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A Study of Clinical Profile of Chronic Obstructive Pulmonary Disease and Evaluation of Cor-Pulmonale with ECG and 2d Echo Findings

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Abstract:
Background*: Chronic Obstructive Pulmonary Disease is one of the most important cause of morbidity and mortality throughout the world. It is now the 4th leading cause of death in the world and it is expected to become the 3rd leading cause of death by 2022. In 2012, COPD was the cause of death in more than 3 million people and it accounted for 6% of all the deaths worldwide. The COPD burden is expected to rise in coming years because of continuous exposure to risk factors and ageing of the population. The aim of this study is to study the clinical profile of patients and to assess right ventricular dysfunction by utilizing right ventricular parameters obtained by clinical methods, electrocardiography & echocardiography and to correlate with the severity of COPD according to GOLD guidelines.*
Materials and Methods*: A two years prospective observational study after ethical committee approval was conducted at a tertiary care hospital in Patients with COPD above 18 years of age diagnosed by symptoms & confirmed by physical, radiographic & pulmonary function tests (PFT). The data was analysed by using IBM SPSS 20.0 version software.*
Results: *Total of 75 patients with 65 males and 10 females were enrolled. Overall incidence of Corpulmonale was found in 76% by echocardiography. Evidence of Corpulmonale by echocardiography was statically significant (p<0.013). All echocardiographic findings correlated linearly with severity of disease. Corpulmonale is a common complication of COPD. Detection of Corpulmonale in early stage is important for therapeutic and prognostic implication. The severity of complications increases with severity of COPD. Thus echocardiography is a useful method for early detection of Corpulmonale and better management of cases. A more aggressive approach to treat the COPD patients can be taken so that the onset of Corpulmonale would be delayed as long as possible.*
Conclusion: *By present study it is recommended to carry out general physical examination, chest X-ray and ECG where ECHO is not available. However for prognostic implication 2D ECHO is required. Echocardiography is more sensitive and better than other modalities to diagnose cardiac comorbidities in COPD patients.*
Key Word*: COPD, Corpulmonale, ECG, 2D ECHO*

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I. Introduction

Chronic Obstructive Pulmonary Disease is one of the most important cause of morbidity and mortality throughout the world. It is now the 4th leading cause of death in the world[1] and it is expected to become the 3rd leading cause of death by 2020. In 2012, COPD was the cause of death in more than 3 million people and it accounted for 6% of all the deaths worldwide. The COPD burden is expected to rise in coming years because of continuous exposure to risk factors and ageing of thepopulation[2].COPD is very common, treatable & preventable disease. It is characterized by respiratory symptoms & airflow limitation which is due to alveolar &airway abnormalities, due to significant exposure to noxious gases or particles. Dyspnea, cough and sputum production are the most common symptoms. Important risk factor is tobacco smoking. Other environmental factors which contribute are air pollution & biomass fuel. Host factors that predispose to COPD are include abnormal lung development, genetic abnormalities and accelerated ageing

RV dysfunction is common in patients with COPD particularly in those with low oxygen saturation. It is essential to assess the extent of impairment of pulmonary function and the pulmonary arterial hypertension (PAH) caused by the same to establish the long-term prognosis of the disease. PAH affects the function of right ventricle leading to Corpulmonale and has a poor prognosis. So, the early recognition of RV dysfunction and PAH may help in treatment and prolonging the survival of the patients with Corpulmonale.

COPD is characterized by chronic airflow limitation which is due to parenchymal destruction i.e. emphysema & mixture of small airway disease i.e. obstructive bronchiolitis& the individual contributions that vary from person to person

RISK FACTORS OF COPD

• The most common risk factor encountered worldwide is tobacco smoking. This includes pipe, cigarette, cigar, water-pipe & other types of smoking as well as environmental smoke.
• Women are affected in developing countries by indoor air pollution from biomass fuel which is used for cooking & heating.
• Occupational exposures due to chemical agents, organic, inorganic dusts & fumes[3-4]
• Outdoor air pollution
• Genetic - Alpha-1 antitrypsin deficiency[5].
• Female sex & Ageing.
• Lung development & growth - factors affecting the lung growth during gestation and childhood like low birth weight and lung infections.
• Socioeconomic status-risk of developing COPD is inversely associated[6].
• Other risk factors include Asthma & childhood respiratory infections

PATHOLOGY AND PATHOGENESIS

The pathological changes are found in the airways, & also in lung parenchyma &pulmonary vasculature.

Airways - Increased numbers of goblet cells, chronic inflammation, mucus gland hyperplasia & fibrosis, narrowing,

reduction in the small airways number & airway collapse due to alveolar wall destruction in emphysema[8]. In chronic bronchitis who have Mucus hypersecretion, goblet cells number are increased & enlarged submucosal glands are seen in chronic bronchitis. Chronic inflammation in emphysema & chronic bronchitis are characterized by the presence of CD8+T-lymphocytes, neutrophils & CD68+ monocytes or macrophages in the airways[9-13].

Lung parenchyma - Emphysema also affects structures distal to terminal bronchiole, which consists of alveolar ducts, alveolar sacs, respiratory bronchiole, and alveoli known collectively as the acinus. Centrilobular emphysema means abnormal dilation or destruction of respiratory bronchiole,central portion of the acinus, which is commonly associated with cigarette smoking but also seen in coal workers' pneumoconiosis.

Panacinar emphysema refers to destruction or enlargement of all the parts of the acinus. Alpha-1 antitrypsin deficiency is most commonly associated with diffuse panacinar emphysema. The alveolar ducts are commonly affected in paraseptal emphysema. Distal acinar emphysema can occur alone or they can occur in combination with panacinar emphysema & proximal acinar emphysema. When it occurs alone, it is associated with spontaneous pneumothorax in a young adult.

Pulmonary vasculature - Includes smooth muscle hypertrophy & hyperplasia and intimal hyperplasia which is due to chronic hypoxic vasoconstriction of small pulmonary arteries[14].Oxidative stress and an excess of proteinases in the lung are likely to further modify lung inflammation. Autoantigens and perturbations in the lung microbiome may play a role. Biomarkers of oxidative stress (hydrogen peroxide, 8-isoprostane) are increased in COPD patients.

