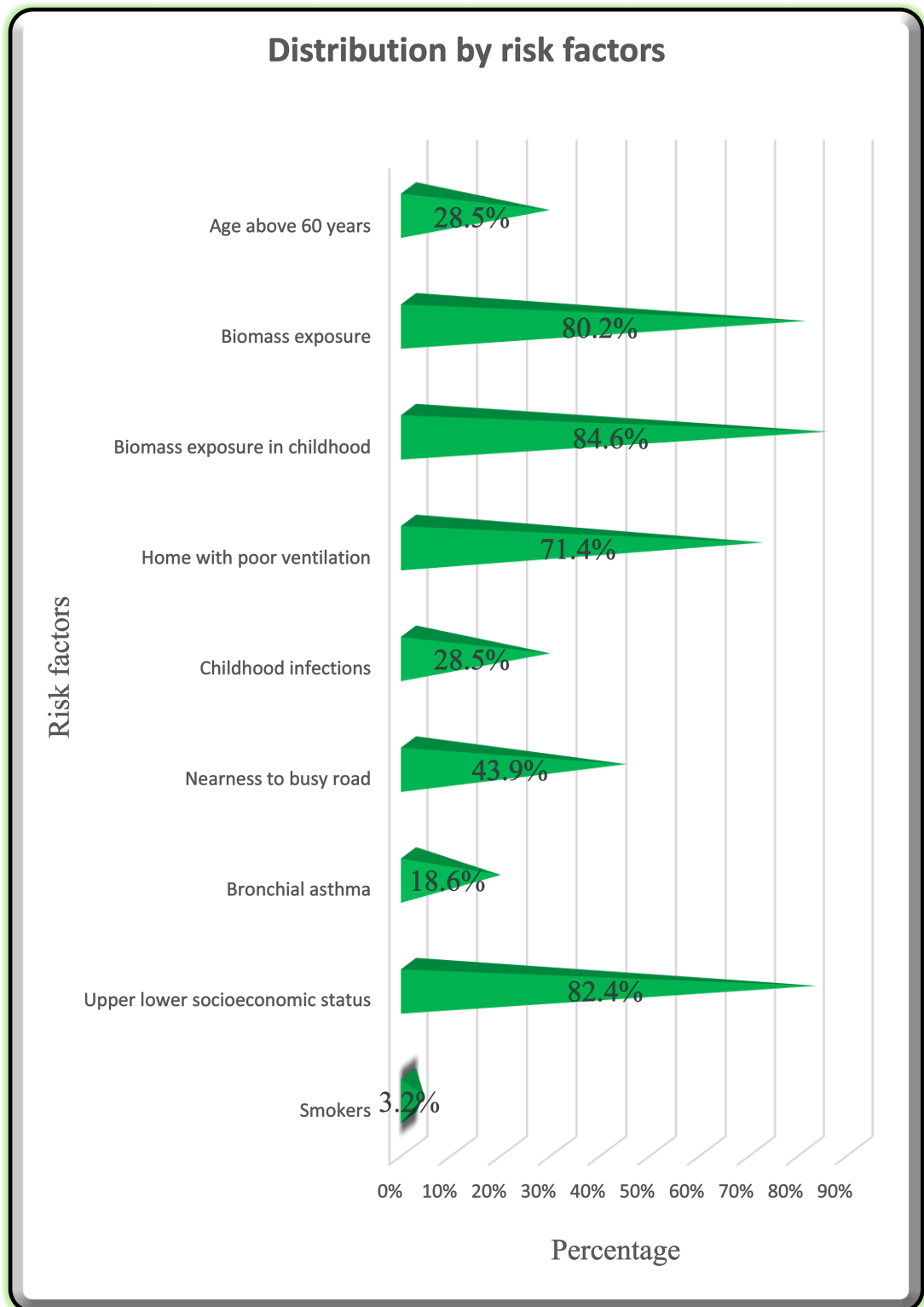


FIGURE 43 :Distribution of Risk Factors



DISCUSSION

This cross sectional study was conducted on 91 female patients aged above 40 years presenting with symptoms suggestive of Chronic Obstructive Pulmonary Disease at the OPD of the department of thoracic medicine in Rajiv Gandhi Government General Hospital and Institute of Thoracic Medicine. This research was done in order to study the prevalence, socio-demographic, clinico-radiological profile and also correlation of COPD assessment test (CAT) and degree of airflow obstruction infemales attending thoracic medicine OPD in this tertiary care centre.

The mean FEV1 of our study population was 53.29% of predicted value. A similar value was observed by *Raherison C et al* in France (52.1%) [85] and *Camp et al* (57.7%) [34]The prevalence of degree of airflow obstruction in the present study was found to be 63.7%, 31.9% and 4.4% respectively for moderate, severe and very severe degrees. None of them were in the mild degree. Possible reasons for this would be the slowly progressing pathology of COPD that renders many asymptomatic for initial years, despite the disease. Another explanation could be lack of awareness by health care providers who may miss a spirometry in the initial stages of the disease and end up in “under-diagnosis”.

Majority (63.7%) of our study subjects were having moderate degree of airflow obstruction. Similarly, more than half of the study population of *Sumer C et al* too was suffering from moderate degree of airflow obstruction as evident by their FEV1 values.[38] *Frei et al* observed the degree of airflow

obstruction as per GOLD staging as II: 64.0%; III: 21.8% and IV: 14.2%. The mean FEV1 was 55.6% of the predicted value.[86]

In our study group 36.1% had significant reversibility of more than 12% and 200ml in FEV1% predicted after bronchodilator. *Dal Negro R W et al* had a prevalence of 22% of ACOS in his study population and also found increased prevalence of bronchiectasis and ACOS in female COPD patients when compared with their male counterparts.[45] In the study by *DeMarco R et al* and *Menezes M et al* had 15 – 25% prevalence of ACOS like *Dal Negro RW et al*. [33,87] The mean eosinophil count of this population was higher than the non reversible population suggesting history of atopy. 80% also gave history of atopy and dust allergy . In this 43.3% of the bronchodilator reversible population also gave history of adulthood asthma.

The mean age of the study subjects in the present study was 56.63 years with a standard deviation of 8.074 years. More than two third of our study subjects were between the age group of 40 to 60 years. This is similar to the observations made by *Jain NK et al*. [88] The mean age in their study was found to be 58.34 ± 9.99 years. In the present study, it was observed that as the age of the study subjects increased, the degree of airflow obstruction also increased significantly ($p=0.002$). This is in line with the observations made by *Sharifi H et al* among the residents of Tehran, where prevalence of COPD increased considerably in elderly individuals.[89] Subjects aged beyond 55 years were two times more likely to suffer from COPD [aOR (95% CI) = 2.17 (1.27- 3.71)] when compared to their younger counterparts. *Hagstad S et al*

noted in their study that age above 65 years was associated with COPD with an aOR of 8.29. [64] *Lee SJ et al* and *Jordan RE et al* too reported advanced age as a risk factor for COPD. [68,91]

In the present study, the most common symptom reported was cough (in 70 patients), followed by sputum production (67), breathlessness (60) and wheeze (26). *Jain NK et al* also observed a similar finding in their study.[88] About 94.7% of their female patients reported cough as a presenting symptom, followed by expectoration (71.3%), fever (37.8%) and pedal edema (12.9%). A slightly contrary observation was noted by *Sharifi H et al* in Tehran, where dyspnea was the most common presenting symptom (21.6%), followed by wheezing (21.0%), sputum production (16.2%) and chronic cough (9.5%).[89]

Percentage of study subjects with mMRC dyspnea grade of 0, 1, 2, 3 and 4 in the present study were 0%, 5.4%, 35.1%, 53.8% and 5.4% respectively. The median mMRC grading of dyspnea in the present study was observed to be higher in patients with high risk of airflow obstruction as described by their FEV1 values. *Ozalevli S et al* in their study, showed that dyspnea scores of patients derived by mMRC grading were significantly linked to their FEV1 values. Significant association between other dyspnea severity scales and spirometry values have been observed by other authors namely *Hajiro T et al*, *Sahebjami H et al* and *Mahler DA et al*. [91,92,93] In fact, *Hajiro T et al* demonstrated better correlations of dyspnea with derangement in the health related quality of life of patients suffering from chronic obstructive pulmonary disorders. [91]

In the present study, almost three-fourths of the study subjects were in the severe or very severe category of the COPD assessment test used to assess the severity of symptoms pertaining to COPD. *Okutan O et al*, in their study observed that out of 90 patients, 24 were in CAT 1, 33 in CAT II, 27 in CAT III and 6 were in CAT IV category. [94]

CAT scoring was significantly associated ($p=0.000$) with the degree of airflow obstruction in the present study. This association of CAT score with the degree of airflow obstruction was also significantly observed by *Sumer C et al*. [38] In their study, a low CAT scoring was observed in mild and moderate COPD cases whereas a high CAT score was seen in severe and very severe cases ($p=0.004$). *Hassan G et al* and *Mackay et al* also observed that patients with severe COPD have significantly ($p<0.032$) higher CAT scores. [36,39] *Ghobadi et al* revealed a significant correlation ($p<0.001$) between GOLD classification of COPD and mean CAT scores. [36]

In the present study, the median CAT score among subjects with 50 – 80% predicted FEV1 was 21, those with 30 – 50% had median CAT score of 29 and those whose predicted FEV1 was less than 30% had median CAT score of 31. A negative correlation was observed between CAT scores and predicted FEV1 values. These results are in line with those of *Chai JJ et al* [95] ($r = -0.567$, $p<0.01$), *Miyazaki et al* [96] ($r = -0.258$, $p< 0.001$), *Dal Negro RW et al* [97] ($r = -0.21$, $p<0.001$) as well as those of *Mishra AR et al*. [98] From the observations by all these authors, it can be affirmatively concluded that health impairment in COPD is associated with high CAT scores and lower values of

FEV1.CAT scoring thus provides a useful tool for screening for COPD patients, especially in settings where availability of spirometry is doubtful. However, the prognostic value of the COPD assessment test needs to be ascertained by a prospective follow-up study.

In the present study, the average number of exacerbations among moderate, severe and very severe degree of obstruction was found to be 0.90, 1.28 and 3.25 respectively. It was evident that as the degree of airflow obstruction increased, the number of exacerbations too increased. *Mackay AJ et al* [39] observed that those with frequent exacerbations had a significantly ($p=0.025$) higher CAT score (19.5) when compared to those with less frequent exacerbations (16.8). During the exacerbations, it was also noted by the authors that the rise in CAT scores were significantly associated with a fall in FEV1 ($p=0.032$). The authors concluded that CAT scores increase during exacerbations and hence reflect the severity of the underlying disease. *Okutan O et al* [94] also observed a similar correlation of exacerbations with CAT scoring in their study.

Marco et al,[33] in their study on European population observed that respiratory infections in childhood and a family history of asthma were significant risk factors of COPD. An adjusted incidence rate ratio of 1.88 (1.02, 3.46) and 1.95 (1.25, 3.04) were observed respectively for childhood respiratory infections and familial asthmatic history for the incidence of chronic obstructive pulmonary disorders. But in the present study, such a significant association could not be demonstrated. Possible explanations may

be due to differences in rates caused by genetic susceptibility or geographic variations.

In our study population out of the 35 who were homemakers, 28 had exposure to biomass fuel at home thus exposing them for longer hours. In our study 17 were constructional workers, another 17 agricultural workers, 5 were sweepers, 5 were cooks, 5 housemaids and 4 were flour handlers. These exposures were proven as significant risk factors for the development of COPD by *John Dement et al*[69]in his study on airways obstruction among construction workers, *Ye M et al* in his literature showed associations between chronic bronchitis and/or COPD and exposures to pesticides/herbicides and grain dusts,[70] *Hamdy A et al* showed significantly higher prevalence of chronic obstructive pulmonary disease in mill workers was work-related (40% vs. 0.0%; $P < 0.0001$) [71,72]]. Though none of the occupation showed significant correlation with severity of the disease may be due to the relatively smaller sample size of the present study or difference in the methodology when compared to those studies.

In the present study, co morbid conditions such as diabetes, hypertension and psychiatric illness did not associate significantly with the airflow obstruction. About 9.8% gave history of ischemic heart disease . In COMCOLD study 20.6% had evidence of symptomatic heart disease.[86] *Mackay AJ et al*[39]in their study did not notice any significant difference between the mean CAT scores among the study subjects having co-morbid conditions such as ischemic heart diseases, cyanotic heart diseases,

hypertension, diabetes, chronic kidney disease, obstructive sleep apnea and obesity.

Raherison C et al[85]in France observed among their women study subjects, the prevalence of co-morbid conditions such as arterial hypertension (32.9%) osteoporosis (19.6%), dyslipidemia (16.7%) and diabetes (6.5%). Except diabetes and hypertension, other co-morbidities were significantly differing between male and female COPD patients. *Lee et al*[68]observed that bronchiectasis or a history of tuberculosis were strong risk factors for COPD. *Frei et al*[86]observed that among the co-morbid conditions, most commonly prevalent was hypertension (42.3%), and followed by arthritis (29.4%). Diabetes was found to have an impact on health status of COPD patients on univariate analysis. But its significance was lost when analyzed along with other co-morbidities in multivariate analysis.

