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ANALYSIS OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE WITH CLINICAL PARAMETERS, ECG AND ECHO

Satish Kinagi[1], Sharan Patil[2], Sayeeda Afiya[3]

ABSTRACT: BACK GROUND: Chronic obstructive pulmonary disease (COPD) is a leading cause of morbidity and mortality in countries of high, middle, and low income. Estimates from WHO's Global Burden of Disease and Risk Factors project show that in 2001, COPD was the fifth leading cause of death in high-income countries, accounting for 3.8% of total deaths, and it was the sixth leading cause of death in nations of low and middle income, accounting for 4·9% of total deaths. **OBJECTIVES:** 1. To study clinical parameters of chronic obstructive pulmonary disease. 2. To find out Electrocardiographic changes of chronic obstructive pulmonary disease. 3. To confirm with echocardiogram the presence of pulmonary hypertension, tricuspid regurgitation and right heart failure and analyze the incidence of right heart failure and pulmonary hypertension. **MATERIALS AND METHODS:** Single center hospital based cross sectional study. Patients diagnosed as COPD based on following steps will be included in the study. The patients with cough, sputum production, dyspnoea (wheeze) was chosen (sputum AFB negative will be confirmed). Pulmonary function test was done to pick up patients with reduced FEV<70% was chosen. They were nebulized with salbutamol bronchodilator and PFT was repeated to select patients with <15% reversibility. All the data was tabulated and subjected to statistical analysis. Descriptive statistics, such as mean, SD and proportion, and other appropriate statistical tests used. **OBSERVATIONS:** Out of 72 patients, 39 patients were males, 34 were smoker. Among 72 patients, 24 patients had P-pulmonalae, 41 patients had right axis deviation, 7 had R-wave height in lead V1 more than 5 mm according to ix electrocardiogram (indicating they had severe right ventricular hypertrophy or pulmonary hypertension). Among 72 patient, 17 were echocardiographically had pulmonary hypertension, 15 had tricuspid regurgitation (which is non-invasive method of measurement of pulmonary artery pressure), which we cannot afford to measure via catheter. 12 out of 72 patients had P-wave amplitude in lead II+ lead-III + lead aVF >9 mm, as this is the one of the indication for life long oxygen therapy as per American Thoracic Society (ATS). Out of 72 patients, 12 had coronary artery disease (CAHD) as this increases the incidence of cor-pulmonale. **CARDIOVASCULAR COMPLICATIONS:** Out of 72 patients, 24% developed pulmonary hypertension, 22% developed tricuspid regurgitation, 34% had p-pulmonale, 18% had p-wave amplitude in lead-II + lead-III + lead a VF >9 mm, this is important because this is one of the indication for life long oxygen therapy. 18% had concomitant coronary artery disease (CAHD), this observation is important because systemic inflammation plays enhanced role in atherosclerosis, diabetes mellitus, tumour necrosis factor is increased in COPD patients. **CONCLUSION:** Pulmonary hypertension was the most common cardiovascular complication leading to corpulmonale, and emphysema was more common in smokers, bronchitis was more common in females, the prevalence of COPD was more in urban areas due to pollution of air by automobiles. The textile based industries were mainly responsible for the increased prevalence of COPD.

KEYWORDS: COPD; Pulmonary hypertension; Corpulmonale; Emphysema.

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INTRODUCTION: DEFINITION: The American Thoracic Society defines COPD as a disorder characterized by abnormal tests of expiratory flow (on a structural or functional basis) that do not change markedly over several months of observation[1]. The diagnosis usually refers to the presence of chronic obstructive bronchitis with varying degrees of emphysema and small air way disease.

The American Thoracic Society, defines chronic obstructive pulmonary Disease (COPD) as a disease state characterized by the presence of airway Obstruction due to chronic bronchitis and/or emphysema, the airflow obstruction is generally progressive may be accompanied by airway hypersensitivity and may be partially reversible. Diagnostic of this non-specific entity are chronic productive cough, wheeze, breathlessness on exertion, physiological evidence of airflow Limitation (reduced FEV1), and poor reversibility (ex, response to bronchodilator).[1]

CHRONIC OBSTRUCTIVE PULMONARY DISEASE: They are divided into chronic bronchitis, Emphysematous patients. Typically they have reduced FEV1 and reversibility less than 15 percent while bronchial asthma patient have more than 15 percent reversibility

Emphysema.[1,2] This entity is defined in anatomic terms–abnormal permanent enlargement of the gas–exchanging units of the lungs (acini) in association with destruction of the alveolar walls and without obvious fibrosis.

The predominant physiologic consequence of these anatomic abnormalities is a decrease in the elastic recoil of the lungs–which in turn causes outward displacement of the chest wall and flattening of the diaphragm. Hyperinflation of the lungs, and increased resistance to airflow, which inurn increases the work of breathing, although imbalances in ventilation Perfusion are seldom marked in as those in chronic bronchitis, derangement sufficient to cause arterial hypoxemia are common.

Chronic bronchitis.[1,2]

Chronic bronchitis, a clinically defined condition with chronic cough and sputum; and small airways disease, a condition in which small bronchioles are narrowed. As has been pointed out chronic bronchitis is not a single clinical entity, and it is the airflow limitation that it produces that leads to morbidity and mortality, this limitation has been identified from a decrease in FEV1.[1,2] The current GOLD and ATS/ERS definition for airflow limitation is an FEV1/FVC ratio of <70%.