Oxidative stress is further increased during exacerbations. They are generated by cigarette smoke and other inhaled particles and released from activated inflammatory cells such as macrophages and neutrophils. There is also a reduction in endogenous antioxidants in COPD patients as a result of reduction in transcription factor Nrf2 that regulates many antioxidant genes.

There is an imbalance in the lungs of COPD patients between proteases that break down connective tissue components and antiproteases. Increased levels of several proteases, derived from inflammatory cells and epithelial cells, have been observed in COPD patients. Protease-mediated destruction of elastin, a major connective tissue component in lung parenchyma, is believed to be an important feature of emphysema. Inflammation may precede the development of fibrosis or repeated injury of the airway wall itself may lead to excessive production of muscle and fibrous tissue. This may be a contributing factor to the development of small airways limitation and eventually the obliteration that may precede the development of emphysema.

PULMONARY HYPERTENSION

Pulmonary hypertension can occur in late stages of COPD and it is due to hypoxic vasoconstriction of small pulmonary arteries, which eventually causes structural changes which includes intimal hyperplasia and smooth muscle hyperplasia & hypertrophy. An inflammatory response is seen in COPD, also with the evidence of endothelial cell dysfunction. The loss of the pulmonary capillary bed in emphysema also contributes to increased pulmonary circulation pressure. Progressive pulmonary hypertension also can cause right ventricular

hypertrophy and eventually right heart failure. Interestingly, the diameter of pulmonary artery as measured on computed tomography scans has been shown to relate to the risk of exacerbation. During exacerbations there is increased hyperinflation and gas trapping, with reduced expiratory flow, thus accounting for increased dyspnea. There is also worsening of VA/Q abnormalities that can result inhypoxemia. The release of NO, a vascular relaxing factor derived from

the intima of the pulmonary vessels, is also believed to inhibit cell proliferation in the pulmonary vessels. Hypoxemia impairs the production in and\or release of NO, thereby causing alteration in pulmonary vascular tone and blood vessel remodeling. Polycythemia due to chronic hypoxia increase blood viscosity, which may bring about a rise in Ppa. In persons with severe COPD, hyperventilation during episodes of COPD exacerbation can increase alveolar pressure, which in turn may increase Ppa.

The Right ventricle in COPD

In the adult, the RV is a thin-walled, crescent-shaped chamber. The RV has a greater ratio of volume to surface area than left ventricle and is able to generate less pressure. The RV is more a volume pump than a pressure pump and adapts better to changing preload than to increase in afterload. This generally slow progression of PAH in COPD provides the RV with time to adapt to a rising afterload. RV stroke work index and RV end diastolic pressure remain normal in most people with mild COPD, but may rise during exercise to compensate for an elevated Ppa. Although there is compelling evidence that RV contractility is well preserved in person with PAH secondary to COPD, some investigators have found a reduced right ventricular ejection fraction (RVEF). Decreased RVEF, if present, is most commonly due to a rise in RV afterload [i.e. increase in Ppa and pulmonary vascular resistance (PVR)]. In addition, RV compliance can decrease secondary to RV free wall hypertrophy and impairment of RV diastolic filling.

COR PULMONALE[17,18]

The natural history of Corpulmonale comprises of three stages:

1) Stage of primary lung disease
2) Stage of pulmonary hypertension with right
ventricular hypertrophy
3) Stage of congestive cardiac failure

Corpulmonale occurs in patients in whom there is longstanding hypoxia from disease of the lung parenchyma, airways
or vasculature. The period of hypoxia and pulmonary hypertension that precedes the onset of Corpulmonale may also
vary greatly from months to years. PAH progresses slowly, with an average increase in Ppa of 0.5 mmHg19. After some years of intermittent course the patient may die either due to respiratory or cardiac failure. However even in advanced emphysema complicated by right heart failure, vigorous treatment may results in a remarkable improvement in a patient who at first appeared to be hopelessly ill. Recent studies have shown that within that group of patients with Corpulmonale prognosis differs. Patients, who can adapt to persistent tissue hypoxia, by developing increased cardiac output, far better than those who rather cannot or those who develop polycythaemia.

DIAGNOSIS AND ASSESSMENT OF COPD

PULMONARY FUNCTION TESTS:

Spirometry: The cornerstone for the diagnostic evaluation of patients with suspected COPD is spirometry. Spirometry is performed pre and post bronchodilator administration (eg, inhalation of albuterol 400 mcg) to determine whether airflow limitation is present and whether it is partially or fully reversible. Airflow limitation that is irreversible or only partially reversible with bronchodilator is the characteristic physiologic feature of COPD. The post bronchodilator ratio of FEV1/FVC determines whether airflow limitation is present[20] .and also determines the severity of airflow limitation

Classification of severity of airflow obstruction in COPD

GOLD Stage Severity Spirometry

I - MILD FEV1/FVC<0.70, FEV1≥80% Predicted
II - MODERATE FEV1/FVC<0.70, 50%≤FEV 1<80% predicted
III - SEVERE FEV1/FVC<0.70, 30%≤FEV 1<50% predicted
IV -VERY SEVERE FEV1/FVC<0.70, FEV1<30% predicted

ELECTROCARDIOGRAM

The electrocardiographic manifestation of emphysema can be

summed up as: 27
1) Increased amplitude and / or tented P waves in leads II, III, aVF. Flat P in lead I, inverted P in aVL P axis + 60 to 90° in frontal plane but especially > + 80°.
2) Increased amplitude of T-waves in lead II, III, aVF.
3) Tendency to right axis deviation of the mean electrical axis of QRS complex.
4) Clockwise rotation in the pre-cordial leads (leftward shift of the transition zone).
5) Low voltage of the QRS especially in leads I and the left pre-cordial leads.
6) Marked respiratory variation of QRS amplitude, usually in leads V1 and V2.
7) Little respiratory variation of QRS complex in lead III