In the present study, it was noted that Body Mass Index of subjects at higher risk of airflow obstruction (21.45 kg/m^2) was significantly less when compared to those at lower risk of obstruction (22.95 kg/m^2). Being underweight was observed to be associated with a higher risk of COPD.*Marco et al*[33]observed that those with a BMI below 18.5 kg/m^2 were thrice more likely to develop COPD [adjusted incidence rate ratio (95% CI) 3.55 (1.59 – 7.93)] when compared to those with adequate nutritional status. Similar results were noted by *Lee et al*[68] also in their multivariate analysis [aOR (95% CI) 3.1 (1.0 – 9.4), $p = 0.045$]. About 29.5% of the female study subjects of *Raherison C et al*[85]were having a BMI below 21.0 kg/m^2 similar to our study

which had 34% BMI below 21.0 kg/m². Biomass exposed COPD patients had a higher BMI compared to tobacco smoke exposed COPD patients in studies done by both *Ramirez Venegas et al* and *Camp et al*. [102,34]

Verhage et al [99] in their study in Netherlands observed that there was a significant correlation between fat free mass index and diffusion capacity. *PothiratC et al* [100] in their study noted that the fat free mass showed a significant positive correlation with the FEV1% values ($r = 0.214$, $p = 0.019$) and a significant negative correlation with CAT scores ($r = -0.278$, $p = 0.002$) and mMRC gradings ($r = -0.315$, $p < 0.001$). The authors also observed a correlation between body mass index and FEV1%, CAT scores and mMRC scores, but the relation was insignificant.

As per World Health Organization reports, about 4.3 million humans die every year prematurely from illnesses attributable to indoor air pollution due to solid fuels. 22% of these premature deaths are due to COPD. [101] Women exposed to high levels of indoor air pollutants are twice at risk to counter COPD than women who use cleaner fuels. *Hu G et al*. [12] in their meta-analysis, have found that exposure to biomass fuel was a significant risk factor for COPD and chronic bronchitis. A Lancet article estimates that about half of the COPD related deaths in under developed countries are due to biomass exposure and more than three – fourths of these deaths are in women.

In the present study, 73 out of 91 females had a history of exposure to biomass fuel during cooking. The mean biomass exposure index was 56.33 with a standard deviation of 38.71. Almost 60% of our study population were

having a higher biomass index of above 60, indicating a chronic exposure to this indoor air pollutant.

In a meta-analysis conducted by *Guoping Hu et al*, [12] the authors concluded that individuals exposed to biomass smoke were more than two times likely to develop COPD compared to those not exposed [OR (95% CI) 2.4 (1.9 – 3.3)]. Exposure to biomass smoke was established as a risk factor for COPD both in men [OR (95% CI) 4.3 (1.9 – 10.1)] and women [OR (95% CI) 2.7 (2.3 – 3.3)]; both in Asian [OR (95% CI) 2.3 (1.4 – 3.8)] and non Asian population [OR (95% CI) 2.6 (1.7 – 3.7)]; and also both in cigarette smokers [OR (95% CI) 4.4 (1.4 – 4.7)] as well as non smokers [OR (95% CI) 2.6 (2.1 – 3.2)].

Kurmi OP et al [41] noted in their study that the prevalence of airflow obstruction was twice as common among users of solid fuels (8.1%) when compared to users of LPG (3.6%). The difference in airflow obstruction based on the type of fuel used was statistically significant ($p = 0.001$). The Odds ratio of airflow obstruction for females using solid fuels 2.38. The spirometry values were significantly reduced among the biomass users across all ages when compared to non users.

Jain et al[88] noted that 89% of their female study subjects were exposed to biomass fuel smoke. Increased prevalence of airflow obstruction due to solid fuel exposure has been observed in COPD patients by various authors worldwide. It was also observed in the present study that as the number

of years of exposure to biomass as well as the biomass exposure index increased, the risk of airflow obstruction also increased significantly.

Reasons for this observation include the higher vulnerability of Indian women to smoke from biomass and tobacco, or gender differences in health care seeking behavior, or ignorance by the health care provider resulting in a diagnostic delay due to knowledge gap about the usage of spirometry. Our findings are in accordance with those of *Pérez-Padilla et al*[16]and *Ramírez-Venegas et al*.[102] In our study 68.1% of patients were in the normal BMI range. *Raherison et al* [85]had 29.5% were below BMI < 21kg /m² similar to our study. Similar to *Mitra et al* [42]we found a correlation between BMI and GOLD staging based on airflow limitation in our study . There was no correlation between fat free mass and disease severity in our study population probably as most of our patients had a normal BMI.

In the present study, 40 of 91 study subjects gave a history of living at the proximity of a busy road. *Schikowski T et al*[22]in Germany observed a significantly decreased lung function test among women living within 100 meters from a busy road and they were having an OR (95% CI) of 1.79 (1.06 – 3.02) to develop COPD, when compared to those living farther away. Various studies have observed a decreased lung function and increased incidence of respiratory diseases due to proximity to busy roads .But a significant association was not demonstrated in the present study. This can be due to the relatively smaller sample size of the present study or difference in the methodology when compared to those studies.

Jordan RE et al[90]in their study in England observed that exposure to passive smoking was found to have an increased risk of COPD, with an aOR (95% CI) of 1.05 (0.93 – 1.18) for 1 to 19 hours and aOR (95% CI) of 1.18 (1.01 – 1.39) for 20 or more hours of exposure a week.

Hagstad S et al[64]observed that the prevalence of COPD increased significantly ($p = 0.003$) as the exposure to environmental tobacco smoke (ETS) increased: prevalence was 4.2% in no ETS, 8.0% in ETS ever at home [OR (95% CI) 1.98 (1.2 – 3.2)], 8.3% in ETS at previous work [2.06 (95% CI) 1.3 – 3.3] and almost 14.7% for ETS both at home and work (previous as well as current) [OR (95% CI) 3.94 (1.4 – 11.0)].

Venegas AR et al[102]observed that a lower mean rate of FEV1 decline in the biomass exposed COPD patients than for the tobacco exposed COPD patients (23 vs. 42). About 11% of the tobacco smoke – COPD group were rapid decliners whereas only one patient was found to be a rapid decliner in the biomass exposed group. The authors concluded that rapid rate of FEV1 decline was a feature more of tobacco smoke exposure than of biomass fuel exposure ($p = 0.01$).

Only 3 of 91 study subjects in the present study gave a history of using smoking tobacco, of which one was diagnosed to have lung cancer. On the other hand, it was observed that 40 (44%) of the study subjects had an exposure to second hand smoke. Second hand smoke exposure did not show a significant association with degree of airway obstruction in the present study. In a study done in Korea by *Lee et al*,[68] second hand smoke was significantly

associated with COPD risk in univariate analysis, but failed to emerge as an independent predictor in multivariate analysis. Contrarily, *Wu CF et al*[103]observed exposure to second hand smoke as a risk factor for COPD. Reasons for this may be attributed to the differences in the methodology of these studies. American Thoracic Society, in their official statement, concludes that review of the present literature presents a suggestive evidence of association between second hand smoke exposure and COPD development. [104]

In our study population 45.1% had airway phenotype, 27.5% had mixed phenotypes and 16.5% had emphysematous phenotypes. Out of the 73 who had exposure to the biomass 6 of them did not take HRCT. In the biomass exposed individual who had taken CT chest 54.5% had airway phenotype, 29.8% had mixed phenotype and 16.7% had emphysematous phenotype in CT . This is consistent with study by *Camp et al* in Mexican women.[34] The airway phenotype was common in biomass smoke exposed women as biomass fuel is inhaled in consistent tidal respiration and women's respiration is mostly thoracoabdominal. They do not inhale deeply and the particles settle in airways and do not go deeper and result in airway predominant phenotype as opposed to men who inhale an extra volume of air when smoking leading to emphysematous predominant type in men.

SUMMARY

- This is a cross sectional study on 91 female patients aged above 40 years presenting to OPD with symptoms suggestive of COPD.
- The mean FEV1 of the study population was 53.29% of predicted.
- Prevalence of airflow obstruction as defined by GOLD guidelines was as follows: 0% in mild degree (FEV1 > 80% of predicted), 63.7% moderate (FEV1 50% to 80% of predicted), 31.9% severe (FEV1 30% to 50% of predicted) and 4.4% were having very severe (FEV1 < 30% of predicted) degree of airflow obstruction.
- Majority (63.7%) were having moderate degree of airflow obstruction.
- Most of the study subjects (72.1%) were aged below 60 years.
- The most commonly reported symptom was cough (70 patients).
- Dyspnea grade 3 (53.8%) followed by grade 2 (35.1%) was reported by most of the study subjects, as per mMRC grading of dyspnea.
- About half of the study subjects (50.5%) were categorized high as per CAT scoring of COPD symptom severity.
- Majority (82.4%) of the study subjects were belonging to upper lower scale of socio economic status as per modifiedKuppuswamy's classification.
- More than half (63%) of the study subjects were illiterates.

- About 53 study subjects had one or more exacerbations.
- The mean fat free mass of the study population was 31.87.
- About 9.9% of our study subjects were undernourished as per Quetlet index, while 68.1% of them were normal.
- 44% of the study subjects reported living in proximity to a busy road.
- About 15.4% of study subjects gave history of asthma more than 10 years.
- Prevalence of diabetes among the study population was found to be 30.8%
- Prevalence of systemic hypertension was found to be 35.2%
- About 40.7% of the study subjects had moderate depression as per PHQ9 questionnaire.
- 41 study subjects had airway phenotype on CT examination, whereas 25 had mixed phenotype and 15 had emphysematous phenotype.
- 30 study subjects had significant reversibility of airflow obstruction.
- Only 3 study subjects revealed a history of tobacco smoking and all were of moderate smoking index (100 – 300).
- Exposure to second hand tobacco smoke was found in 44%.
- 59.3% of the study subjects had a biomass exposure index greater than 60.

- Variables significantly associated with degree of airflow obstruction in COPD patients include elderly age, mMRC grade of dyspnea, CAT scoring of symptom severity, exacerbations, biomass exposure index, number of years of biomass exposure, pulmonary hypertension, exercise capacity by 6MWD, body mass index and CT phenotype.

CONCLUSION

1. In this study the prevalence of airflow obstruction in female COPD (as defined by GOLD guidelines) as follows: 0% mild degree, 63.7% moderate, 31.9% severe and 4.4% very severe degree of airflow obstruction.
2. About 3.2% of the female COPD patients were smokers.
3. Two third of the study population had exposure to solid biomass fuel at home with biomass exposure index above 60 as the most common risk factor.
4. One third of the female COPD patients were home makers who were at risk of increased exposure to biomass at home.
5. Two third of the study population had exposure to occupational dusts and chemicals as they were doing occupations like construction work (18.6%), agricultural work (18.6%), sweeping (5.4%), cooking (5.4%) and handling flour (5.4%).
6. Majority of the study subjects were middle aged and belonged to Upper Lower scale of Socio-economic status
7. Most of the study subjects had cough as the commonly reported symptom, with a high CAT score of symptom severity and mMRC grade 3 dyspnea.
8. Only one tenth of the female COPD patients were underweight based on Quetlet index.

9. About half of the female COPD patients had moderate degree of depression.
10. One third of the patients had significant reversibility after a bronchodilator with increased peripheral eosinophil count probably suggesting an asthma overlap.
11. The most common CT phenotype was Airway Predominant Phenotype followed by Mixed Phenotype and Emphysematous Predominant Phenotype being the least common type.
12. Among the socio demographic variables age of the study population was found to have a statistically significant association with the degree of airflow obstruction.
13. Of the clinico-radiological parameters, mMRC grade of dyspnea, CAT scoring of symptom severity, number of exacerbations, exercise capacity by 6MWD, body mass index and CT phenotype were found to have a statistically significant association with the degree of airflow obstruction.
14. Both number of years of biomass exposure and biomass exposure year hours (biomass exposure index) both had a significant association with degree of airflow obstruction with number of years of exposure being more significant than biomass exposure year hours (Biomass exposure index).

15. This study stresses the need for increased awareness in the society especially in the women population in avoiding solid biomass fuel at home and changing to alternate fuels for cooking.
16. Spirometry should be included as primary screening tool in women with history of biomass exposure and respiratory symptoms for early diagnosis and better treatment of their airway disease.

Limitations

1. No controls were included in the study.
2. Most of the risk factors obtained were self reported answers for questionnaires which were recall based
3. The risk of exposure was not quantitatively measured.
4. Clinical parameters such as arterial blood gas analysis, diffusion capacity of lung of carbon monoxide and sputum eosinophil percentage could not be assessed.
5. Institution based study hence does not reflect the true profile of COPD in females at community level.