Although this "fixed" ratio is easy to remember and simple, there is some concern that it may underestimate COPD in younger populations, overestimate it in older ones, and misclassify other patients declining lung function over time is an important component in understanding the natural history of COPD. The concept that different populations (i.e. susceptible smokers, non-susceptible smokers, nonsmokers) have different trends in their lung function decline was developed, although individual patients may have a great deal of variability in their lung function decline over time.

Global initiative of obstructive lung disease (GOLD) has classified COPD as "a disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases."[3,4] This definition has also been adopted in the new ATS/ERS guidelines, with the important observation that COPD is both preventable and treatable.

Impaired lung function is of central importance in the diagnosis of COPD. Airflow limitation is the slowing of expiratory airflow as measured by spirometry, with a persistently low forced

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expiratory volume in one second (FEV1) and a low FEV1/ forced vital capacity (FVC) ratio despite treatment.[3,4]

Lung Function[3,4]

GOLD has classified COPD as "a disease state characterized by airflow limitation that is not fully reversible.

The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases". This definition has also been adopted in the new ATS/ ERS guidelines, with the important observation that COPD is both preventable and treatable. Impaired lung function is of central importance in the diagnosis of COPD.

Airflow limitation is the slowing of expiratory airflow as measured by spirometry, with a persistently low forced expiratory volume in one second (FEVI) and a low FEVI/ forced vital capacity (FVC) ratio despite treatment. The current GOLD and ATS/ ERS definition for airflow limitation is an FEVI/ FVC ratio of <70%. Although this 'fixed' ratio is easy to remember and simple, there is some concern that it may underestimate COPD in younger populations, overestimate it in older ones, and misclassify other patients.

Declining lung function over time is an important component in understanding the natural history of COPD. The concept that different populations (i.e., susceptible smokers, non-susceptible smokers, non-smokers) have different trends in their lung function decline was developed by Peto et al and expanded by Burrows et al. Interventions, such as smoking cessation, have been shown to alter this trend in populations, although individual patients may have a great deal of variability in their lung function decline over time.[4]

MATERIALS AND METHODS: Source of data: The study was conducted in 72 COPD patients admitted in Basaveshwar Teaching and General Hospital, Gulbarga attached to Mahadevappa Rampure Medical College from November 2011 to May 2013. Detailed history and thorough examination and routine investigations were done. The sputum was sent for routine examination, sputum for AFB negative confirmed and other investigations like electrocardiogram, echocardiograph, pulmonary function test and others were performed.

Methods of collection of data (including sampling procedure, if any):
SAMPLE SIZE: 72 patients.

$n = z^2 \times P \times q / m^2$ where.

n = number of subject required.

z = value of the normal deviate considered level of confidence = 1.96.

p = 4.9% = 0.0497.

q = 1-p=0.951.

m = margin of error at 5%.

n = 1.962x0.049x.951.

n = 72.

STUDY SUBJECTS: COPD patients, admitted in Basaveshwar Teaching and General Hospital, attached to Mahadevappa Rampure Medical College. Study period one and half years Nov 2011 to May 2013.

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INCLUSION CRITERIA: SELECTION CRITERIA:

Step 1: The patients with cough, sputum production dyspnea [wheeze] are chosen and sputum AFB negative confirmed.

Step 2: Pulmonary function test was done to pick up patients with reduced FEV<80% were chosen.

Step 3: They are nebulized with salbutamol bronchodilator and PFT was done to select patients with < 15% reversibility.

Step 4: They were clinically examined for over inflation, ascites, edema, parasternal heave, JVP, loud P2, tricuspid murmur.

Step 5: X ray chest PA view, lateral view taken to rule out the right pulmonary artery dilatation >16 mm [characteristic of pulmonary arterial hypertension] lateral view to rule out retrosternal space obliteration [RVH].

Step 6: ECG was taken in patients to look for p wave morphology, amplitude, 2, +3 +, avF P amplitude > 9mm, p-axis, R-wave > 5mm, r/s> 50%, RBBB.

Step 7: Echocardiogram was done for the chosen patients chosen Looked for pulmonary arterial hypertension, and tricuspid regurgitation [as it is the non-invasive measure of pulmonary arterial pressure, surrogate, for measuring PAP].

 The patients were selected from the Basaveshwar Teaching & General Hospital, Gulbarga attached to M.R. Medical College, Gulbarga. They are interviewed for the symptoms like cough, dyspnea, and wheeze. They were examined to look for signs of pulmonary hypertension, Corpulmonale.

EXCLUSION CRITERIA:
- Presence of asthma or other chronic restrictive respiratory diseases justifying the ventilator disorder.
- Presence of malignancy or any serious comorbidity that would prevent the study completion.
- Patients with pulmonary function tests with FEV1>80% and reversibility >15%.
- Patients with pulmonary tuberculosis were excluded. After patients labelled as COPD as mentioned above in the inclusion criteria, patients were subjected to following investigations as follows. They were examined for over inflation, ascites, edema, parasternal heave, raised JVP, loud p2, tricuspid murmur. ECG was taken and looked for P wave morphology and p wave amplitude in lead-II, lead-III, and a VF, also P wave axis, R wave amplitude in chest leads V1 to V6, RBBB etc. Echocardiogram was done and will be looked for Pulmonary artery hypertension and tricuspid regurgitation as it is non-invasive measure of pulmonary arterial pressure, surrogate for measuring PAP.