ECHOCARDIOGRAM

2D echocardiography allows improved recognition of RV chamber size and wall thickness. RV pressure overload is detected by hypertrophy of the anterior wall and by dilatation of the chamber. The other manifestation is septal hypertrophy and paradoxical septal movement i.e. the septum may appear flattened or bulged towards right. The elevation in pressure leads to increased thickness of the right ventricle with paradoxical bulging of the septum into the left ventricle during systole. At a later stage, right ventricular dilatation occurs and the septum shows abnormal diastolic flattening. However, this is eventually followed by right ventricular hypokinesis, associated with right atrial dilatation and tricuspid regurgitation. Using M-mode echocardiography, PAH can be detected by the presence of abnormal motion of the pulmonary valve (delayed opening or mid-systolic closure), or an increased ratio of the RV ejection time to the total ejection time. Doppler echocardiography is the most reliable non invasive estimation of the pulmonary artery pressure. The maximum tricuspid regurgitant jet velocity is recorded and the pulmonary artery pressure (PAP) is then calculated by the modified Bernoulli equation:PAP systolic = (4 x tricuspid jet velocity2) +RAP
RAP is the right atrial pressure estimated from the size and respiratory variation of flow in the inferior vena cava.

II. Material And Methods

This is a prospective study was carried out on patients admitted in Basaweshwara Teaching and general Hospital attached to Mahadevappa rampure medical college, Kalaburagi ,Karnataka .
A total 75 adult subjects (both male and females) of aged ≥ 18, years were for in this study.
Study Design: Prospective open label observational study
Study Location:. Basaweshwara Teaching and general Hospital attached to Mahadevappa rampure medical college, Kalaburagi ,Karnataka .
Study Duration: from November 2019- April 2021 .
Sample size: 75 patients.
Subjects & selection method:
1. A detailed case history is to be taken in all patients & meticulous examination to be done as per the proforma.
2. Routine blood investigations are to be done.
3. All patients should undergo PFT & diagnosis of COPD is made based on WHO GOLD criteria.
4. All the patients should be subjected to ECG ,Chest X-ray & ECHO for evidence of right ventricular hypertrophy, right ventricular dilatation and pulmonary hypertension. Pulmonary function tests were Carried out in all the patients with the help of spirometer:

Inclusion Criteria:
Patients with COPD above 30 years of age diagnosed by symptoms & confirmed by physical, radiographic & pulmonary function tests (PFT).

Exclusion Criteria:
1. Primary diagnosis of bronchial asthma, TB, bronchiectasis &lung malignancy.
2. Interstitial lung disease.
3. Coronary artery disease.
4. Hypertension.
5. Valvular heart disease.
6. Congenital heart disease.

Statistical Analysis

Statistical data was analyzed by IBM SPSS 20.0 version software. Collected data were spread on excel sheet and prepared master chart. Through the master chart tables and graphs were constructed. For quantitative data analysis un-paired t-test was applied. For qualitative data analysis chi-square test and Fisher exact tests were applied for statistical significance. If P-value was less than 0.05 considered as significant.

III. Result

Table No.1: Age and gender wise distribution of COPD cases

Age in years	Males		Females		Total	
	No.	%	No.	%	No.	%
41-50	9	13.9	1	10.0	10	13.3
51-60	26	40.0	2	20.0	28	37.4
61-70	27	41.5	6	60.0	33	44.0
71-80	2	3.1	1	10.0	3	4.0
81-90	1	1.5	0	0.0	1	1.3
Total	65	100.0	10	100.0	75	100.0
Mean ± SD	59.29 ± 8.53		64.60 ± 7.87		59.97 ± 8.51	
t-test value and P-value	**t = 1.780 P = 0.83 NS**					

NS= not significant, S=significant, HS=highly significant, VHS=very highly significant

The mean age of cases was 59.97 years, males mean age was 59.29 years and females mean age was 64.60 years. Male to female ratio was 6.5:1 There was no statistical significant difference of mean age of cases between males and females (P>0.05).

Multiple bar diagram represents age wise distribution of COPD cases

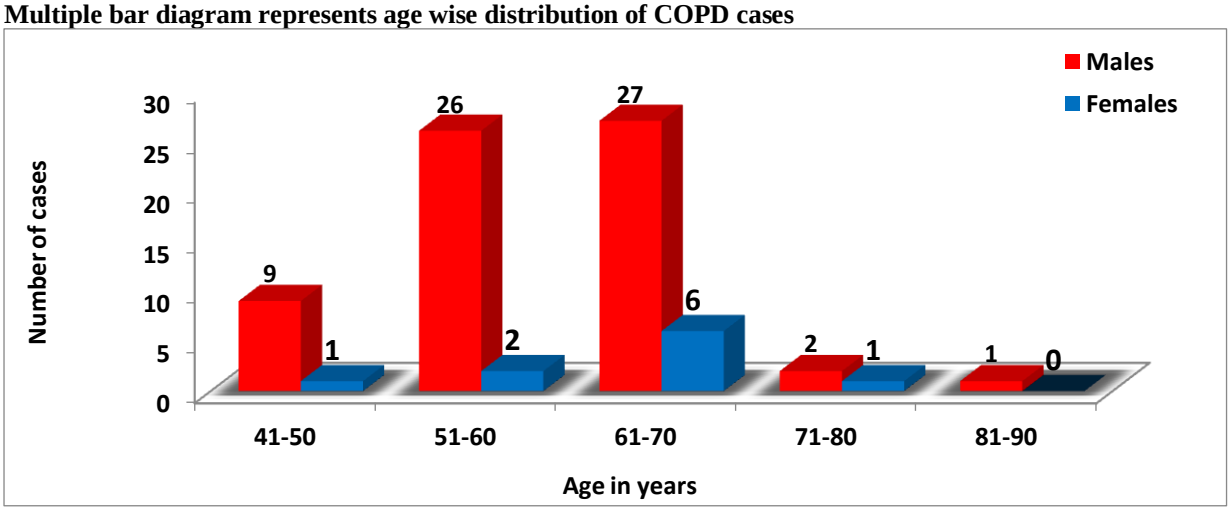


Table No.2: Gender wise distribution of COPD cases

Gender	Number of cases	Percentage
Males	65	86.7
Females	10	13.3
Total	75	100.0

In the present study, Male cases were preponderance 65 (86.7%) and female cases were 10 (13.3%). Male to Female ratio was seen 6.5:1