Recommendations

1. There is a paradigm shift of fuel used for cooking from biomass to LPG in both rural and urban population due to various government schemes like Pradhan Mantri Ujjwala Yojana scheme. Intensified implementation of these schemes by the government can cause a reduction in biomass exposure thereby reducing the prevalence of biomass induced COPD in the female population.
2. Stringent measures like protective gears or mechanisation of certain processes can be strictly enforced in occupations like construction work, agricultural work, sweeping and flour mill workers.

BIBLIOGRAPHY

1. Global Initiative for Chronic Obstructive Lung Disease GLOBAL STRATEGY FOR THE DIAGNOSIS, MANAGEMENT, AND PREVENTION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE UPDATED 2016 Chapter 1 pg no.1
2. WHO The Top 10 causes of death Fact Sheet N^o310 Updated May 2014
3. Rivera RM, Cosío MG, Ghezzi H, et al comparison of lung morphology in COPD secondary to cigarette and biomass smoke. *Int J Tuberc Lung Dis* 2008; 12: 972-977.
4. Chapman K, Tashkin D, Pye D. Gender bias in the diagnosis of COPD. *Chest* 2001;119:1691–1695
5. J. Ancochea, M. Miravittles, F. García-Río, L. Muñoz, G. Sánchez, V. Sobradillo . Infradiagnóstico de la enfermedad pulmonar obstructiva crónica en mujeres: cuantificación del problema, determinantes y propuestas de acción *Arch Bronconeumol*, (2013), <http://dx.doi.org/10.1016/j.arbres.2012.11.010>
6. M Montes de Oca, R J Halbert Chronic bronchitis phenotype in subjects with and without COPD: the PLATINO study *European Respiratory Journal* 2012; DOI:10.1183/09031936.00141611
7. Becklake MR, Kauffmann F. Gender differences in airway behaviour over the human life span. *Thorax* 1999;54:1119–1138
8. Taraseviciene-Stewart L, Douglas IS, Nana-Sinkam PS, Lee JD, Tuder RM, Nicolls MR, Voelkel NF. Is alveolar destruction and emphysema in

chronic obstructive pulmonary disease an immune disease? Proc Am Thorac Soc 2006;3:687–690.

9. Silverman E, Weiss S, Drazen J, Chapman H, Carey V, Campbell E, Denish P, Silverman R, Celedon J, Reilly J, et al. Gender-related differences in severe, early-onset chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2000;162:2152–2158.
10. Patrick DL, Cheadle A, Thompson DC, Diehr P, Koepsell T, Kinne S. The validity of self-reported smoking: a review and meta-analysis. Am J Public Health 1994;84:1086–1093.
11. Barr RG, Wentowski CC, Grodstein F, Somers SC, Stampfer MJ, Schwartz J, Speizer FE, Camargo CA Jr. Prospective study of postmenopausal hormone use and newly diagnosed asthma and chronic obstructive pulmonary disease. Arch Intern Med 2004;164:379–386.
12. Hu G, Zhou Y, Tian J, Yao W, Li J, Li B, et al. Risk of COPD from exposure to biomass smoke: a metaanalysis. Chest.2010;138:20–31.
13. Caballero A, Torres-Duque CA, Jaramillo C, Bolivar F, Sanabria F, Osorio P, Orduz C, Guevara DP, Maldonado D. Prevalence of COPD in five Colombian cities situated at low, medium, and high altitude (PREPOCOL Study). Chest 2008;133:343–349.
14. Behera D, Jindal SK. Respiratory symptoms in Indian women using domestic cooking fuels. Chest. 1991;100:385–8.
15. P.A. Mahesh, B.S. Jayaraj, A.K. Prabhakar,* S.K. Chaya, and R. Vijaysimha* Identification of a threshold for biomass exposure index for

chronic bronchitis in rural women of Mysore district, Karnataka, India
Indian J Med Res. 2013 Jan; 137(1): 87–94.

16. Isabelle Romieu, Horacio Riojas-Rodríguez, Adriana Teresa Marrón-Mares, Astrid Schilmann, Rogelio Perez-Padilla, Omar Masera-Improved biomass stove intervention in rural Mexico: impact on the respiratory health of women; American Journal of Respiratory and Critical Care Medicine 2009 October 1, 180 (7): 649-56
17. Jonathan Grigg "Particulate Matter Exposure in Children", Proceedings of the American Thoracic Society, Vol. 6, No. 7 (2009), pp. 564-569.
18. C LANDBO;"Prognostic Value of Nutritional Status in Chronic Obstructive Pulmonary Disease", American Journal of Respiratory and Critical Care Medicine, Vol. 160, No. 6 (1999), pp. 1856-1861.doi: 10.1164/ajrccm.160.6.9902115
19. Eleni Ischaki, MD; Georgios Papatheodorou, PhD; Eleni Gaki, MD; Ioli Papa, MD; Nikolaos Koulouris, MD, PhD; Stelios Loukides, MD, FCCP; Body Mass and Fat-Free Mass Indices in COPD*:Relation With Variables Expressing Disease Severity COPD | July 2007
20. GATS India Report 2009-2010; International Institute for Population Sciences, Mumbai.
21. Martinez FD What have we learned from the Tucson Children's Respiratory Study?; Paediatr Respir Rev. 2002 Sep;3(3):193-7.
22. Schikowski T, Sugiri D, Ranft U, Gehring U, Heinrich J, Wichmann HE, Kramer U. Long-term air pollution exposure and living close to

- busy roads are associated with COPD in women. *Respir Res* 2005;6:152.
23. Rachel E Jordan¹, Kar Keung Cheng¹, Martin R Miller², Peymané Adab¹ Passive smoking and chronic obstructive pulmonary disease: cross-sectional analysis of data from the Health Survey for England; *BMJ Open* 2011;1:e000153 doi:10.1136/bmjopen-2011-000153
 24. Yin P, Jiang CQ, Cheng KK, Lam TH, Lam KH, Miller MR, Zhang WS, Thomas GN, Adab P. Passive smoking exposure and risk of COPD among adults in China: the Guangzhou Biobank Cohort Study. *Lancet* 2007;370:751–757.
 25. Sezer H, Akkurt I, Guler N, Marakoglu K, Berk S. A case–control study on the effect of exposure to different substances on the development of COPD. *Ann Epidemiol* 2006;16:59–62.
 26. Doney B, Hnizdo E, Syamlal G, et al. Prevalence of Chronic Obstructive Pulmonary Disease Among US Working Adults Aged 40 to 70 Years: National Health Interview Survey Data 2004 to 2011. *Journal of occupational and environmental medicine / American College of Occupational and Environmental Medicine*. 2014;56(10):1088-1093. doi:10.1097/JOM.0000000000000232.
 27. Dr. Brent Doney, PhD, MS, MPH, Dr. Eva Hnizdo, PhD, Dr. Girija Syamlal, MBBS, MPH, Dr. Greg Kullman, PhD, Dr. Cecil Burchfiel, PhD, Dr. Christopher J. Martin, MD, MSc, and Dr. Priscah Mujuru, DrPH Prevalence of Chronic Obstructive Pulmonary Disease Among US

- Working Adults Aged 40 to 70 Years National Health Interview Survey Data 2004 to 2011; *J Occup Environ Med.* 2014 Oct; 56(10): 1088–1093.
28. Ford ES, Croft JB, Mannino DM, Wheaton AG, Zhang X, Giles WH. COPD surveillance—United States, 1999–2012. *Chest.* 2013;144:284–305.
 29. Bakke PS, Hanoa R, Gulsvik A. Educational level and obstructive lung disease given smoking habits and occupational airborne exposure: a Norwegian community study. *Am J Epidemiol* 1995;141:1080-8
 30. Gershon AS¹, Dolmage TE, Stephenson A, Jackson B. Chronic obstructive pulmonary disease and socioeconomic status: a systematic review. *COPD.* 2012 Jun;9(3):216-26. doi: 10.3109/15412555.2011.648030. Epub 2012 Apr 12.
 31. Soriano JB¹, Visick GT, Muellerova H, Payvandi N, Hansell AL. Patterns of comorbidities in newly diagnosed COPD and asthma in primary care. *Chest.* 2005 Oct;128(4):2099-107.
 32. Miriam Barrecheguren, Cristina Esquinas and Marc Miravittles The asthma-COPD overlap syndrome: a new entity? *COPD Research and Practice* 2015;1:8 DOI: 10.1186/s40749-015-0012-z
 33. de Marco R, Accordini S, Marcon A, Cerveri I, Antó JM, Gislason T, *et al.* Risk factors for chronic obstructive pulmonary disease in a European cohort of young adults. *Am J Respir Crit Care Med.* 2011 Apr 1;183(7):891-7.

34. Pat G. Camp, Alejandra Ramirez-Venegas, Raul H. Sansores, Luis F. Alva, Jill E. McDougall, Don D. Sin, Peter D. Paré, Nestor L. Müller, C. Isabela S. Silva, Carlos E. Rojas, Harvey O. Coxson COPD phenotypes in biomass smoke- versus tobacco smoke-exposed Mexican women; *European Respiratory Journal* Mar 2014, 43 (3) 725-734; DOI: 10.1183/09031936.00206112.
35. Paul W Jones, Margaret Tabberer and Wen-Hung Chen Creating scenarios of the impact of copd and their relationship to copd assessment test (CAT™) scores *BMC Pulmonary Medicine* 2011;11:42
36. Hassan Ghobadi, Saeid Sadeghieh Ahari, Azadeh Kameli, and Sharzad M. Lari. The Relationship between COPD Assessment Test (CAT) Scores and Severity of Airflow Obstruction in Stable COPD Patients *Tanaffos*. 2012; 11(2): 22–26.
37. Jones PW, Brusselle G, Dal Negro RW, Ferrer M, Kardos P, Levy ML, et al. Patient-centered assessment of COPD in primary care: Experience from a cross-sectional study of health-related quality of life in Europe. *Prim Care Respir J*. 2012,21(3):329-36
38. Choudhary S, Rini A, Tayade B, Khan S, Doshi V, More V. CAT Score in Chronic Obstructive Pulmonary Disease, Impact on Health: Assessment in Our Region. *PANACEA JOURNAL OF MEDICAL SCIENCES*. 2016 Apr 30;6(1):13-9.
39. Alex J. Mackay, Gavin C. Donaldson, Anant R. C. Patel, Paul W. Jones, John R. Hurst, and Jadwiga A. Wedzicha "Usefulness of the

Chronic Obstructive Pulmonary Disease Assessment Test to Evaluate Severity of COPD Exacerbations", American Journal of Respiratory and Critical Care Medicine, Vol. 185, No. 11 (2012), pp. 1218-1224.

40. Johannes A. Luoto, Sölve Elmståhl, Per Wollmer, Mats Pihlsgår
Incidence of airflow limitation in subjects 65–100 years of age European Respiratory Journal 2015; **DOI:** 10.1183/13993003.00635 2015
41. Om P. Kurmi, Graham S. Devereux, W. Cairns S. Smith, Sean Semple, Markus F.C. Steiner, Padam Simkhada, Kin-Bong Hubert Lam and Jon G. Ayres Reduced lung function due to biomass smoke exposure in young adults in rural Nepal Eur Respir J 2013; 41: 25–30
42. Mitra M, Ghosh S, Saha K, Saha A, Panchadhyayee P, Biswas A, Malik T, Roy A, Barma P. A study of correlation between body mass index and GOLD staging of chronic obstructive pulmonary disease patients. J Assoc Chest Physicians 2013;1:58-61
43. Bhanurekha.B1 , Sasisekhar T.V.D IOSR Correlation of MRC Dyspnoea Scale and Forced Expiratory Volume in First Second (FEV1) In Chronic Obstructive Pulmonary Diseases Journal of Dental and Medical Sciences (IOSR-JDMS) e-ISSN: 2279-0853, p-ISSN: 2279-0861. Volume 7, Issue 2 (May.- Jun. 2013), PP 55-57
44. Agusti A, Caverley PM, Celli B, Coxson HO, Edwards LD, Lomas DA, et al. Characterisation of COPD heterogeneity in the ECLIPSE cohort. Respir Res 2010; 11 : 122-36

45. R. W. Dal Negro, L. Bonadiman, and P. Turco Prevalence of different comorbidities in COPD patients by gender and GOLD stage Multidiscip Respir Med. 2015; 10(1): 24.
46. Boschetto P, Beghe B, Fabbri LM, Ceconi C. Link between chronic obstructive pulmonary disease and coronary artery disease: implication for clinical practice. *Respirology* 2012; 17 : 422-31
47. Yiqing Song, MD, ScD, Anna Klevak, *Diabetes Res Clin Pract.* 2010 Dec; 90(3): 365–371. Asthma, Chronic Obstructive Pulmonary Disease, and Type 2 Diabetes in the Women’s Health Study
48. Paola Rogliani, Gabriella Lucà and Davide Lauro Chronic obstructive pulmonary disease and diabetes *COPD Research and Practice* 2015; 1:3
49. Sin DD, Wu L, Man SF. The relationship between reduced lung function and cardiovascular mortality: a population based study and a systematic review of the literature. *Chest* 2005; 127 : 1952-9. 81.
50. Anthonisen NR, Connett JE, Kiley JP, Altose MD, Bailey WC, Buist AS, et al. Effects of smoking intervention and the use of an inhaled anticholinergic bronchodilator on the rate of decline of FEV1. The Lung Health Study. *JAMA* 1994; 272 : 1497-505. 82.
51. Curkendall SM, DeLuise C, Jones JK, Lanes S, Stang MR, Goehring E Jr, et al. Cardiovascular disease in patients with chronic obstructive pulmonary disease, Saskatchewan Canada cardiovascular disease in COPD patients. *Ann Epidemiol* 2006; 16 : 63-70. 83.