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STUDY DESIGN:
CROSS SECTIONAL STUDY:
STATISTICAL ANALYSIS:

- Single center hospital based cross sectional study.
- All the data was tabulated and subjected to statistical analysis. Descriptive statistics, such as mean, SD and proportion, and other appropriate statistical tests used.

Table-1: GOLD criteria for COPD severity[5]

| GOLD stage	Severity	Symptoms	Spirometry
0	At risk	Chronic cough, sputum production	Normal
I	Mild	With or without chronic cough or sputum production	$FEV_1/ FVC <0.7$ and $FEV_1 \geq 80\%$ predicted
IIA	Moderate	With or without chronic cough or sputum production	$FEV_1/ FVC <0.7$ and $50\% \leq FEV_1 <80\%$ predicted
III	Severe	With or without chronic cough or sputum production	$FEV_1/ FVC <0.7$ and $30\% \leq FEV_1 <50\%$ predicted
IV	Very severe	With or without chronic cough or sputum production	$FEV_1/ FVC <0.7$ and $FEV_1 <30\%$ $FEV_1 <50\%$ predicted with respiratory failure or signs of right heart failure

Abbreviation: GOLD – Global initiative for Lug Disease

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RESULTS:

Table-2: Occupation wise distribution of cases (n=72)

Occupation	Number	Percentage
Farmers (Rural)	15	21
Urban people	28	39
Industrial labours	15	21
Agriculture laborers (rural)	7	9
Others	7	10

Figure-1: Occupation wise distribution of cases
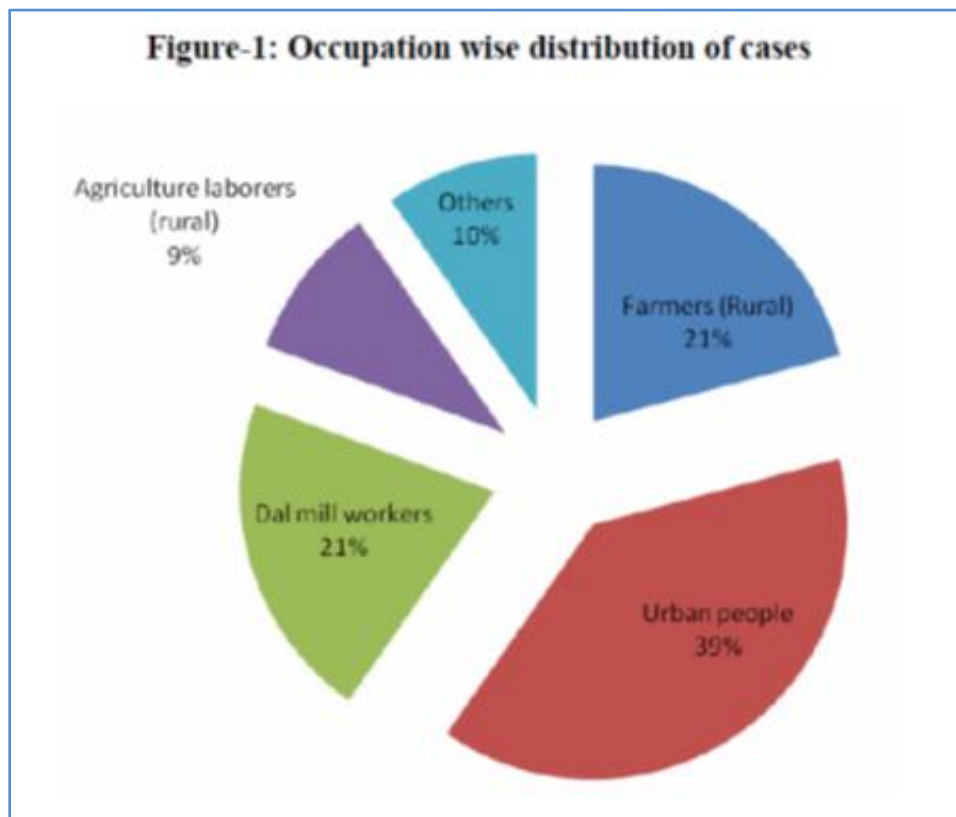
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Table-3: Sex wise distribution of cases (n=72)		
	Number	**Percentage**
Male	58	80.6
Female	14	19.4
Total	72	100