Pie diagram represents gender wise distribution of COPD cases

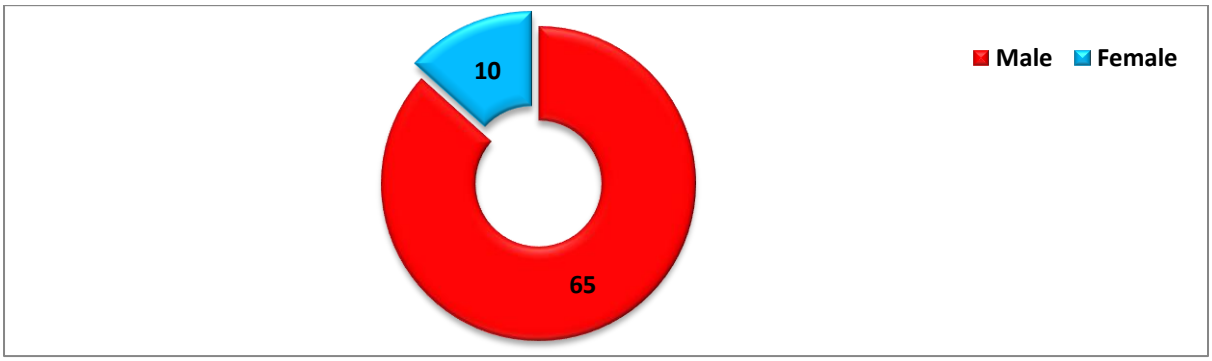


Table No.3: History of smoking wise distribution of COPD cases

History of smoking	Males		Females		Total	
	No.	%	No.	%	No.	%
Smokers	65	100.0	0	0.0	65	86.7
Non-smokers	0	0.0	10	100.0	10	13.3
Total	65	100.0	10	100.0	75	100.0
Fisher exact test	P = 0.0002 VHS					

NS= not significant, S=significant, HS=highly significant, VHS=very highly significant

Study observed that, 65 (86.7%) of cases were smokers among them 65 (100.0%) of cases were males. 10 (13.3%) of COPD cases females were non-smokers.
There was statistically very highly significant difference of history of smoking between males and females (P<0.001).

Table No.4: Duration of smoking wise distribution of COPD cases

Duration of smoking	Number of cases	Percentage
≤ 10 years	8	12.3
11—20 years	22	33.8
21—30 years	30	46.2
>30 years	5	7.7
Total	65	100.0
Mean ± SD	20.37 ± 7.79	

In the study, majority of smokers 30 (46.2%) of cases had the duration of smoking was in the interval of 21—30 years, followed by 22 (33.8%) of cases had the duration of smoking was 11—20 years. Mean duration of smoking was 20.37 years.

Simple bar diagram represents duration of smoking wise distribution of COPD cases

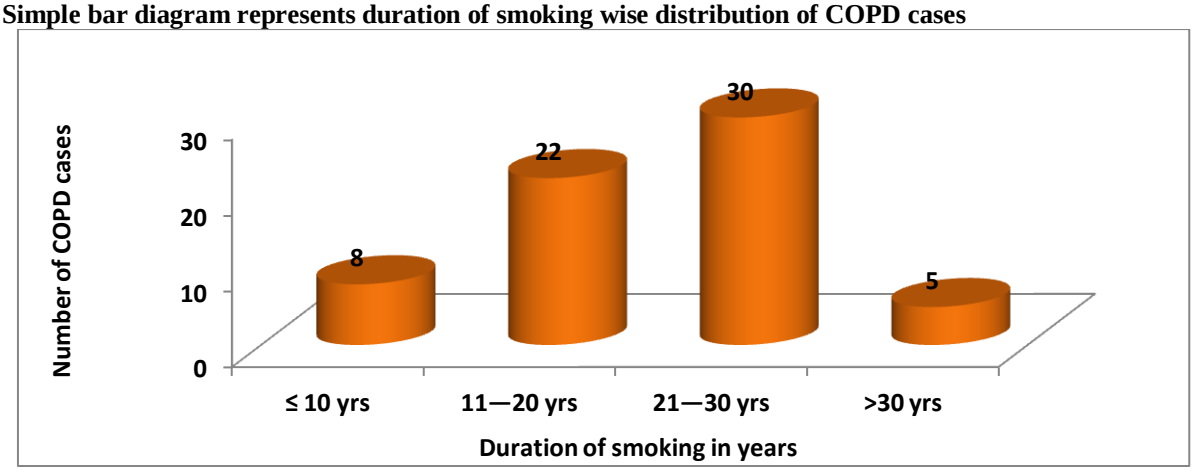


Table No.5: Distribution of COPD cases according to symptoms at presentation

Symptoms at presentation	Number of cases	Percentage
Breathlessness	73	97.3

Cough with expectoration	72	96.0
Pedal edema	33	44.0
Decreased urine output	4	5.3
Fever	3	4.0

Study observed that, the most common symptoms at presentation was breathlessness and cough with expectoration were 73 (97.3%) and 72 (96.0%) respectively.

Simple bar diagram represents symptoms at presentation wise distribution of COPD cases