52. de Lucas-Ramos P, Izquierdo-Alonso JL, Moro JMR, Frances JF, Lozano PV, Bellón-Cano JM, CONSISTE study group. Chronic obstructive pulmonary disease as a cardiovascular risk factor: Results of a case-control study (CONSISTE study). *Int J Chron Obstruct Pulmon Dis* 2012; 7 : 679-86
53. J G van Manen,PJE Bindels, F W Dekker, C J IJzermans, J S van der Zee, E SchadéRisk of depression in patients with chronic obstructive pulmonary disease and its determinants *Thorax* 2002;57:412–416
54. Cornela Schneider, MSc; Susan S. Jick, DSc; Ulrich Bothner, MD, MSc; and Christopher R. Meier, PhD, MSc COPD and the Risk of Depression *CHEST*. 2010;137(2):341-347.
55. Global Initiative for Chronic Obstructive Lung isease GLOBAL STRATEGY FOR THE DIAGNOSIS, MANAGEMENT, AND PREVENTION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE UPDATED 2016 Chapter 2,pg no.10
56. The Global Strategy for the Diagnosis, Management and Prevention Of COPD, Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2016. Availablefrom: <http://www.goldcopd.org/>
57. Launois, Claire, et al. "The modified Medical Research Council scale for the assessment of dyspnea in daily living in obesity: a pilot study." *BMC pulmonary medicine*12.1 (2012): 61.

57. Jones, P. W., et al. "Properties of the COPD assessment test in a cross-sectional European study." *European Respiratory Journal* 38.1 (2011): 29-35
58. Fletcher, Charles M., et al. "Significance of respiratory symptoms and the diagnosis of chronic bronchitis in a working population." *British Medical Journal* 2.5147 (1959): 257.
59. Mahler, D. A., and Carolyn K. Wells. "Evaluation of clinical methods for rating dyspnea." *CHEST Journal* 93.3(1988): 580-586.
60. Dyspnea, A. T. S. "Mechanisms, assessment, and management: a consensus statement. American Thoracic Society." *Am J Respir Crit Care Med* 159.1 (1999): 321-40.
61. Kuppuswamy B. *Manual of Socioeconomic Status (urban)*, Manasayan, Delhi, 1981.
62. Sukhvinder Singh Oberoi Updating income ranges for Kuppuswamy's socio-economic status scale for the year 2014 *Indian journal of public health* Year : 2015 | Volume : 59 | Issue : 2 | Page : 156-157
63. Jindal SK, Malik SK. Smoking index-a measure to quantify cumulative smoking exposure. *Lung India*. 1988;6:195-6.
64. Hagstad S, Bjerg A, Ekerljung L, Backman H, Lindberg A, Rönmark E, *et al*. Passive smoking exposure is associated with increased risk of COPD in never smokers. *Chest*. 2014 Jun;145(6):1298-304.

65. Seif O. Shaheen, David J. P. Barker, and Stephen T. Holgate "Do Lower Respiratory Tract Infections in Early Childhood Cause Chronic Obstructive Pulmonary Disease?" *American Journal of Respiratory and Critical Care Medicine*, Vol. 151, Supplement: Infections, Asthma, and COPD (1995), pp. 1649-1652.
66. Cassino C, Berger KI, Goldring RM, Norman RG, Kammerman S, Ciotoli C, Reibman J. Duration of asthma and physiologic outcomes in elderly nonsmokers. *Am J Respir Crit Care Med* 2000;162:1423–1428.
67. Connolly CK, Chan NS, Prescott RJ. The relationship between age and duration of asthma and the presence of persistent obstruction in asthma. *Postgrad Med J* 1988;64:422–425.
68. Lee SJ, Kim SW, Kong KA, Ryu YJ, Lee JH, Chang JH. Risk factors for chronic obstructive pulmonary disease among never-smokers in Korea. *Int J Chron Obstruct Pulmon Dis*. 2015 Mar 5;10:497-506.
69. John Dement, PhD, Laura Welch, MD, Knut Ringen, Dr. PH, A Case-Control Study of Airways Obstruction Among Construction Workers
AMERICAN JOURNAL OF INDUSTRIAL MEDICINE 9999:1–15
(2015)
70. Ye M, Beach J, Martin JW, Senthilselvan A. 2013. Occupational pesticide exposures and respiratory health. *Int J Environ Res Public Health* 10:6442–6471.

- 71 Hamdy A. Mohammadien , Mona T. Hussein Effects of exposure to flour dust on respiratory symptoms and pulmonary function of mill workers · Egyptian Journal of Chest Diseases and Tuberculosis Volume 62, Issue 4, October 2013, Pages 745–753
- 72 Anwar SK, Mehmood N, Nasim N, Khurshid M, Khurshid B. Sweeper's lung disease: a cross-sectional study of an overlooked illness among sweepers of Pakistan. *Int J Chron Obstruct Pulmon Dis.* 2013;8:193-7. doi: 10.2147/COPD.S40468. Epub 2013 Apr
- 73 Kroenke K, Spitzer RL, Williams JBW. The PHQ-9: Validity of a brief depression severity measure. *J GenIntern Med.* 2001;16:606-613.
74. Spitzer, Robert L., et al. "Validity and utility of the PRIME-MD patient health questionnaire in assessment of 3000 obstetric-gynecologic patients: the PRIME-MD Patient Health Questionnaire Obstetrics Gynecology Study." *American journal of obstetrics and gynecology* 183.3 (2000): 759-769
75. Kroenke, Kurt, and Robert L. Spitzer. "The PHQ-9: a new depression diagnostic and severity measure." *Psychiatr Ann* 32.9 (2002): 1-7w

76. Vestbo, Jørgen, et al. "Body mass, fat-free body mass, and prognosis in patients with chronic obstructive pulmonary disease from a random population sample: findings from the Copenhagen City Heart Study." *American journal of respiratory and critical care medicine* 173.1 (2006): 79-83.
78. Durnin, J. V. G. A., and M. M. Rahaman. "The assessment of the amount of fat in the human body from measurements of skin folds thickness." *British Journal of Nutrition* 21.03 (1967): 681-689.
79. Miller, Martin Raymond, et al. "General considerations for lung function testing." *European Respiratory Journal* 26.1 (2005): 153-161.
80. ATS statement: guidelines for the six-minute walk test. ATS Committee on Proficiency Standards for Clinical Pulmonary Function Laboratories. *Am J Respir Crit Care Med* 2002;166(1): 111–117
81. Srikanth, Avinash M., et al. "Reference equations for the six-minute walk distance in the Indian population." *CHEST Journal*
82. Madani A, Zanen J, de Maertelaer V, Gevenois PA. Pulmonary emphysema: objective quantification at multi-detector row CT—comparison with macroscopic and microscopic morphometry. *Radiology* 2006;238(3):1036–1043

83. Lee YK, Oh YM, Lee JH, et al.. Quantitative assessment of emphysema, air trapping, and airway thickening on computed tomography. *Lung* 2008;186(3):157–165
84. Goddard PR, Nicholson EM, Laszlo G, Watt I. Computed tomography in pulmonary emphysema. *Clin Radiol* 1982;33(4):379–387.
85. Raheison C, Tillie-Leblond I, Prudhomme A, Taillé C, Biron E, Nocent-Ejnaini C, et al. Clinical characteristics and quality of life in women with COPD: an observational study. *BMC Womens Health*. 2014 Feb 20;14(1):31.
86. Frei A, Muggensturm P, Putcha N, Siebeling L, Zoller M, Boyd CM, et al. Five comorbidities reflected the health status in patients with chronic obstructive pulmonary disease: the newly developed COMCOLD index. *J Clin Epidemiol*. 2014 Aug;67(8):904-11.
87. Menezes AMB, de Oca MM, Perez-Padilla R, Nadeau G, Wehrmeister FC, Lopez-Varela MV. Increased risk of exacerbation and hospitalization in subjects with an overlap phenotype. *Chest*.2014;145:297–304.
88. Jain NK, Thakkar MS, Jain N, Rohan KA, Sharma M. Chronic obstructive pulmonary disease: Does gender really matter? *Lung India*. 2011 Oct;28(4):258-62.

89. Sharifi H, Masjedi MR, Emami H, Ghanei M, Eslaminejad A, Radmand G, *et al.* Burden of obstructive lung disease study in Tehran: Prevalence and risk factors of chronic obstructive pulmonary disease. *Lung India.* 2015 Nov-Dec;32(6):572-7.
90. Jordan RE, Cheng KK, Miller MR, Adab P. Passive smoking and chronic obstructive pulmonary disease: cross-sectional analysis of data from the Health Survey for England. *BMJ Open.* 2011 Jan 1;1(2):e000153.
91. Hajiro T, Nishimura K, Tsukino M, Ikeda A, Koyama H, Izumi T. Analysis of clinical methods used to evaluate dyspnea in patients with chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 1998;158:1185-9.
92. Sahebjami H, Sathianpitayakul E. Influence of body weight on the severity of dyspnea in chronic obstructive pulmonary disease. *Am J Respir Crit Care Med.* 2000 Mar;161(3 Pt 1):886-90.
93. Mahler DA, Weinberg DH, Wells CK, Feinstein AR. The measurement of dyspnea. Contents, interobserver agreement, and physiologic correlates of two new clinical indexes. *Chest* 1984;85:751-8.

94. Okutan O, Tas D, Demirer E, Kartaloglu Z. Evaluation of quality of life with the chronic obstructive pulmonary disease assessment test in chronic obstructive pulmonary disease and the effect of dyspnea on disease-specific quality of life in these patients. *Yonsei Med J.* 2013 Sep;54(5):1214-9.
95. Chai JJ, Liu T, Cai BQ. Evaluation of clinical significance of chronic obstructive pulmonary disease assessment test. *Zhonghua Jie He He Hu Xi Za Zhi.* 2011 Apr;34(4):256-8.
96. Miyazaki M, Nakamura H, Chubachi S, Sasaki M, Haraguchi M, Yoshida S, *et al.* Analysis of comorbid factors that increase the COPD assessment test scores. *Respir Res.* 2014 Feb 6;15:13.
97. Dal Negro RW, Bonadiman L, Turco P. Sensitivity of the COPD assessment test (CAT questionnaire) investigated in a population of 681 consecutive patients referring to a lung clinic: the first Italian specific study. *Multidiscip Respir Med.* 2014 Mar 15;9(1):15.
98. Aditi R Mishra, Rajesh Swarnakar. Clinical correlation of chronic obstructive pulmonary disease assessment test (CAT) score with pulmonary function test (PFT) of patients in a health care setup in rural India to assess level of control of symptoms and treatment as per global initiative for obstructive lung disease (GOLD) guidelines. *American Journal of Respiratory and Critical Care Medicine* 2012;185:1512.

99. Verhage TL, Heijdra Y, Molema J, Vercoulen J, Dekhuijzen R. Associations of muscle depletion with health status. Another gender difference in COPD? *Clin Nutr.* 2011 Jun;30(3):332-8.
100. Pothirat C, Chaiwong W, Phetsuk N, Liwsrisakun C, Bumroongkit C, Deesomchok A, *et al.* The Relationship between Body Composition and Clinical Parameters in Chronic Obstructive Pulmonary Disease. *J Med Assoc Thai.* 2016 Apr;99(4):386-93.
101. WHO Factsheet. Household air pollution and health. Feb 2016. Available from URL: <http://who.int/mediacentre/factsheets/fs292/en/> [Accessed on 09/08/2016].
102. Ramírez-Venegas A, Sansores RH, Pérez-Padilla R, Regalado J, Velázquez A, Sánchez C, *et al.* Survival of Patients with Chronic Obstructive Pulmonary Disease Due to Biomass Smoke and Tobacco. *Am J Respir Crit Care Med* 2006;173:393-7.
103. Wu CF, Feng NH, Chong IW, *et al.* Second-hand smoke and chronic bronchitis in Taiwanese women: a health-care based study. *BMC Public Health.* 2010;10:44.
104. Mark D. Eisner, Nicholas Anthonisen, David Coultas, Nino Kuenzli, Rogelio Perez-Padilla, Dirkje Postma, Isabelle Romieu, Edwin K. Silverman, and John R. Balmes, on behalf of the Environmental and Occupational Health Assembly Committee on Nonsmoking COPD An Official American Thoracic Society Public Policy Statement: Novel Risk Factors and the Global Burden of Chronic Obstructive

Pulmonary Disease APPROVED BY THE ATS BOARD OF
DIRECTORS, MARCH 2010

105. Bunyamin Sertogullarindan, Hasan Ali Gumrukcuoglu, Cengizhan Sezgi, Mehmet Ata Akil Frequency of Pulmonary Hypertension in Patients with COPD due to Biomass Smoke and Tobacco Smoke 2012; 9(6):406-412. doi: 10.7150/ijms.4715.