Figure-2: Sex wise distribution of cases
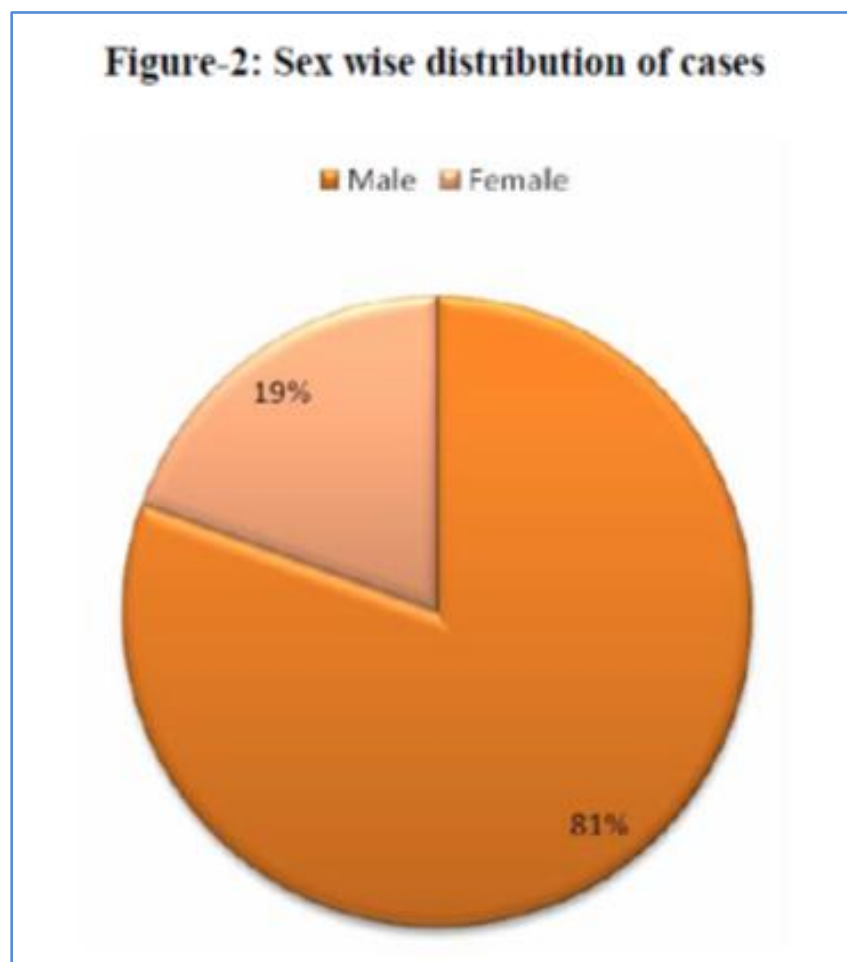
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Table-4: Age wise distribution of cases			
Age(years)	Male (%)	Female (%)	Total
40 – 50	7 (12)	4 (29)	11
50 – 60	18 (31)	3 (21)	21
60 – 70	21 (36)	5 (36)	26
70 – 80	11 (19)	2 (14)	13
> 80	1 (2)	0	1
Total	58	14	72
Mean (SD) age 62.9 ± 9.7			

Figure-3: Age wise distribution of cases
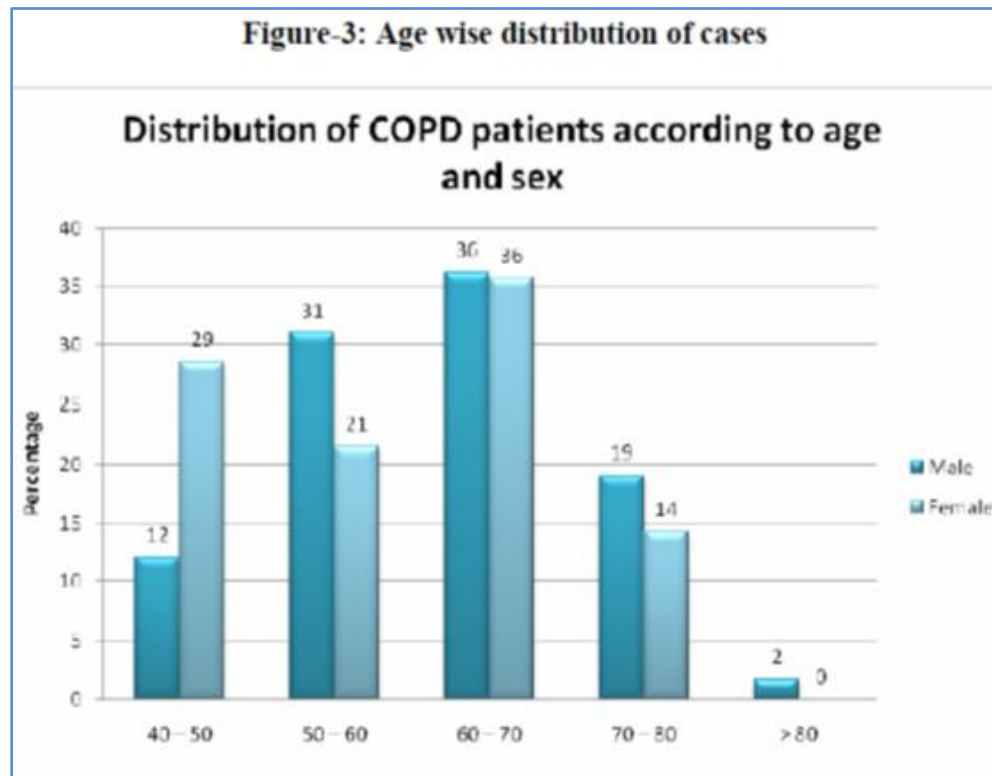
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Table-5: Symptoms		
Symptoms	Number	Percentage
Cough	72	100
Dyspnea	64	88.9
Wheeze	72	100

Figure-4:

Distribution of COPD patients according to symptoms
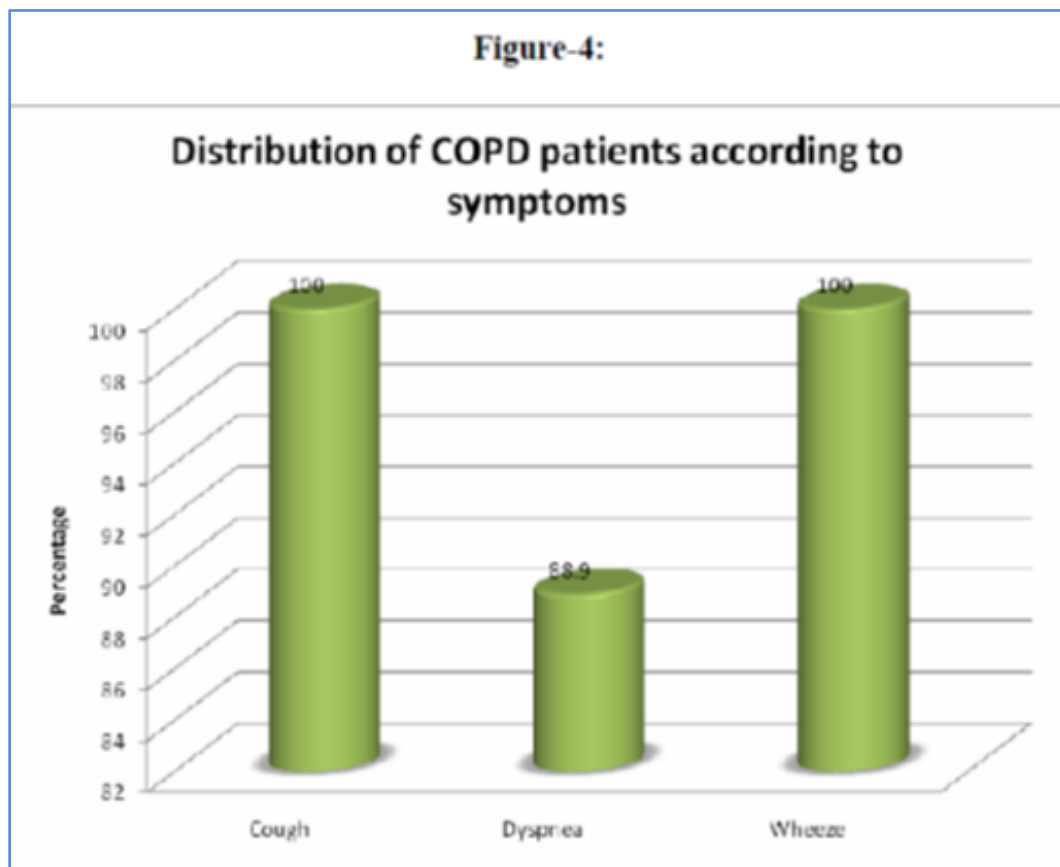
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Table-6: Signs		
Sign	Number	Percentage
JVS	14	19.4
Ascites	16	22.2
Pedal ede	15	20.8
Parasthe	15	20.8
loud_p2	17	23.6

Figure-5:

Distribution of COPD patients according to sign
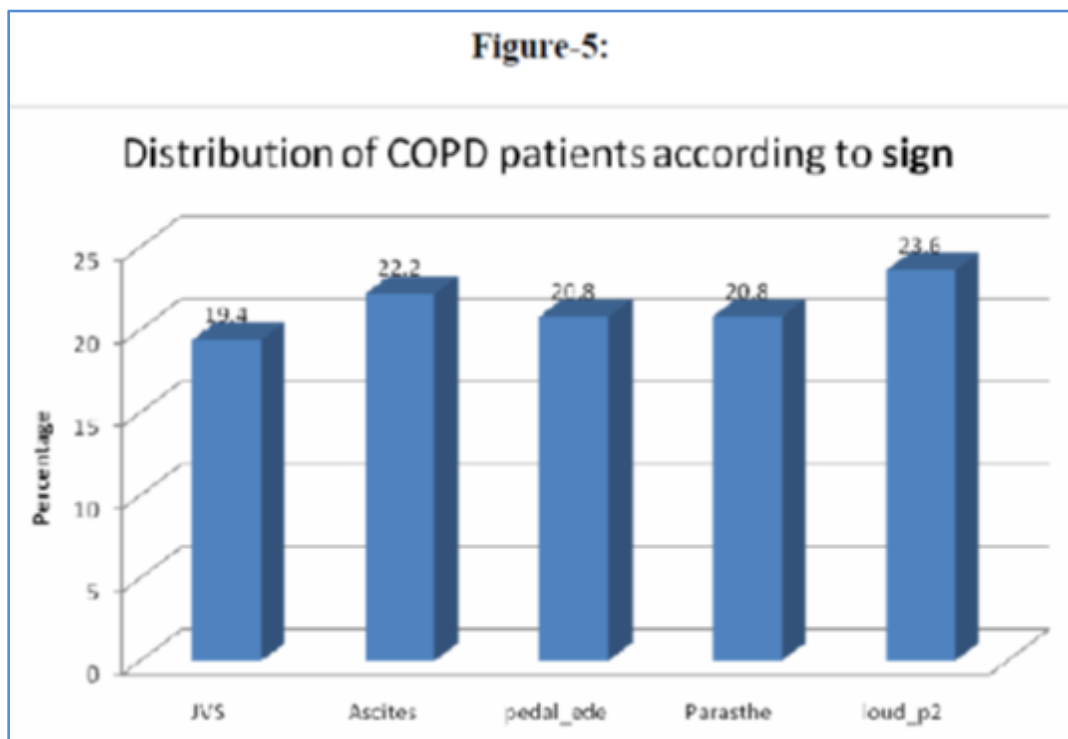
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Table-7: History of smoking		
	Number	Percentage
Smoker	33	45.8