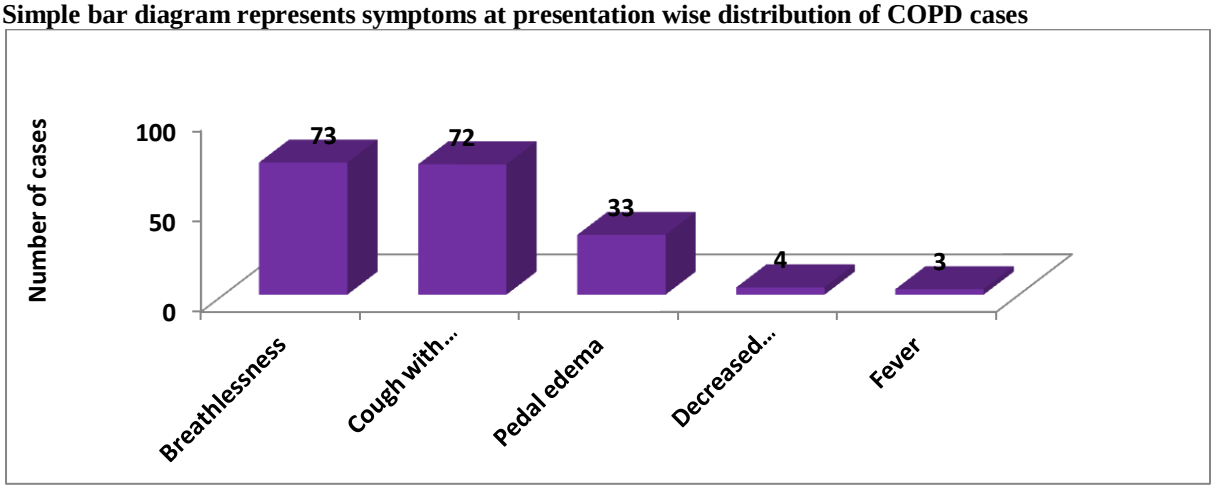


Table No.6: Duration of illness wise distribution of COPD cases

Duration of illness	Number of cases	Percentage
≤ 5 years	28	37.3
6—10 years	39	52.0
>10 years	8	10.7
Total	75	100.0
Mean ± SD	6.89 ± 3.44	

Out of 75 COPD cases 39 (52.5%) of cases had the duration of illness was in the interval of 6—10 years, followed by 28 (37.3%) of cases had the duration of illness was ≤ 5 years and 8 (10.7%) of cases had the duration of illness was >10 years. The mean duration of illness was 6.89 days

Simple bar diagram represents duration of illness wise distribution of COPD cases

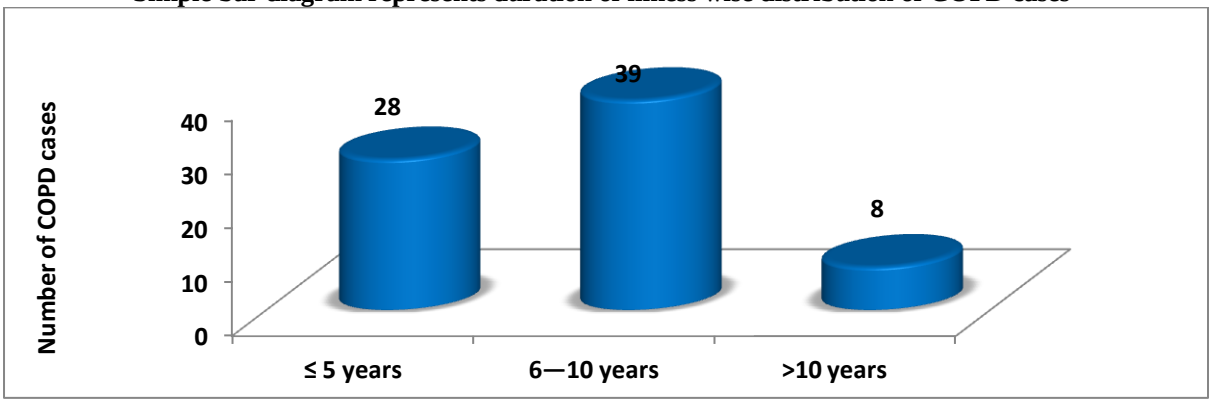


Table No.7: Distribution of COPD cases according to signs at presentation

Signs at presentation	Number of cases	Percentage
Ascites	9	12.0
Tender liver	10	13.3
PSM at TA	20	26.7
Loud p2	32	42.7
Epigastric pulsation	30	40.0
Parasternal heave	31	41.3
Crepts	44	58.7
Ronchi	61	81.3
Hyperresonant on percussion	36	48.0

Reduced chest expansion	75	100.0
Working of accessory muscles of respiration	65	86.7
Tachypnoea	74	98.7
Raised jvp	33	44.0
Pedal edema	31	41.3
Cyanosis	31	41.3

Study observed that, the most common signs at presentation were tachypnea (98.7%), use of accessory muscles of respiration(86.7),reduced chest expansion (100%), crepts (58.7%),palpabable and loud p2 in (42.7%), raised JVP in (44%),pedal edema (41.3%)