நான் ஒருபோதும் இருமுன்தில்லை	0 1 2 3 4 5	நான் எப்பொழுதும் இருமுகிறேன்	
என் மார்பில் சளி (கபம்) சிறிதும் இல்லை	0 1 2 3 4 5	என் மார்பு முழுமையாக சளி (கபம்) நிறைந்திருக்கிறது	
என் மார்பு இறுக்கமாய் உணர்வதே இல்லை	0 1 2 3 4 5	என் மார்பு மிகவும் இறுக்கமாய் உணர்கிறது	
நான் ஒரு குன்றின் மீது அல்லது மாடிப்படிக்களில் நடந்து ஏறும்பொழுது எனக்கு மூச்சுத்திணறல் இல்லை	0 1 2 3 4 5	நான் ஒரு குன்றின் மீது அல்லது மாடிப்படிக்களில் நடந்து ஏறும்பொழுது எனக்கு மிகவும் மூச்சுத்திணறல் இருக்கிறது	
வீட்டில் நான் எந்த நடவடிக்கைகள் செய்வதிலும் மட்டுப்படுத்தப்படவில்லை	0 1 2 3 4 5	வீட்டில் நான் நடவடிக்கைகள் செய்வதில் மிகவும் மட்டுப்படுத்தப்படுகிறேன்	
என்னுடைய நுரையீரல் பிரச்சினை இருக்கிற போதிலும்கூட நான் என் வீட்டிலிருந்து வெளியே செல்வதில் தன்னம்பிக்கையுடன் இருக்கிறேன்	0 1 2 3 4 5	என்னுடைய நுரையீரல் பிரச்சினை காரணமாக நான் என் வீட்டிலிருந்து வெளியே செல்வதில் சிறிதும் தன்னம்பிக்கை இல்லாதிருக்கிறேன்	
நான் ஆழமாக உறங்குகிறேன்	0 1 2 3 4 5	என்னுடைய நுரையீரல் பிரச்சினை காரணமாக நான் ஆழமாக உறங்குவதில்லை	
நான் ஏராளமான சக்தி படைத்துள்ளேன்	0 1 2 3 4 5	எனக்குச் சிறிதும் சக்தி இல்லை	

Click here to get your total score

NAME: _____

DATE: _____

Over the last 2 weeks, how often have you been bothered by any of the following problems?

(use "✓" to indicate your answer)

	Not at all	Several days	More than half the days	Nearly every day
1. Little interest or pleasure in doing things	0	1	2	3
2. Feeling down, depressed, or hopeless	0	1	2	3
3. Trouble falling or staying asleep, or sleeping too much	0	1	2	3
4. Feeling tired or having little energy	0	1	2	3
5. Poor appetite or overeating	0	1	2	3
6. Feeling bad about yourself—or that you are a failure or have let yourself or your family down	0	1	2	3
7. Trouble concentrating on things, such as reading the newspaper or watching television	0	1	2	3
8. Moving or speaking so slowly that other people could have noticed. Or the opposite — being so fidgety or restless that you have been moving around a lot more than usual	0	1	2	3
9. Thoughts that you would be better off dead, or of hurting yourself	0	1	2	3

add columns + +

(Healthcare professional: For interpretation of TOTAL, TOTAL:
 please refer to accompanying scoring card).

Originality GradeMark PeerMark

TO STUDY THE PREVALENCE,

BY 201627001 MD TB RAJESWARI.P



8% SIMILAR

-- OUT OF 0

“TO STUDY THE PREVALENCE, SOCIODEMOGRAPHIC, CLINICO-RADIOLOGICAL PROFILE AND ALSO CORRELATION OF COPD ASSESSMENT TEST (CAT) AND DEGREE OF AIRFLOW OBSTRUCTION IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE IN FEMALES ATTENDING TERTIARY CARE HOSPITAL”

22 Dissertation submitted to The Tamil Nadu Dr.M.G.R. Medical University in partial fulfilment of the requirements for the degree of

11 Doctor of Medicine (M.D) in Tuberculosis and Respiratory Diseases Branch - XVII

Institute of Thoracic Medicine, Madras Medical College & Rajiv Gandhi Government General Hospital



Match Overview

1	erj.ersjournals.com Internet source	1%
2	www.ijmr.org.in Internet source	<1%
3	www.goldcopd.it Internet source	<1%
4	www.goldcopd.org Internet source	<1%
5	www.thoracic.org Internet source	<1%
6	www.dovepress.com Internet source	<1%
7	"Abstracts of the 50th E... Publication	<1%
8	tunisie.wv.stmra.com Internet source	<1%
9	www.eomsociety.org	<1%



Digital Receipt

This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

The first page of your submissions is displayed below.

Submission author: 201527001 Md T b RAJESWARIP
Assignment title: 2015-2015 plagiarism
Submission title: TO STUDY THE PREVALENCE, SO..
File name: rajl_maam_plag_check_1.docx
File size: 1.46M
Page count: 82
Word count: 14,721
Character count: 80,495
Submission date: 01-Oct-2016 09:43AM
Submission ID: 713781559

"TO STUDY THE PREVALENCE, SOCIODEMOGRAPHIC, CLINICO-PATHOLOGICAL PROFILE AND ALSO CORRELATION OF COPD ASSESSMENT TEST (CAT), AND DEGREE OF AIRFLOW OBSTRUCTION IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE IN FEMALES ATTENDING TERTIARY CARE HOSPITAL"

Dissertation submitted to The Tamil Nadu Dr.M.G.R. Medical University
in partial fulfillment of the requirements for the degree of

Doctor of Medicine (MD) in
Tuberculosis and Respiratory Diseases
Branch - VIII

Institute of Thoracic Medicine,
Madhav Medical College &
Rajiv Gandhi Government General Hospital



The Tamil Nadu Dr. M.G.R. Medical University
Chennai - 600029
Tamil Nadu
India
April 2017

**INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003**

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr.P.Rajeswari
P.G. in MD (TB & Chest Diseases)
Madras Medical College/RGGGH
Chennai 600 003

Dear Dr.P.Rajeswari,

The Institutional Ethics Committee has considered your request and approved your study titled "**TO STUDY THE PREVALENCE SOCIODEMOGRAPHIC, CLINICO-RADIOLOGICAL PROFILE AND ALSO CORRELATION OF COPD ASSESSMENT TEST (CAT) AND DEGREE OF AIRFLOW OBSTRUCTION IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE IN FEMALES ATTENDING TERTIARY CARE HOSPITAL**" - NO.25012016.

The following members of Ethics Committee were present in the meeting hold on **05.01.2016** conducted at Madras Medical College, Chennai 3

1.Dr.C.Rajendran, MD.,	:Chairperson
2.Dr.R.Vimala,MD.,Dean,MMC,Ch-3	:Deputy Chairperson
3.Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	: Member Secretary
4.Prof.B.Vasanthi,MD.,Inst.of Pharmacology,MMC,Ch-3	: Member
5.Prof.Md.Ali,MD.,DM.,HOD-MGE, MMC,Ch-3	: Member
6.Prof.K.Ramadevi,MD, Director, Inst. of Bio-Chem,MMC,Ch-3:	Member
7.Prof.M.Saraswathi, MD.,Director, Inst.of Path,MMC,Ch-3	: Member
8.Prof.Srinivasagalu,MD.Director,Inst.of Int.Med.MMC,Ch-3	:Member
9.Tmt.J.Rajalakshmi, JAO,MMC,Ch-3	: Lay Person
10.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
11.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.



Member Secretary - Ethics Committee
MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

PATIENT INFORMATION SHEET

TITLE OF THE STUDY: **“To study the prevalence, sociodemographic, clinico-radiological profile and also correlation of COPD assessment test (CAT) and degree of airflow obstruction in Chronic Obstructive Pulmonary Disease in females”**

We are conducting a study among patients attending thoracic medicine department in Rajiv Gandhi Government General Hospital and Institute of Thoracic Medicine, Chennai.

The purpose of this study is to analyse the **prevalence, sociodemographic, clinico-radiological profile and also correlation of COPD assessment test (CAT) and degree of airflow obstruction in Chronic Obstructive Pulmonary Disease in females”**

We are selecting female patients aged 40 yrs and above presenting with symptoms of cough, sputum, breathlessness, wheeze for more than 2 weeks with spirometry post bronchodilator FEV1/FVC < 0.70. Patients will undergo basic blood investigations, chest X-ray / CT chest (if needed), DLCO to arrive at a diagnosis and subsequently treat the patient.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Investigator
Date :

Signature of Participant

ஆராய்ச்சி தகவல் தாள்

ஆராய்ச்சி தலைப்பு

ராஜீவ் காந்தி அரசு பொது மருத்துவமனையின் நெஞ்சக மருத்துவத்துறைக்கு வரும் பெண் நோயாளிகளில், நாள்பட்ட மூச்சுக்குழாய் அடைப்பு நோயின் சமூகம் சார்ந்த புள்ளிவிவரம், நோயின் தன்மை மற்றும் கதிர்வீச்சு பண்புகள் ஆகியவற்றை பற்றியும் நாள்பட்ட மூச்சுக்குழாய் அடைப்பு மதிப்பீடு கேள்வித்தாளாக்கும் (CAT), மூச்சுக்குழாய் அடைப்பின் அளவிற்கும் உள்ள தொடர்பையும் கண்டறியும் ஆய்வு.

ஆராய்ச்சியாளர் பெயர் : மருத்துவர்.ப.ராஜேஸ்வரி

பங்கேற்பாளர் பெயர் :

ஆராய்ச்சியின் நோக்கம்

ராஜீவ் காந்தி அரசு பொது மருத்துவமனையின் நெஞ்சக மருத்துவத்துறைக்கு வரும் பெண் நோயாளிகளில், நாள்பட்ட மூச்சுக்குழாய் அடைப்பு நோயின் சமூகம் சார்ந்த புள்ளிவிவரம், நோயின் தன்மை மற்றும் கதிர்வீச்சு பண்புகள் ஆகியவற்றை பற்றியும் நாள்பட்ட மூச்சுக்குழாய் அடைப்பு மதிப்பீடு கேள்வித்தாளாக்கும் (CAT), மூச்சுக்குழாய் அடைப்பின் அளவிற்கும் உள்ள தொடர்பையும் கண்டறிதல்.

இதன் மூலம் பெண் நோயாளிகளின் மூச்சிரைக்கு நாள்பட்ட மூச்சுக்குழாய் அடைப்பு நோயின் பங்களிப்பையும், அதன் தன்மையையும் அறிந்து அதற்கு ஏற்ற சிகிச்சை அளிக்கப்படும்.

ஆய்வு முறை

ராஜீவ் காந்தி அரசு பொது மருத்துவமனையின் நெஞ்சக மருத்துவத்துறைக்கு வரும் 40 வயதிற்கு மேற்பட்ட பெண்களுக்கு இருமல், சளி மற்றும் மூச்சிரைப்பு இரண்டு வாரங்களுக்கு மேல் இருந்தால் அவர்களுக்கு மூச்சுக்குழாய் அடைப்பு நோய் மதிப்பீடு கேள்வித்தாள் கொடுக்கப்பட்டு நுரையீரல் செயல்திறன் பரிசோதனையும் செய்யப்படும். மூச்சுக்குழாய் விரிவடையச் செய்யும் மருந்து கொடுத்து மீண்டும் நுரையீரல் செயல்திறன் பரிசோதனை செய்யப்படும்.

அதில் FEV1/ FVC மதிப்பு ≤ 0.70 இருந்தால் இந்த ஆய்வில் சேர்த்துக்கொள்ளப்படுவர். அவர்களுக்கு நோய் சம்பந்தமான வரலாறு மற்றும் நோயின் தன்மை பற்றி முற்றிலுமாக கேட்டு அறியப்படும். அவர்களுக்கு நுரையீரல் மற்றும் இதர உறுப்புகள் சம்பந்தப்பட்ட மருத்துவ பரிசோதனை செய்யப்படும். சளி, பரிசோதனை, இரத்தப் பரிசோதனை, நெஞ்சுப்படம் (எக்ஸ்ரே) தேவைப்பட்டால் சி.டி.ஸ்கேன் மற்றும் DLCO பரிசோதனைகள் செய்யப்படும்.