Table-8: Gender-wise history of smoking			
Smoker	Male (%)	Female (%)	Total
Absent	32 (82)	7 (18)	39
Present	26 (79)	7 (21)	33
Total	58	14	72

Figure-6:

Distribution of COPD patients according to sex and smoking

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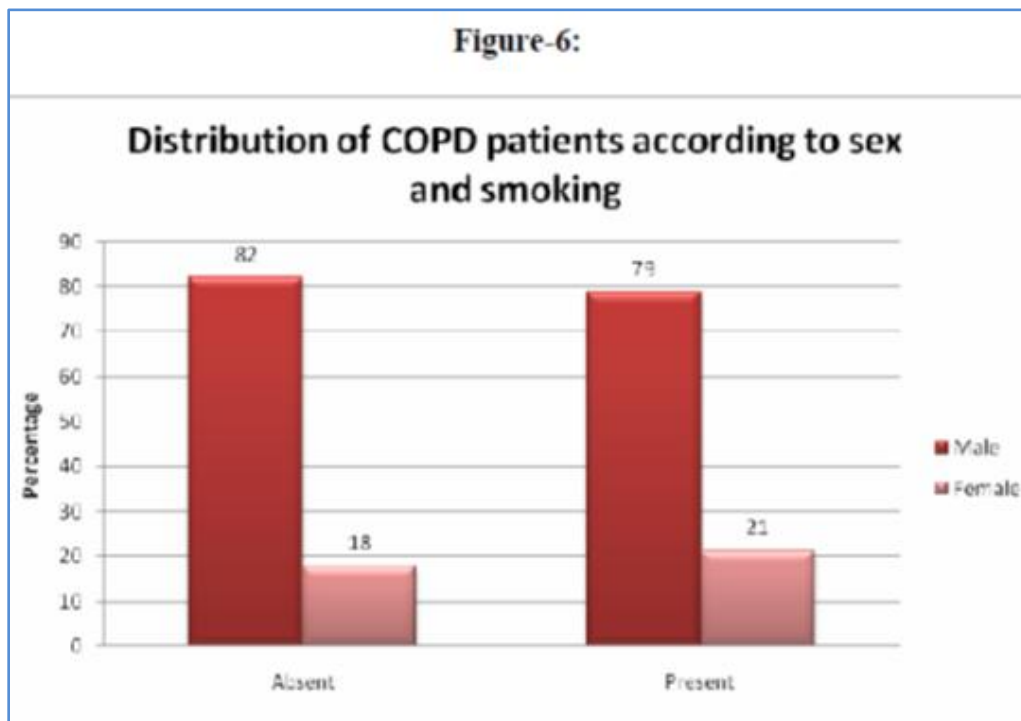
Table-9: Pulmonary function test analysis		
	Number	Percentage
PFT FEV$_1$ < 80%	72	100
Reversibility < 15 %	68	94.4

Figure-7:

Pulmonary function test analysis
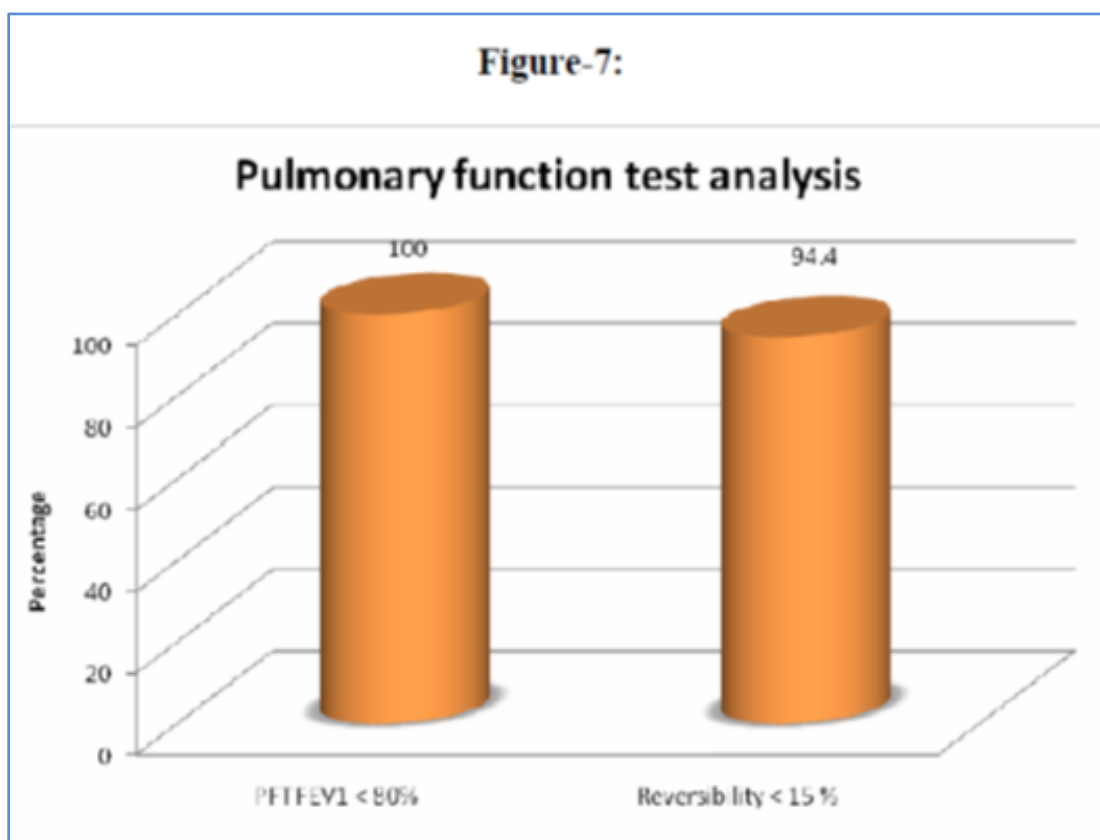
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Table-10: ECG finding		
	Number	Percentage
P-Pulmonale	24	33.3
Rt axis	41	56.9
R > 5 mm in lead-V_1	7	9.7
Lead-II+III+avF p wave amplitude> 9mm	12	16.7
RBBB	2	2.8
CAHD	12	16.7