Bar diagram represents signs at presentation wise distribution of COPD cases

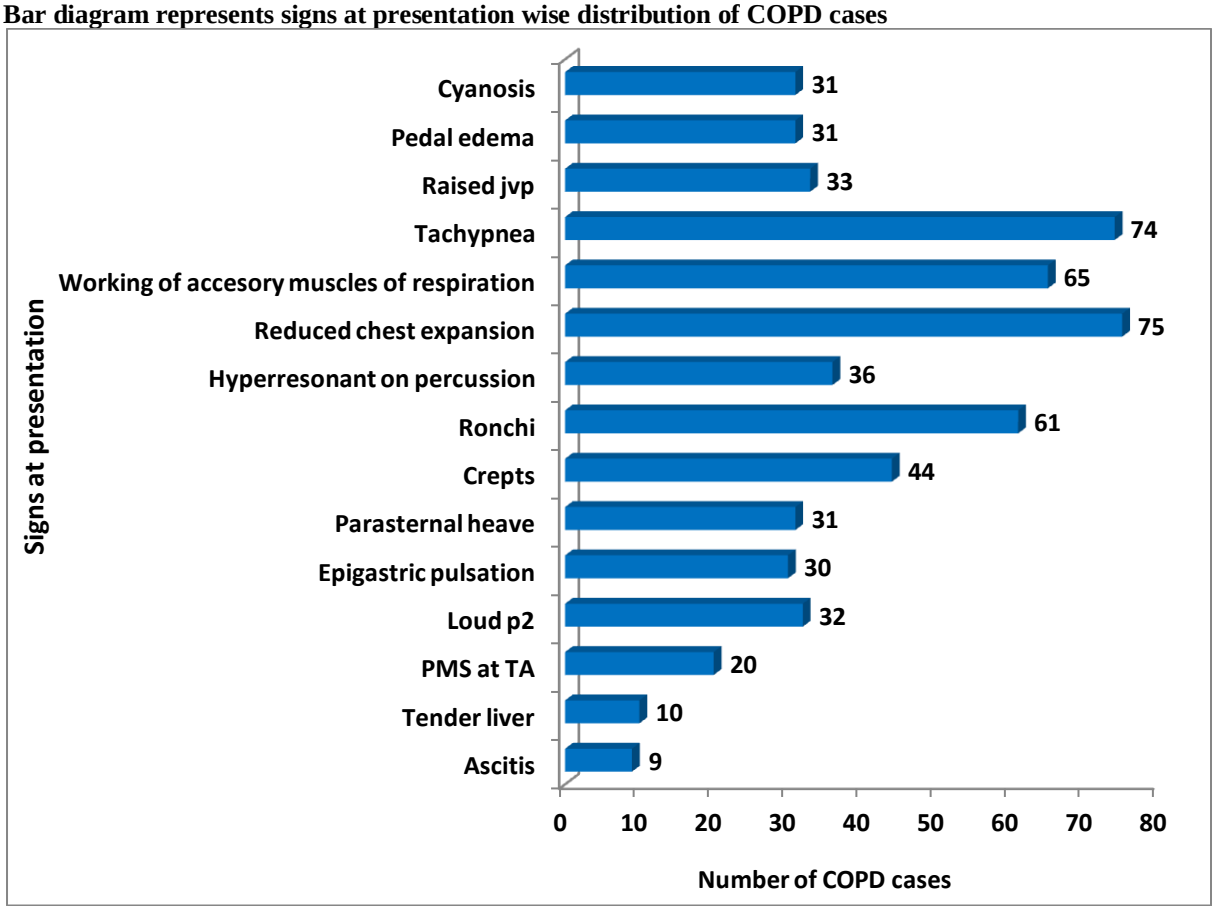


Table No.8: Pulmonary function test wise distribution of COPD cases

Pulmonary function test	Number of cases	Percentage
Mild	1	1.3
Moderate	10	13.3
Severe	34	45.4
Very severe	30	40.0
Total	75	100.0

Study observed that, majority of COPD cases 34 (45.4%) were severe pulmonary function test and 30 (40.0%) of cases were very severe pulmonary function test, 10 (13.3%) of cases were moderate pulmonary function test and 1 (1.3%) case was mild pulmonary function test.

Table No.9: Radiological findings wise distribution of COPD cases

Radiological findings	Number of cases	Percentage
Hyper inflated lungs	64	85.3
Increased bvm	41	54.7
Cardiomegaly	20	26.7
Dilated RDPA >16	32	42.7

Study observed that, 64 (85.3%) of cases had shown hyper inflated lungs in the radiological findings, 41 (54.7%) of cases had shown increase bvm and 20 (26.7%) of cases had shown cardiomegaly and 32 (42.7%) of cases had shown dilated RDPA >16

Table No.10: ECG findings wise distribution of COPD cases

ECG findings	Number of cases	Percentage
P-Pulmonale	32	42.7
IC RBBB	14	18.7
RVH	49	65.3
PPRW	14	18.7
RAD	52	69.3
Low voltage complex	24	32.0

Study observed that, 52 (69.3%) of cases had shown RAD in the ECG findings, 49 (65.3%) of cases had shown RVH, 32 (42.7%) of cases had shown P-Pulmonale and each 14 (18.7%) of cases had shown IC RBBB and PPRW respectively

Bar diagram represents ECG findings wise distribution of COPD cases

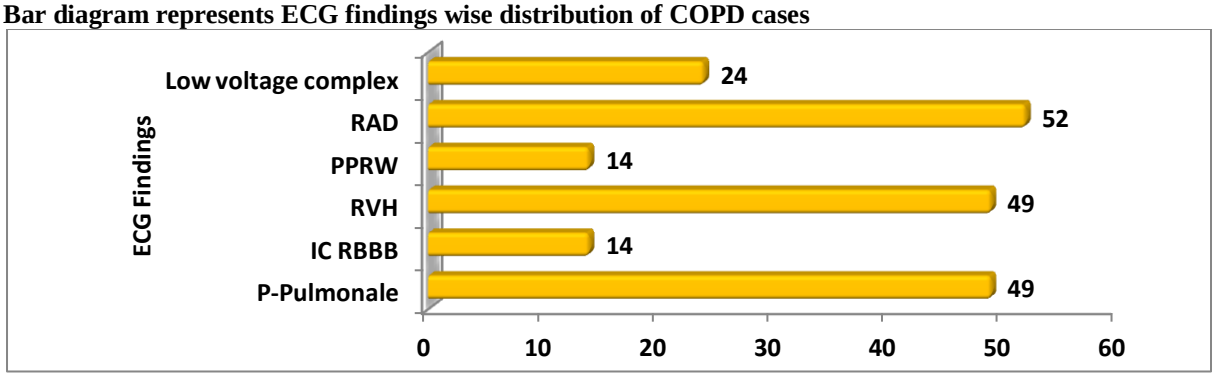


Table No.10: 2D Echo findings wise distribution of COPD cases

2D Echo findings	Number of cases	Percentage
RA/RV dilated	50	66.7
RVH	27	36.0
PAH	57	76.0
RVSD	16	21.3

57 (76.0%) of cases had shown PAH in the 2D Echo findings, 50 (66.7%) of cases had shown RA/RV dilated, 27 (36.0%) of cases had shown RVH and 16 (21.3%) of cases had shown RVSD