நன்மைகள்

இந்த ஆய்வு மூலம் பெண்களுக்கு நாள்பட்ட மூச்சுக்குழாய் அடைப்பு நோயினால் ஏற்படும் பாதிப்பு மற்றும் அந்நோயின் அபாய காரணிகள் எவை என கண்டறியப்பட்டு அந்நோய் தக்க சிகிச்சை அளிக்க முடியும்.

இந்த நோய் பற்றிய முறையான ஆய்வு ஏற்கனவே பல இடங்களில் நடத்தப்பட்டுள்ளது. மேலும் இதன் பாதுகாப்பு உறுதிசெய்யப்பட்டுள்ளது. உங்கள் பாதுகாப்பே எங்களின் முக்கிய நோக்கம்.

இந்த ஆய்வு சம்பந்தமான எல்லா புள்ளி விவரங்கள் மற்றும் நோயாளிகளின் விவரங்கள் ரகசியமாக வைக்கப்படும். இந்த ஆய்வு சம்பந்தப்பட்ட எல்லா பரிசோதனைகள், மருந்துகள் மற்றும் மருத்துவ சேவைகள் அனைத்தும் நோயாளிகளுக்கு இலவசமாக வழங்கப்படும்.

ஆய்வாளரின் பெயர்

பங்குபெறுபவரின் பெயர்

ஆய்வாளரின் கையொப்பம்

பங்குபெறுபவரின் கையொப்பம்

PATIENT CONSENT FORM

Study detail: "To study the prevalence, sociodemographic, clinico-radiological profile and also correlation of COPD assessment test (CAT) and degree of airflow obstruction in Chronic Obstructive Pulmonary Disease in females attending tertiary care

Study centre: Rajiv Gandhi Government General Hospital, Institute Of Thoracic Medicine, Chennai.

Patient's name:

Patient's age:

ID No:

Patient may check (√) these boxes

a) I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction.

b) I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected

c) I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study.

d) I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms.

e) I hereby consent to participate in this study.

f) I hereby give permission to undergo detailed clinical examination, Radiographs ,blood investigations and surgical procedure as required.

Signature/thumb impression

Signature of Investigator

Patient's Name and Address:

Study Investigator's Name:

Dr.Rajeswari.P

ஆராய்ச்சி ஒய்யுதல் படவம்

ஆராய்ச்சியின் தலைப்பு

ராஜீவ் காந்தி அரசு பொது மருத்துவமனையின் நெஞ்சு மருத்துவத்துறைக்கு வரும் பெண் நோயாளிகளில், நாள்பட்ட மூச்சுக்குழாய் அடைப்பு நோயின் சமூகம் சார்ந்த புள்ளிவிவரம், நோயின் தன்மை மற்றும் கதிர்வீச்சு பண்புகள் ஆகியவற்றை பற்றியும் நாள்பட்ட மூச்சுக்குழாய் அடைப்பு மதிப்பீடு கேள்வித்தாளுக்கும் (CAT), மூச்சுக்குழாய் அடைப்பின் அளவிற்கும் உள்ள தொடர்பையும் கண்டறியும் ஆய்வு.

ஆய்வு நிலையம் : நெஞ்சு நோய் மருத்துவத் துறை,
சென்னை மருத்துவக் கல்லூரி சென்னை - 3.

பங்கு பெறுவரின் பெயர் :

பங்குபெறுபவரின் எண் :

பங்குபெறுபவர் இதனை (✓) குறிக்கவும்

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது.

நான் இவ்வாய்வில் தன்னிச்சையாகதான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.

இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக்கொள்ளவும் அதை பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கின்றேன்.

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வதுடன் 'இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம் இடம்..... தேதி.....

கட்டைவிரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம்

ஆய்வாளரின் கையொப்பம் இடம்..... தேதி.....

ஆய்வாளரின் பெயர்

EVALUATION FORM

Name :

Age :

Sex :

IP /OP number :

Sociodemographic history :

Presenting complaints :

History of presenting illness :

Past history :

Personal history :

Occupational history :

CAT questionnaire scoring :

Modified Medical Council grades(mMRC) :

Patient health questionnaire – 9 score :

Nutritional status : Body Mass Index :

 Fat free mass :

General examination :

Systemic examination :

Blood investigations :

Radiological findings :

Chest xray :

CT-Chest :

Sputum investigations :

Spirometric analysis :

DLCO :

Final diagnosis

#	Name	IP/OP number	Centre	Age	Age	Symptoms	symptom analysis	MMRC grading	CAT score	CAT score grading	FEV1 score	FEV1 range	No. of exacerbations	No. of hospitalisations	Combined assessment	Height (metres)	Weight (kgs)	BMI score	BMI range	6MWD Expected value	6MWD observed value	6MWD %	6MWD % range	Skin fold Thickness	Density	% Body fat	Fat free mass	biomass years	Biomass exposure (hours)	biomass exposure
1	manjula	30372	A	65	2	cs	1	2	19	3	69	2	0	0	B	1.41	48	24.14	2	274.1	280	102.2	4	85	1.009	40.36	28.62	33	99	1
2	kalyani	29550	A	50	1	cs	1	2	15	3	68	2	0	0	B	1.54	46	19.40	2	291.7	230	78.8	4	58	1.020	35.22	29.80	0	0	2
3	salomi	123	A	58	1	cs	1	3	33	5	35	3	3	0	D	1.61	50	19.29	2	289.3	220	76.0	3	61	1.019	35.89	32.05	35	105	1
4	rajam	2528	A	48	1	bw	2	3	20	3	44	2	2	1	B	1.45	41	19.50	2	282.8	180	63.7	3	47	1.031	30.13	28.65	30	45	1
5	visalakshi	983	A	43	1	cs	1	2	25	4	64	3	4	1	D	1.51	45	19.74	2	299.8	310	103.4	4	51	1.029	31.14	30.98	10	20	1
6	parameswari	2748	A	44	1	cs	1	2	29	4	73	2	0	0	B	1.4	55	28.06	3	322.4	300	93.1	4	78	1.018	36.48	34.93	10	15	1
7	vasanthakumari	30403	A	50	1	cs	1	3	24	4	54	2	1	0	B	1.54	60	25.30	3	325.3	320	98.4	4	75	1.013	38.67	36.80	31	62	1
8	lourdhamary	4621	B	72	4	bw	2	4	31	5	28	4	3	1	D	1.49	48	21.62	2	263.6	150	56.9	3	64	1.017	36.53	30.46	40	100	1
9	saraswathi	4466	B	68	3	bw	2	4	29	4	31	3	0	0	D	1.5	42	18.67	2	255.2	130	51.0	3	48	1.025	32.71	28.26	20	60	1
10	rani	5103	B	45	1	b	7	2	31	5	39	3	2	0	D	1.55	55	22.89	2	320.8	210	65.5	3	57	1.026	32.53	37.11	0	0	2
11	shantha	4012	B	44	1	bw	2	3	31	5	45	3	0	0	D	1.54	52	21.93	2	315.1	275	87.3	4	59	1.025	32.96	34.86	20	60	1
12	vallyammal	803	B	47	1	csb	3	3	29	4	53	2	1	0	B	1.52	48	20.78	2	301.0	240	79.7	4	55	1.027	32.09	32.60	30	45	1
13	mangatha	1989	B	73	4	b	7	3	33	5	31	3	2	0	D	1.53	40	17.09	1	242.8	110	45.3	2	45	1.027	31.86	27.26	50	100	1
14	dinakasundari	7989	B	65	3	csb	3	3	25	4	39	3	1	0	D	1.45	30	14.27	1	230.9	180	78.0	4	38	1.032	29.65	21.11	40	100	1
15	suguna	28600	A	55	2	csb	3	3	17	3	59	2	0	1	B	1.41	59	29.68	3	315.5	260	82.4	4	98	1.005	42.31	34.04	46	161	1
16	grasmary	32697	A	69	3	csb	3	2	32	5	49	3	1	0	D	1.64	58	21.56	2	292.0	190	65.1	3	47	1.026	32.43	39.19	40	80	1
17	kamatchi	32496	A	57	2	csb	3	2	15	3	61	2	0	0	B	1.28	41	25.02	3	269.3	230	85.4	4	72	1.014	38.12	25.37	13	39	1
18	mariammal	21349	A	45	1	c	6	2	23	4	55	2	0	0	B	1.32	41	23.53	2	287.3	290	100.9	4	75	1.019	35.99	26.25	20	60	1
19	latha	32499	A	42	1	csb	3	1	15	3	66	2	0	0	B	1.62	65	24.77	2	349.3	270	77.3	4	78	1.018	36.48	41.28	0	0	2
20	mohanasundari	2535	A	52	2	cs	1	2	21	4	59	2	0	0	B	1.39	44	22.77	2	284.0	260	91.6	4	46	1.027	32.15	29.85	26	65	1
21	jayanthi	3341	A	48	1	cs	1	3	25	4	52	2	2	0	B	1.48	65	29.67	3	340.4	310	91.1	4	54	1.027	31.86	44.29	30	75	1
22	shanthi	30793	A	51	1	bw	2	2	29	4	57	2	0	0	B	1.49	68	30.63	4	343.1	250	72.9	2	93	1.007	41.59	39.72	33	66	1
23	amul	32716	A	49	1	csbw	4	1	20	3	62	2	1	0	B	1.62	80	30.48	4	374.8	320	85.4	4	110	1.008	40.89	47.29	0	0	2
24	ponnamma	2771	A	55	2	csb	3	3	21	4	58	2	0	0	B	1.56	51	20.96	2	296.2	220	74.3	3	75	1.013	38.67	31.28	33	66	1
25	chinnakannu	1042	A	67	3	cs	1	4	30	5	40	3	0	0	D	1.54	60	25.30	3	299.8	130	43.4	2	57	1.021	34.99	39.01	45	135	1
26	padma	587	A	58	2	cs	1	0	7	2	79	2	0	0	A	1.56	60	24.65	2	313.3	300	95.7	4	63	1.018	36.32	38.21	30	60	1
27	reetha	31656	A	48	1	cs	1	1	9	2	68	2	0	0	A	1.48	49	22.37	2	302.0	280	92.7	4	47	1.031	30.13	34.24	0	0	2
28	jaya	1182	A	47	1	bw	2	2	18	3	59	2	1	1	B	1.56	65	26.71	3	341.8	260	76.1	4	84	1.016	37.43	40.67	26	65	1
29	ramasubbulakshn	31273	A	51	2	cs	1	2	29	4	35	3	2	0	D	1.45	48	22.83	2	295.1	200	67.8	3	58	1.020	35.22	31.10	35	70	1
30	kasturi		B	54	2	bw	2	2	13	3	77	2	0	0	B	1.41	43	21.63	2	278.6	240	86.2	4	49	1.025	32.98	28.82	16	40	1
31	bakkamma		B	62	3	cs	1	3	31	5	29	4	1	0	B	1.45	35	16.65	1	247.4	110	44.5	2	39	1.031	29.99	24.50	23	68	1
32	egavalli		B	68	3	cs	1	3	28	4	46	3	3	0	D	1.45	51	24.26	2	276.8	100	36.1	2	72	1.014	38.12	31.56	40	60	1
33	jaya		B	60	2	bw	2	2	20	4	53	2	1	0	B	1.53	59	25.20	3	307.9	250	81.2	4	68	1.016	37.35	36.97	30	60	1
34	karpagam		B	58	2	cs	1	2	22	4	54	2	0	0	B	1.37	38	20.25	2	260.6	220	84.4	4	49	1.025	32.98	25.47	35	70	1
35	mohanasundari	3983	A	62	3	cs	1	3	12	3	65	2	1	0	B	1.35	37	20.30	2	252.2	140	55.5	3	51	1.024	33.51	24.60	17	34	1
36	gowri	31599	A	50	1	cs	1	2	21	4	54	2	2	0	D	1.38	47	24.68	2	294.2	270	91.8	4	73	1.014	38.30	29.00	25	65	1
37	kumudha	2694	A	49	1	csb	3	1	15	3	76	2	0	0	A	1.43	48	23.47	2	298.1	280	93.9	4	71	1.020	35.29	31.06	0	0	2
38	saroja	19766	A	60	2	csb	3	3	31	5	29	4	4	0	D	1.42	43	21.33	2	269.6	180	66.8	3	62	1.018	36.11	27.47	36	108	1
39	shanthi		B	49	1	bw	2	3	26	4	33	3	1	0	D	1.55	50	20.81	2	302.8	140	46.2	2	62	1.024	33.58	33.21	0	0	2
40	kanagammal	12387		54	2	bw	2	3	20	3	57	2	0	0	B	1.39	42	21.74	2	276.2	280	101.4	4	50	1.024	33.25	28.04	20	50	1
41	tamilselvi	764		69	3	csb	3	3	32	5	46	2	3	1	D	1.51	46	20.17	2	263.2	280	106.4	4	49	1.025	32.98	30.83	35	70	1