Figure-8:

ECG Findings
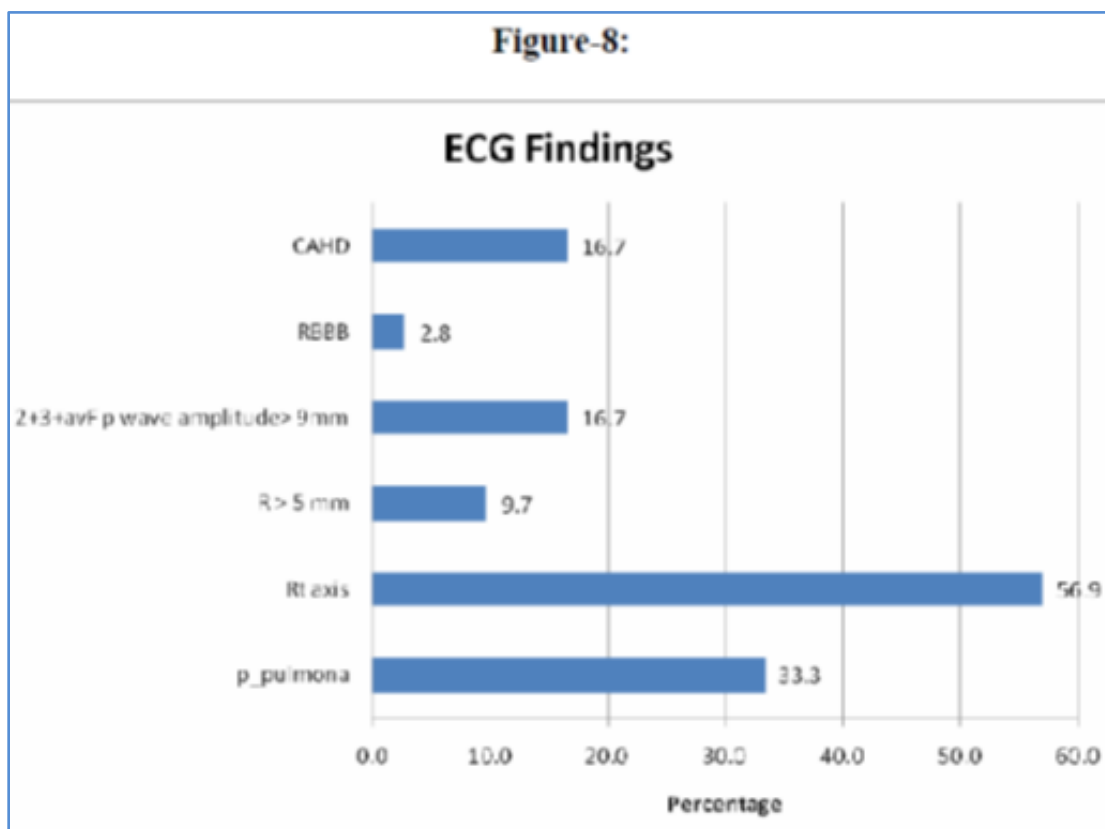
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DISCUSSION: From the above data and analysis, It is clear male patients with Smoking history is more prone for COPD, in particular Emphysema. Out of the 72 patients, 39 patients were males among them 34 Patients were smokers. Out of these 34 males presented with, cough, Dyspnea, Wheeze. Among the 72 patients ECG 24 patients had p pulmonale, Patients had right axis, deviation 7 had R wave height more than 5 mm (indicating they had severe Right ventricular hypertrophy or pulmonary hypertension).

12/72 had 2+3+avF P-wave amplitude > 9mm as this the one of the indication for Long Term Oxygen Therapy as per The American Thoracic Society. Also the ECGs were looked for any ischemic heart disease 12 Out of 72 had coronary artery disease, as this increases the incidence of corpulmonale smoking is an risk factor for both COPD [Emphysema, Bronchitis] and coronary artery disease. Among the 72, were echo-cardio-graphically had pulmonary hypertension 15 had tricuspid regurgitation (which is a noninvasive measure of pulmonary arterial pressure), which we cannot afford to measure via catheter.

INDUSTRIALIZATION AND IMPACT: Out of the 72 patients 15 patients were working in industries in Gulbarga. 15 were working in industries, generally the incidence of emphysema was more common in smokers 34 /39 in the group of 72 patients. In smokers working in textile industries, it was the bronchitis that was more common, while smoking commonly produce centri acinar emphysema, in textile industry workers the fine fabric related chemical particles increase the incidence of bronchitis in these patients.