Simple bar diagram represents 2D Echo findings wise distribution of COPD cases

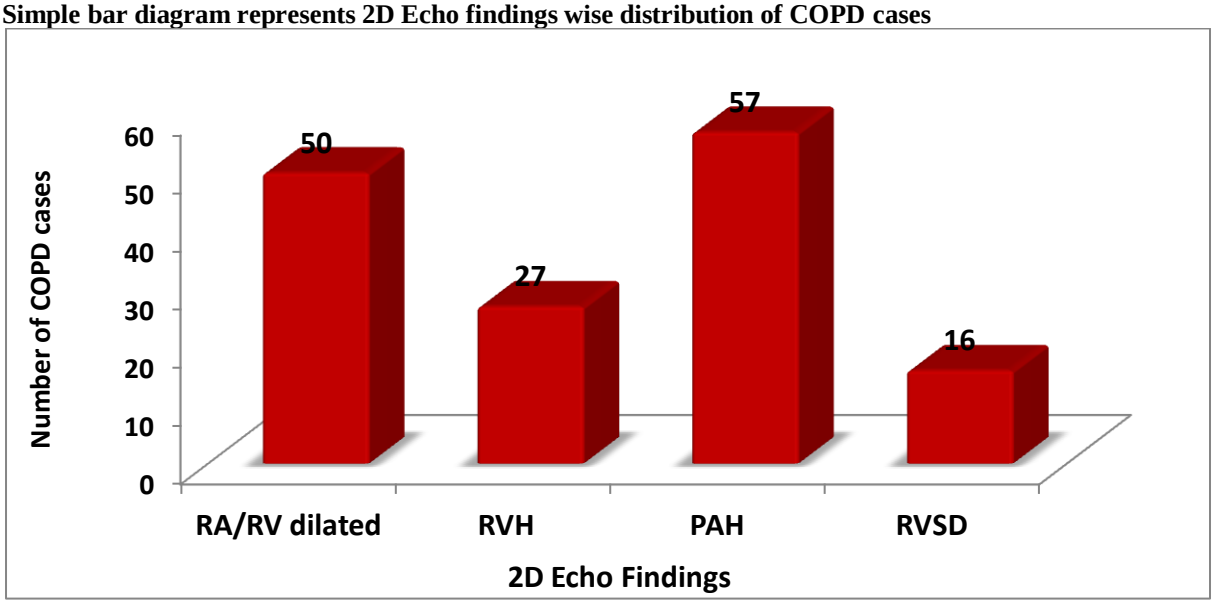


Table 11: Correlation of duration of smoking exposure and disease severity

Duration of smoking	Number of cases	Disease severity			
		Mild	Moderate	Severe	Very severe
≤ 10 years	8	0 (0.0%)	4 (66.7%)	3 (10.3%)	1 (3.3%)
11—20 years	22	0 (0.0%)	2 (33.3%)	11 (37.9%)	9 (30.0%)
21—30 years	30	0 (0.0%)	0 (0.0%)	14 (48.3%)	16 (53.3%)
>30 years	5	0 (0.0%)	0 (0.0%)	1 (3.5%)	4 (13.3%)
Total	65	0 (0.0%)	6 (9.2)	29 (44.6%)	30 (46.2%)
Fisher exact test		P = 0.0023 HS			

NS= not significant, S=significant, HS=highly significant, VHS=very highly significant

Study reveals that, There was statistically highly significant association (correlation) of duration of smoking and disease severity (P<0.01). Higher the duration of smoking cases had higher severity of disease.

Table No.12: Comparison of ECG findings and severity of COPD

ECG Findings	No. of cases	Disease severity				P-value & significant
		Mild (1)	Moderate (10)	Severe (34)	Very severe (30)	
P-Pulmonale	32	0 (0.0%)	2 (20.0%)	8 (23.5%)	22 (73.3%)	P= 0.01, HS
IC RBBB	14	0 (0.0%)	1 (10.0%)	5 (14.7%)	8 (26.7%)	P= 0.08, NS
RVH	49	0 (0.0%)	5 (50.0%)	16 (47.1%)	28 (93.3%)	P= 0.01, HS
PPRW	14	0 (0.0%)	1 (10.0%)	3 (8.82%)	10 (33.3%)	P= 0.03, S
RAD	52	0 (0.0%)	4 (40.0%)	19 (55.9%)	29 (96.7%)	P= 0.01, HS
Low voltage complex	24	0 (0.0%)	2 (20.0%)	11 (32.3%)	11 (36.7%)	P= 0.04, S

NS= not significant, S=significant, HS=highly significant, VHS=very highly significant

Study reveals that, There was statistically highly significant difference of diseases severity with ECG findings of P-Pulmonale, RVH and RAD (P<0.01). There was statistical significant difference of diseases severity with ECG findings of PPRW and Low voltage complex (P<0.05) and There was no statistical significant difference of diseases severity with ECG findings of IC RBBB (P>0.05)