#	Name	IP/OP number	Centre	Age	Age	Symptoms	symptom analysis	MMRC grading	CAT score	CAT score grading	FEV1 score	FEV1 range	No. of exacerbations	No. of hospitalisations	Combined assessment	Height (metres)	Weight (kgs)	BMI score	BMI range	6MWD Expected value	6MWD observed value	6MWD %	6MWD % range	Skin fold thickness	Density	% Body fat	Fat free mass	biomass years	Biomass exposure (hours)	biomass exposure
42	sundari	35243		65	3	csb	3	2	31	5	48	3	2	0	D	1.46	45	21.11	2	266.9	240	89.9	4	44	1.028	31.57	30.80	30	90	1
43	sarasammal	8722		69	2	csbw	4	3	30	4	46	3	2	0	D	1.47	48	22.21	2	268.1	320	119.4	4	68	1.016	37.35	30.07	22	66	1
44	muthulakshmi	7352		59	2	cbw	5	4	31	5	64	2	5	2	D	1.33	40	22.61	2	263.9	240	90.9	4	58	1.020	35.22	25.91	15	45	1
45	nandini	5142		49	1	csb	3	3	24	4	70	2	0	1	D	1.47	42	19.44	2	283.7	250	88.1	4	56	1.026	32.31	28.43	0	0	2
46	durgadevi	8973		53	2	bw	2	2	27	4	56	3	3	0	D	1.43	56	27.39	3	311.3	280	90.0	4	82	1.010	39.88	33.67	10	20	1
47	kamala	4327		65	3	bw	2	3	28	4	48	3	0	2	D	1.62	54	20.58	2	288.4	270	93.6	4	50	1.024	33.25	36.05	30	90	1
48	bakkiyam	9273		49	2	bw	2	3	29	4	44	3	1	1	D	1.55	65	27.06	3	338.8	250	73.8	3	89	1.014	38.17	40.19	0	0	2
49	saraswati	65432		61	3	bw	2	3	32	5	51	2	1	0	B	1.55	43	17.90	1	268.0	250	93.3	4	38	1.032	29.65	30.25	25	75	1
50	mangal	4999		60	3	csb	3	2	33	5	37	3	2	0	D	1.53	62	26.49	3	315.1	130	41.3	2	46	1.027	32.15	42.07	0	0	2
51	mariammal	7893		65	2	bw	2	3	15	3	53	2	0	0	B	1.43	51	24.94	3	281.3	260	92.4	4	58	1.020	35.22	33.04	20	30	1
52	saroja	7231		57	2	csb	3	3	21	4	57	2	2	1	D	1.53	53	22.64	2	298.0	220	73.8	3	42	1.029	30.96	36.59	36	90	1
53	stellavid	933		67	3	bw	2	3	24	4	64	2	3	1	D	1.37	42	22.38	2	256.7	300	116.9	4	55	1.022	34.51	27.50	0	0	2
54	chitra	1447		47	1	cbw	5	2	12	3	73	2	2	0	D	1.39	38	19.67	2	277.1	310	111.9	4	45	1.032	29.59	26.75	0	0	2
55	nalini	5243		54	2	csbw	4	2	19	3	48	3	0	0	D	1.46	57	26.74	3	312.2	240	76.9	4	74	1.013	38.49	35.06	15	45	1
56	panimalar	876		57	2	csb	3	2	23	4	45	3	0	0	D	1.46	42	19.70	2	271.7	260	95.7	4	41	1.030	30.64	29.13	20	40	1
57	malarkodi	9981		59	2	cs	1	4	28	4	38	3	2	0	D	1.45	45	21.40	2	275.9	270	97.9	4	51	1.024	33.51	29.92	34	68	1
58	vennila	1034		60	2	csb	3	2	32	5	52	2	1	0	B	1.5	58	25.78	3	305.6	230	75.3	4	78	1.012	39.20	35.27	0	0	2
59	kanimozhi	6650		56	2	bw	2	2	17	3	63	2	1	0	B	1.52	46	19.91	2	282.7	300	106.1	4	38	1.032	29.65	32.36	12	18	1
60	meena	12999		62	3	bw	2	3	29	4	38	3	0	0	D	1.51	43	18.86	2	266.5	320	120.1	4	48	1.025	32.71	28.93	22	66	1
61	rukamani	18231		61	3	csb	3	3	20	3	67	2	0	0	B	1.48	44	20.09	2	270.5	280	103.5	4	57	1.021	34.99	28.61	28	84	1
62	zairunisa	5221		58	2	csb	3	3	29	4	52	2	4	0	D	1.42	45	22.32	2	277.4	270	97.3	4	48	1.025	32.71	30.28	0	0	2
63	zubeda	8888		58	2	csb	3	3	31	5	45	3	0	0	D	1.39	51	26.40	3	291.8	290	99.4	4	63	1.018	36.32	32.48	16	48	1
64	chinnammal	12008		42	1	bw	2	1	10	4	72	2	0	0	A	1.45	47	22.35	2	306.2	260	84.9	4	54	1.027	31.86	32.03	0	0	2
65	kumari	18543		57	2	cs	1	3	28	4	59	2	2	0	D	1.56	43	17.67	1	274.0	280	102.2	4	35	1.034	28.58	30.71	30	105	1
66	alamelu	16665		58	2	csb	3	3	18	3	57	2	1	0	B	1.54	41	17.29	1	267.7	300	112.0	4	42	1.029	30.96	28.31	25	75	1
67	lakshmi	19263		44	1	csb	3	2	21	4	65	2	1	0	B	1.38	42	22.05	2	291.2	280	96.2	4	52	1.028	31.39	28.82	0	0	2
68	samundeshwari	11423		48	1	csb	3	3	13	3	71	2	1	0	B	1.41	45	22.63	2	292.4	220	75.2	4	48	1.030	30.39	31.32	5	15	1
69	sundari	10987		58	2	cs	1	3	23	4	49	3	0	0	D	1.44	40	19.29	2	265.4	270	101.7	4	39	1.031	29.99	28.00	10	10	1
70	alagarasi	11149		61	3	csb	3	2	30	4	43	3	0	0	D	1.36	40	21.63	2	260.9	320	122.7	4	50	1.024	33.25	26.70	15	45	1
71	ambika	890		49		cs	1	2	28	4	52	2	0	0	B	1.45	51	24.26	3	305.3	170	55.7	3	69	1.021	34.93	33.18	0	0	2
72	amudham	21862		50	1	csb	3	3	27	4	57	2	2	0	D	1.61	48	18.52	1	296.5	240	80.9	4	39	1.031	29.99	33.61	33	99	1
73	chellammal	28654		62	2	cs	1	3	29	4	55	2	2	1	D	1.42	45	22.32	2	271.4	230	84.8	4	52	1.023	33.77	29.80	32	81	1
74	chitra	14263		58	1	csb	3	2	32	5	59	2	1	0	B	1.39	40	20.70	2	265.4	200	75.4	3	50	1.024	33.25	26.70	35	105	1
75	elavarasi	12634		57	2	cs	1	3	14	3	71	2	1	0	B	1.54	46	19.40	2	281.2	330	117.3	4	41	1.030	30.64	31.91	0	0	2
76	godavari	13827		55	2	csb	3	3	29	4	39	2	3	1	D	1.48	49	22.37	2	291.5	190	65.2	3	50	1.024	33.25	32.71	24	60	1
77	eshwari	18876		64	1	csb	3	2	18	3	65	2	0	0	B	1.52	52	22.51	2	285.1	300	105.2	4	44	1.028	31.57	35.59	23	69	1
78	kalatarasi	13267		41	1	cs	1	3	23	4	73	2	0	0	B	1.39	45	23.29	2	302.9	220	72.6	3	54	1.027	31.86	30.66	22	77	1
79	loganayaki	19949		52	2	csb	3	3	23	4	54	2	0	0	B	1.46	39	18.30	1	272.0	230	84.6	4	38	1.032	29.65	27.44	12	36	1
80	malarvill	11114		56	2	cs	1	3	31	5	48	2	1	1	D	1.35	39	21.40	2	266.0	150	56.4	3	45	1.027	31.86	26.57	35	105	1
81	thulasi	2360		45	1	csb	3	3	28	4	58	2	1	0	B	1.52	65	28.13	3	344.8	310	89.9	4	89	1.014	38.17	40.19	28	84	1
82	aayishabegum	4632		56	2	cs	1	2	25	4	43	3	2	0	D	1.55	46	19.15	2	282.7	200	70.7	3	46	1.027	32.15	31.21	31	93	1
83	vaijayanthi	8889		57	2	cs	1	3	26	4	51	2	0	0	B	1.44	44	21.22	2	256.8	250	97.4	4	45	1.027	31.86	29.98	30	90	1
84	kouslya	3124		68	3	cs	1	3	28	4	59	2	2	0	D	1.54	72	30.36	3	327.1	150	45.9	2	87	1.009	40.68	42.71	30	90	1
85	parvathy	12594		63	3	csbw	4	3	29	4	51	2	0	0	B	1.41	36	18.11	1	248.3	160	64.4	3	37	1.033	29.30	25.45	40	40	1
86	radha	4094		65	3	cs	1	3	32	5	45	3	1	0	D	1.47	45	20.82	2	266.9	200	74.9	3	48	1.025	32.71	30.28	25	75	1
87	lakshmiammal	21356		66	3	csb	3	3	30	5	43	3	1	1	D	1.43	48	23.47	2	272.6	220	80.7	4	58	1.020	35.22	31.10	33	99	1
88	poongothai	11378		67	3	csb	3	3	34	5	29	4	5	2	D	1.55	50	20.81	2	275.8	160	58.0	3	53	1.023	34.02	32.99	45	135	1
89	panimalar	8649		65	3	cs	1	3	23	4	59	2	0	0	B	1.42	49	24.30	2	276.5	240	86.8	4	79	1.012	39.37	29.71	21	84	1
90	kaliammal	783		69	3	csb	3	2	20	3	63	2	0	0	B	1.47	45	20.82	2	260.9	300	115.0	4	64	1.017	36.53	28.56	32	96	1
91	meenakshi	507		68	3	csbw	4	3	29	4	49	3	2	0	D	1.53	54	23.07	2	283.9	260	91.6	4	68	1.016	37.35	33.83	35	105	1