Out of the 72 COPD patients, 28 were from urban areas, among the 28, 9 were urban females without smoking history, but they had COPD. 7 had bronchitis, one had mild COPD, one had emphysema. This indicates the increased level pollution in urban areas. 19 males were COPD patients. All the 19 were smokers and 17 out the 19 were emphysematous, 2 were bronchitis patients.

While just 15 out of 72 were from rural areas, the major air pollutants were released from automobiles like carbon monoxide, carbon dioxide, sulpurdioxide, lead, so urbanization had its toll, by increasing the level of pollutants. In the air and increasing the number of COPD patients even though Gulbarga has green trees all around but had high level of COPD patients, so we want strict pollution control acts which should be implemented strictly.

From the above data we can infer that the rapid pace of Urbanization, pollution of air, industry, farming activities, smoking are major triggers for COPD more over the complications of COPD like pulmonary hypertension corpulmonale were better found by ECG and echocardiogram. The use of ECG was in detecting the patients who need O2 therapy [if the combined 2+3+avF p-wave amplitude more than 9 mm] the evidence of pulmonary hypertension right ventricular hypertrophy, detected well.

Normally it is difficult to estimate pulmonary arterial pressure invasively via catheter but Easy to measure it indirectly if we measure it by detecting Tricuspid regurgitation non-invasively by echocardiogram, 15 patients had tricuspid regurgitation.

Cardiovascular complications: Out of the 72 COPD patients, 24 % developed pulmonary hypertension, 22% developed tricuspid regurgitation, the chronic hypoxia of COPD is responsible for

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the development of pulmonary hypertension. The tricuspid regurgitation is the best possible non-invasive way of measuring pulmonary artery pressure.

34 % patients had p-pulmonale, 18% patients had the total p wave amplitude in 2, 3, avF more than 9 mm, this is important because this one of the indication for life long oxygen therapy. 18% patients had concomitant coronary artery disease, this observation is important, because systemic inflammation plays enhanced role in atherosclerosis, diabetes. TNF alpha is increased in COPD, patients.

CONCLUSION: Pulmonary hypertension was the major cardiovascular complication of COPD, the chronic hypoxia of COPD is responsible for the development of pulmonary hypertension. 24% of the total cases had pulmonary hypertension. The need for life long oxygen therapy was re-emphasized in this study by establishing that nine patients had p- wave amplitude summation in lead 2, 3, avF, of more than 9 mm. The study also established that the best non-invasive way to detect raised pulmonary arterial pressure was detecting tricuspid regurgitation by echocardiogram.

Males predominantly had emphysema female had predominantly chronic bronchitis with corpulmonale. 12/72 had 2+3+avF P wave amplitude >9m as this the one of the indication for Long term oxygen Therapy as per The American Thoracic Society. Urbanization, pollution of air, industry, farming activities, smoking are major triggers for COPD more over the complications of COPD like pulmonary hypertension, cor-pulmonale were better found by ECG and echocardiogram. The use of ECG was in detecting the patients who need O2 therapy [if the combined 2+3+avF p-wave amplitude more than 9 mm].

The evidence of pulmonary hypertension, right ventricular hypertrophy were detected well. Normally it is difficult to estimate pulmonary arterial pressure invasively via catheter but easy to measure it indirectly if we measure it by detecting Tricuspid regurgitation non-invasively by echocardiogram. 18% patients had concomitant coronary artery disease, this observation is important, because systemic inflammation plays enhanced role in atherosclerosis, diabetes (TNF alpha is increased in COPD, patients).

Finally the study by analyzing COPD patients with clinical parameters, ECG, echocardiogram concluded that pulmonary hypertension was the most common cardiovascular complication leading to corpulmonale, and emphysema was more common in smokers, bronchitis was more common in females, the prevalence of COPD was more in urban areas due to pollution of air by automobiles. The textile based industries were mainly responsible for the increased prevalence of COPD.

SUMMARY:
- 72 COPD patients were studied from November 2011 to May 2013.
- Among 72 patients, 39 patients were smokers and 24 patient's ECG shows p-pulmonale, 41 patients had right axis deviation, 7 had R-wave height in lead V1 more than 5 mm (indicting they had severe right ventricular hypertrophy or pulmonary hypertension).
- Out of 72 patients, 12 had wave amplitude in lead – II + lead - III aVF >9 mm, this is the indication for ife long oxygen therapy according to American Thoracic Society (ATS).
- Out of 72 patients, 12 had coronary artery disease, as this increase the incidence of corpulmonale.
- Out of 72 patients, 17 were echo-cardio-graphically had pulmonary hypertension.

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- 15 out of 72 had tricuspid regurgitation (which is non-invasive method of measurement of pulmonary artery pressure), which we cannot afford to measure via catheter.
- Out of 72 patients, 24% developed pulmonary hypertension, 22% developed tricuspid regurgitations, 34% had p-pulmonale, 18% had p-wave amplitude in lead- II + lead- III+ lead aVF >9 mm, important because it is indication for life long oxygen therapy. 18% had concomitant coronary plays enhanced role in atherosclerosis.

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17. Navaid Asad, Vanessa MP, Johnson and David H Spodick. Acute Right Atrial Strain Regression in Normal as Well as Abnormal P-Wave Amplitudes with Treatment of Obstructive Pulmonary Disease.

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