Multiple bar diagram represents comparison of ECG findings and severity of COPD

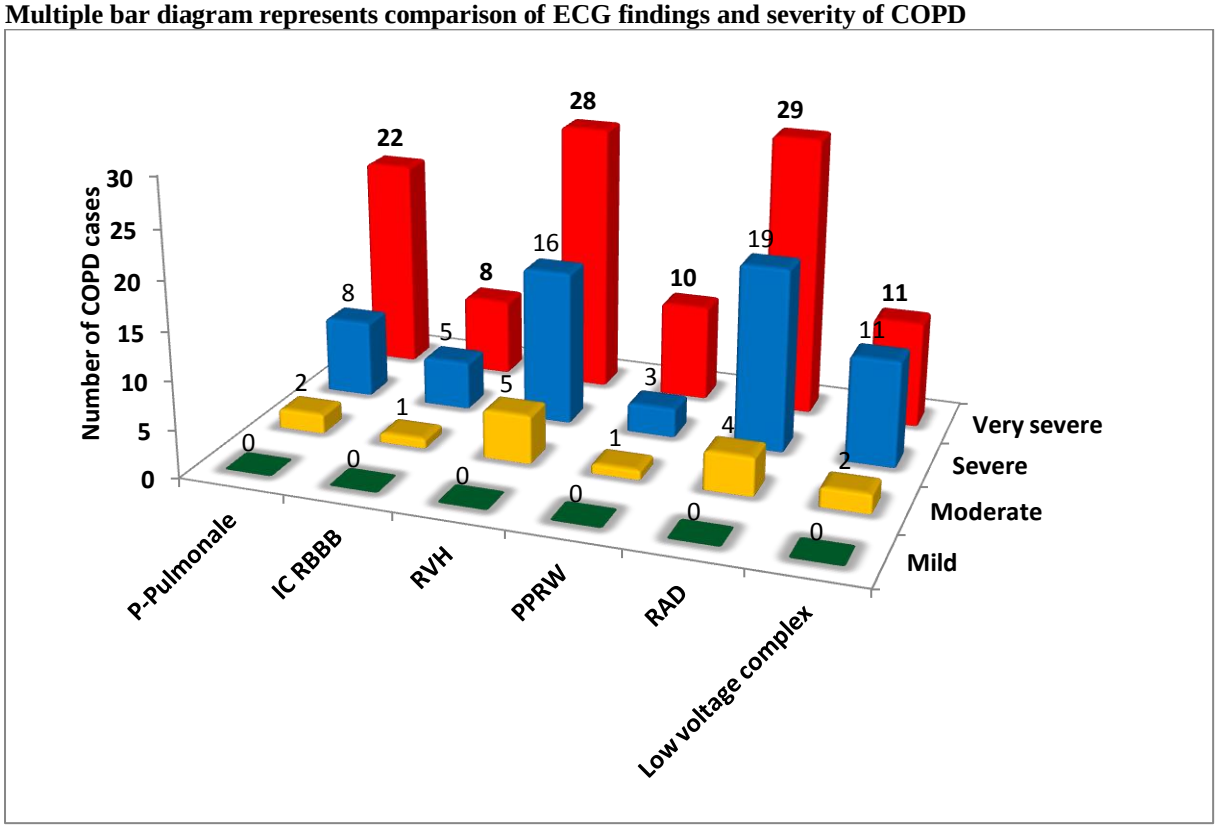
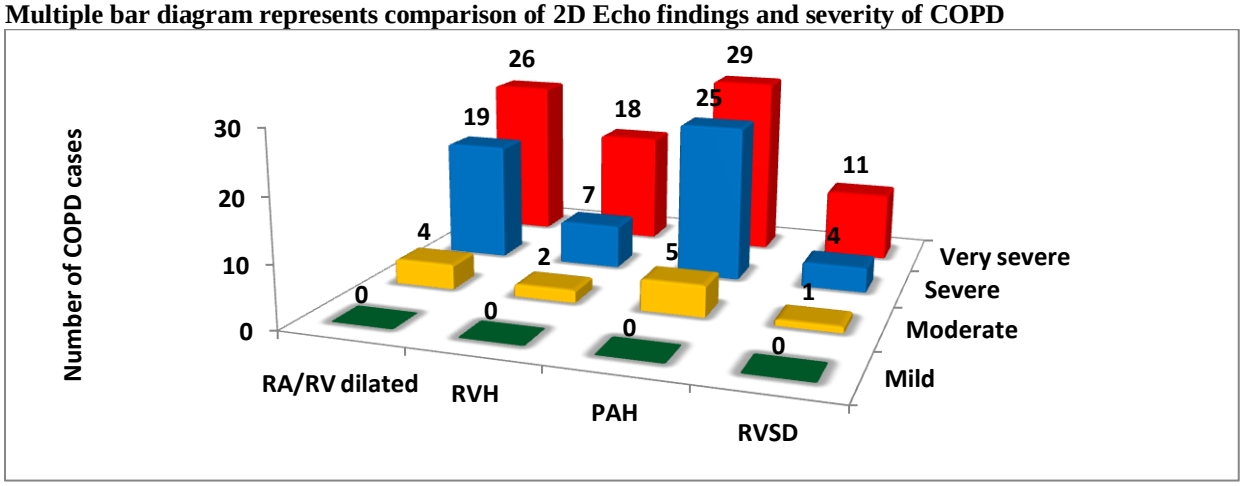


Table No.14: Comparison of 2D Echo findings and severity of COPD

2D Echo findings	No. of cases	Disease severity				P-value & significant
		Mild (1)	Moderate (10)	Severe (34)	Very severe (30)	
RA/RV dilated	50	0 (0.0%)	4 (40.0%)	19 (55.9%)	26 (90.0%)	P= 0.019, S
RVH	27	0 (0.0%)	2 (20.0%)	7 (20.6%)	18 (60.0%)	P= 0.031, S
PAH	57	0 (0.0%)	5 (50.0%)	25 (73.5%)	29 (96.7%)	P= 0.013, S
RVSD	16	0 (0.0%)	1 (10.0%)	4 (11.8%)	11 (36.6%)	P= 0.043, S

Study reveals that, There was statistical significant difference of diseases severity with 2D Echo findings of RA/RV dilated, RVH, PAH and RVSD (P<0.05)

Multiple bar diagram represents comparison of 2D Echo findings and severity of COPD



IV. Discussion

COPD is a common respiratory disease. It is more common in smokers with male to female ratio 6.14:1 and in the 5th and 6th decade. Majority of patients had a history of smoking duration between 20-29 years. The major symptoms were breathlessness and chronic cough with expectoration.

Majority of patients (74%) had severe and very severe COPD by spirometry classification. Clinical Evidence of RV failure was seen as increased JVP (33%), Parasternal heave (31%) pedal edema (31%), ascites (9%), loud and palpable P2 (32%). By chest X-ray, hyperinflated lung fields followed by increased broncho vascular markings were the most frequent changes. 65.3% of the patients had ECG evidence of right ventricular hypertrophy (RVH) and it correlate significantly with severity of disease. It can be inferred that ECG is a useful bedside test to assess the severity of COPD when spirometry is not available.

Overall incidence of Corpulmonale was found in 76% by echocardiography. Evidence of Corpulmonale by echocardiography was statically significant (p<0.013). All echocardiographic findings correlated linearly with severity of disease. Corpulmonale is a common complication of COPD. Detection of Corpulmonale in early stage is important for therapeutic and prognostic implication. The severity of complications increases with severity of COPD. Thus echocardiography is a useful method for early detection of Corpulmonale and better management of cases. A more aggressive approach to treat the COPD patients can be taken so that the onset of Corpulmonale would be delayed as long as possible.

V. Conclusion

This study conclude that diagnosis of corpulmonale in COPD patients is 36% by general physical examination, 30% by chest X-ray, 45% by ECG and 59% by 2D echocardiography. If we see the result of physical examination, CXR and ECG collectively, diagnosis of corpulmonale in COPD patients is 51% while by 2D ECHO alone is 59%. By present study it is recommended to carry out general physical examination, chest X-ray and ECG where ECHO is not available. However for prognostic implication 2D ECHO is required. Echocardiography is more sensitive and better than other modalities to diagnose cardiac comorbidities in COPD patients.

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