BM range	no.smoke /no.yrs	Smoking index	Second hand exposure	Type of housing	Separate kitchen	Number of windows	Nearness to busy road	Recurrent resp. infection	childhood infections	BA	Family h/o COPD	Education	no. years of schooling / college	Occupation	Occupation	Income per month	Socio economic class	DM	No. of years (diabetes)	SHT	No. of years (SHT)	IHD	PHQ 9 score	Psychiatric illness	Chest Xray	Eosinophils %	TC	AEC	other comorbid conditions	ct phenotype	CT PHENotype	PHT	reversibility	
2			2	1	1	0	1	1	1	1	2	1	0	HOUSEWIFE	1	10000	3	2	2			12	1	increased bvm	11	6460	711		M	3		1		
2			2	2	2	1	2	1	2	1	2	1	0	construction	5	6000	4	2	2			15	2	increased bvm	10	8670	867		M	3				
2			2	1	2	0	2	1	2	2	2	2	5	agricultural	6	4000	4	1	3	2			21	5	increased bvm	2	2430	49		M	3		1	
1			1	1	1	1	1	1	1	3	1	1	0	kolamavu maki	8	8000	4	2	2	2			15	4	increased bvm	26	8540	2220		E	1		1	
1			2	2	2	2	1	1	2	1	2	2	6	cook	3	7000	4	2	2			8	2	normal	4	3780	151		M	3				
1			2	1	2	0	1	2	2	1	2	2	6	garment	8	5000	4	2	2	2			11	3	normal	8	6430	514		A	1			
2			1	2	1	1	1	1	2	2	2	2	12	construction	5	4000	4	1	7	1	7			13	3	increased bvm	3	3820	115		A	1		1
2			1	1	1	0	2	2	2	1	2	1	0	agriculture	6	4000	4	1	10	1	10			9	2	hyperinflated	6	4046	243	DLE			1	
2			2	3	1	0	2	2	2	1	2	2	10	housewife	1	8000	3	2	2	2			14	3	hyprinflated	11	7630	839						
			2	1	2	0	1	2	2	2	2	1	0	housewife	1	2000	4	2	2	2			22	5	hyperinflated	4	5050	202		M	3		1	
2			2	4	2	0	2	2	2	1	2	2	5	housewife	1	4000	4	1	2	2			15	3	ystic translucenc	7	12040	843		B	2			
1			1	2	1	1	2	2	2	1	2	1	0	housewife	1	4000	4	2	2	2			21	5	normal	4	4840	194		A	1			
2			2	2	1	0	2	2	2	1	2	1	0	construction	5	2000	4	2	2	1	5	1		11	3	hyperinflated	6	3380	203					
2			1	1	2	0	2	1	2	1	2	2	5	housewife	1	5000	4	1	3	2			17	4	normal	13	9550	1242		B	2		1	
2			2	2	2	0	2	1	2	3	2	1	0	agriculture	6	12000	4	2	2	2			15	4	increased bvm	6	3400	204		A	1			
2			1	2	2	0	1	1	2	1	2	2	5	caretaker	8	8000	4	1	11	1	8			8	2	bronchiectasis	10	5420	542		B	2		
1			2	2	1	1	2	1	2	3	2	2	7	housewife	1	6000	4	2	2	2	5			14	3	airtrappingRUZ	5	7060	353	hypothyroid	M	3		
2			2	1	2	0	2	2	2	1	2	1	0	agriculture	6	5000	3	2	2	2			11	3	increased bvm	8	4320	346		M	3			
			2	3	1	5	2	2	2	1	2	2	10	housewife	1	8000	2	2	2	2			6	2	normal	10	6450	645						
2			1	2	2	2	2	1	2	1	2	2	6	flourpacking30yrs	7	3000	4	2	2	2			13	3	normal	9	9400	846					1	
2			2	1	2	0	2	1	1	1	2	1	0	sweeper	4	4000	4	2	2	1	3			9	2	increasedbvm	4	8200	328		A	1		
2			2	2	1	3	2	1	2	3	2	2	12	housewife	1	12000	3	1	6	2			14	3	normal	9	11099	999		A	1		1	
			2	2	2	1	1	1	2	1	2	1	0	housewife	1	15000	3	2	2	2			7	2	tparacardiac haz	6	12090	725		A	1			
2			1	3	1	1	1	2	2	1	2	2	10	construction	5	5000	3	1	8	2			14	3	normal	4	3290	132		M	3			
2			2	1	2	0	2	2	2	1	2	1	0	agriculture	6	4000	4	2	2	2			21	5	increased bvm	5	5320	266				1		
2			1	1	2	0	2	2	1	3	2	2	3	construction	5	5000	4	1	5	2			2	1	normal	10	8130	813		A	1		1	
			2	2	1	1	2	1	2	1	2	1	0	housewife	1	5000	4	2	2	2			12	3	normal	7	6600	462						
2			2	1	2	0	2	1	1	2	2	1	0	construction	5	2000	5	1	3	1	3			9	2	emphysemall	10	5500	550		M	3		
2			2	3	1	2	2	1	2	1	2	1	0	agriculture	6	2000	5	2	2	2			8	2	bronchiectasis	14	5730	802		B	2		1	
1			2	2	1	0	2	2	2	1	2	1	0	agriculture	6	4000	4	2	2	2			12	3	normal	11	6180	680					1	
2			2	1	2	0	1	2	1	3	2	2	2	sand	8	3000	4	2	2	2			17	4	normal	4	3430	137		A	1			
2			1	1	2	0	1	1	2	1	2	1	0	housewife	1	5000	4	2	2	2			12	3	bronchiectasis	15	7060	1059		B	2			
2			1	1	2	0	1	1	2	1	2	1	0	construction	5	4000	5	2	2	1	5			19	4	bronchiectasis	9	7890	710		B	2		
2			2	2	2	2	2	2	1	2	2	1	0	housewife	1	3000	5	1	10	1	7	1	10	3	increased bvm	8	5033	403		M	3		1	
1			1	1	1	1	2	2	2	1	2	2	6	wheatflourpacking	7	4800	4	2	2	2			11	3	increased bvm	12	8036	964						
2			2	3	1	2	2	2	1	2	1	0	cook	3	3000	4	1	1	1	4			8	2	bronchiectasis	4	7090	284		B	2			
			2	3	1	1	2	2	1	3	2	1	0	hou sewife	1	5000	4	2	2	2			11	3	normal	11	9985	1098	hypothyroid	A	1		1	
2			1	2	2	0	1	1	2	1	2	1	0	cook	3	4000	4	1	5	1	4	1	20	5	increased bvm	18	13028	2345						
			2	2	1	1	2	1	1	3	2	1	8	betelnut	6	4500	4	2	0	1	3			14	3	normal	6	4056	243	systemic sclerosis	A	1		1
1			1	1	2	0	2	1	2	1	2	1	0	housewife	1	8000	4	2	0	1	8			14	3	increased bvm	5	3740	187		E	4		
2			2	1	2	0	1	1	2	1	2	2	5	agriculture	6	6500	4	2	0	2	0			19	4	bronchiectasis	4	8070	323		B	2		1

BM range	no.smoke /no.yrs	Smoking index	Second hand exposure	Type of housing	Separate kitchen	Number of windows	Nearness to busy road	Recurrent resp. infection	childhood infections	BA	Family h/o COPD	Education	years of schooling / college	Occupation	Occupation	Income per month	Socio economic class	DM	No. of years (diabetes)	SHT	No. of years (SH T)	IHD	PHQ 9 score	Psychiatric illness	Chest Xray	Eosinophils %	TC	AEC	other comorbid conditions	ct phenotype	CT PHENOTYPE	PHT	reversibility	
2			1	3	1	1	2	2	2	2	2	2	3	flourpacking	7	10000	4	1	7	2	0		21	5	normal	4	4032	161		M	3		1	
2			1	2	1	1	2	2	2	1	2	1	0	housewife	1	3000	4	2	0	1	6	1	17	4	increased bvm	6	7640	458		M	3			
1			2	1	2	0	1	2	1	1	2	1	0	agriculture	6	4000	4	2	0	1	5		18	4	hyperinflated	8	6820	546		E	4			
1	10/25	2	1	1	2	0	2	1	1	1	2	1	0	agriculture	6	6000	4	1	5	2	0		8	2	normal	10	8540	854		E	4			
2			1	1	2	0	1	2	2	1	2	1	0	housewife	1	5000	4	2	0	2	0		10	3	L.Ulmass	11	7440	818	lung ca	E	4			
2			1	1	2	0	1	2	2	1	2	1	0	housewife	1	2000	5	2	0	2	0	1	8	2	hyperinflated	7	3830	268		E	4			
2			1	2	1	2	1	1	1	1	2	2	10	housemaid	2	5000	4	1	8	1	8		15	3	normal	4	8640	346		E	4			
2	10/15	2	1	2	2	0	1	1	1	3	2	2	2	housemaid	2	6000	4	2	0	2	0		12	3	normal	5	3860	193		A	1			
1			2	1	2	0	1	1	2	3	2	2	6	construction	5	3000	4	2	0	2	0		13	3	hyperinflated	5	4066	203		E	4			
2			2	1	2	0	2	2	2	1	2	2	4	cook	3	8000	4	2	0	2	0		18	4	hyperinflated	4	4500	180		E	4			
2			1	1	2	0	2	1	2	1	2	1	0	construction	5	3000	4	2	0	2	0		21	5	normal	3	2890	87		A	1			
			2	1	2	0	2	2	1	3	2	1	0	housemaid	2	4000	4	2	0	2	0		15	4	hyperinflated	11	3420	376		M	3		1	
			2	4	2	0	1	2	1	2	2	3		housewife	1	2000	4	2	0	1	5		23	5	hyperinflated	5	5086	254		M	3		1	
1			1	1	2	0	1	1	1	1	2	1	0	agriculture	6	4500	4	1	7	2	0		7	2	normal	6	3034	182		E	4			
1			1	2	1	1	1	1	2	2	2	2	8	housewife	1	10000	3	2	0	2	0		9	2	increased bvm	14	8170	1144		M	3		1	
2			2	1	2	1	2	1	2	3	2	2	4	sweeper	4	3000	4	2	0	2	0		11	3	hyperinflated	12	11080	1330		A	1		1	
			2	1	2	0	2	2	2	1	2	2	3	housewife	1	7000	4	2	0	1	3		18	4	hyperinflated	5	5042	252		E	4			
1			2	1	2	1	2	2	1	2	2	1	0	construction	5	4000	4	1	4	1	6		13	3	increased bvm	14	9830	1376		A	1		1	
2			1	2	2	0	1	2	1	1	2	2	2	sweeper	4	10000	4	1	5	2	0		4	1	hyperinflated	9	6580	592		M	3			
2			1	1	2	0	1	1	2	1	2	1	0	housemaid	2	6000	4	2	0	2	0		14	3	normal	13	6046	786		M	3			
			1	1	2	0	1	2	2	1	2	1	0	housewife	1	4000	4	2	0	1	6		6	2	normal	6	4320	259		A	1		1	
1			2	1	2	0	2	2	1	1	2	1	0	housewife	1	4000	4	2	0	2	0		21	5	bronchiectasis	7	13768	964		B	2		1	
			2	3	1	2	1	1	2	3	2	1	0	construction	5	15000	4	2	0	2	0		8	2	normal	18	5840	1051		M	3			
2			1	2	1	1	2	2	2	1	2	1	0	construction	5	5000	4	2	0	2	0		15	4	increased bvm	15	9876	1481		A	1		1	
2			2	1	2	0	2	2	1	1	2	1	0	housewife	1	5000	4	1	8	1	8	1	21	5	increased bvm	12	8650	1038		M	3		1	
			2	2	2	1	2	1	1	2	2	1	0	housewife	1	4000	4	2	0	1	4		13	3	increased bvm	4	3460	138		E	4		1	
1			1	1	2	0	1	2	2	1	2	2	10	housewife	1	10000	3	1	5	1	10		14	3	normal	2	2850	57		M	3			
1			2	1	2	0	1	2	2	1	2	2	5	construction	5	5000	4	1	6	2	0		9	2	increased bvm	1	3076	31		A	1		1	
1			1	1	2	0	2	1	2	1	2	1	0	agriculture	6	4000	4	2	0	2	0		17	4	normal	4	3504	140		A	1			
			1	1	2	0	1	2	2	1	2	1	0	construction	5	4000	4	2	0	1	4		15	4	increased bvm	3	4920	148		A	1			
2			2	1	2	0	1	1	2	1	2	2	3	cook	3	7000	4	2	0	2	0		15	4	normal	8	4062	325		M	3		1	
2			1	1	2	0	1	1	1	2	2	1	0	housewife	1	3000	4	2	0	1	2		18	4	increased bvm	13	9860	1282		B	2		1	
2			1	2	1	1	2	2	1	2	2	1	0	housewife	1	6000	4	2	0	2	0		12	3	increased bvm	7	4320	302		E	4			
			2	3	1	1	1	1	2	1	2	1	0	housemaid	2	5000	4	2	0	2	0		7	2	normal	18	8053	1450		A	1		1	
2			2	1	2	0	1	2	2	1	2	2	6	housewife	1	4000	4	2	0	1	3		11	3	bronchiectasis	10	8090	809		B	2			
2			2	1	2	0	2	2	2	1	2	2	1	0	construction	5	7000	4	1	4	2	0		13	3	increased bvm	3	3680	110		A	1		1
2			2	1	2	0	2	2	1	1	2	1	0	housewife	1	5000	4	1	3	2	0		15	4	increased bvm	10	7420	742		E	4			
1			1	2	2	0	1	1	1	2	2	1	0	agriculture	6	4000	4	2	0	2	0		15	4	normal	14	10540	1476		A	1		1	
2			2	1	2	0	2	2	2	1	2	1	0	agriculture	6	2000	5	2	0	2	0		9	3	increased bvm	9	4043	364		E	4			
2			1	1	1	1	1	2	2	3	2	1	0	sweeper	4	5000	4	2	0	1	6		10	3	increased bvm	11	11060	1217		M	3			
2			2	1	2	0	1	2	2	2	2	1	0	housewife	1	12000	3	1	3	1	3		11	3	normal	10	7840	784		A	3		1	
2			2	1	2	0	1	1	2	1	2	2	5	housewife	1	5000	4	2	0	2	0		13	3	increased bvm	7	9766	684		A	1		1	
			1	1	2	1	2	1	2	1	2	1	0	sweeper	4	5000	4	2	1	10	1	10	1	19	4	increased bvm	6	3860	232		B	2		
2	7/28	2	2	1	2	1	2	2	1	2	1	0	0	housewife	1	5000	4	1	8	2	0		13	3	increased bvm	4	5840	234	lung ca	E	4			
2			2	1	2	0	2	2	2	1	2	1	0	agriculture	6	4000	4	1	10	1	15	1	13	3	increased bvm	10	6748	675		A	1		1	
2			1	1	2	0	2	1	2	1	2	2	6	construction	5	4000	4	2	0	1	12		21	5	increased bvm	12	5832	700		M	3		1	
2			1	1	2	1	1	1	2	1	2	1	0	housewife	1	4000	4	2	0	2	0		20	5	increased bvm	6	9870	592		B	2		1	
2			2	1	2	1	2	2	1	1	2	1	0	housewife	1	6000	4	1	20	2	0		15	4	increased bvm	6	6324	379		M	3			
2			1	1	2	1	1	2	1	2	2	2	2	construction	5	3000	4	2	0	2	0		12	3	increased bvm	8	8432	675		A</				