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Clinical study of cardiac changes in patients suffering fromchronic obstructive pulmonary disease

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Abstract

Introduction: Chronic obstructive pulmonary disease (COPD) is a global health issue with smoking being the most important risk factor. Chronic Obstructive Pulmonary Disease (COPD) is a major cause of chronic morbidity and mortality, being 4th leading cause of death worldwide and further increase in its prevalence and mortality can be expected in coming years.

Aims: To study the electrocardiographic and echo cardio graphic findings in COPD, correlate these findings with duration and severity of the disease and compare the results of clinical, electro cardio graphic and echo cardio graphic examination findings in detecting right ventricular dysfunction in COPD.

Materials and method: The present study was 50 cases were selected randomly over 1 year. The average of COPD cases admitted in Patna Medical College and Hospital, Patna over the last 1years June 2021 to June 2022.

Result: FVC (Forced vital capacity) maximal volume of air that can be exhaled during a forced Manoeuvre. FEV1Volume of air expired in the first second of maxi mal expiration after a maximal inspiration. This is a measure of how quickly the lungs can be emptied. FEV1 expressed as a percentage of FVC gives a clinically useful index of air flow limitation when itis less than 70 %. In this study most of the cases had FEV1 / FVC % in the range between 40 to 60% and most of them had moderate to severe COPD. More than half of them had

increased broncho vascular markings. Evidence of Pulmonary hypertension was found in six patients.

Conclusion: The presence of raised pulmonary artery pressure was confirmed using theDoppler Echo in 75 % of the patients showing ECG evidence of right ventricular hypertrophy.

Keywords: COPD, Right ventricular dys function, Electro cardio graphic and Echo cardio graphic examination.

Introduction

Chronic obstructive pulmonary disease (COPD) is a global health issue with smoking being the most important risk factor. There is a crude estimate of about 30 million people in India suffering from COPD, and the death rate is among the highest in the world, data suggests that about 556,000, i.e. (>20%) of total 2,748, 000 die in India annually[1]

Chronic Obstructive Pulmonary Disease (COPD) is a major cause of chronic morbidity and mortality, being 4th leading cause of death worldwide and further increase in its prevalence and mortality can be expected in coming years. This disease process is manifest by progressive airflow limitation, hyperinflation and air trapping, hypoxemia, hypercapnea, and elevations in pulmonary vascular pressures. Clinically, individuals with COPD develop breathlessness, cough, sputum pro duction and disease exacerbations that impair quality of life. Factors that portend a poor prognosis include severity of airflow limitation, ventilatory capacity, hypercapnea, and pulmonary hypertension [2,3].

COPD is characterized by slowly progressive air flow obstruction, resulting in dyspnea and exercise limitation, and pulmonary arterial hypertension is its major cardiovascular com plication. Right ventricular (RV) dys function is common in patients with COPD particularly in those with low oxygen saturation. It occurs in up to 50% of the patients with moderate to severe COPD. When present, it can reduce exercise tolerance, increase dyspnoea, and contribute to an overall decrease in functional status, and portends a higher mortality rate.Its recognition and treatment may lead to prolonged survival and improved quality of life [4,5].

Echo cardiography provides a rapid, non-invasive port able, and almost accurate method to evaluate the right ventricle function, right ventricular filling pressure, tri cuspid regurgitation, left ventricular function, and valvular functions.

It has been studied that echo cardiography measured pulmonary arterial pressure closely correlates with pressure measured by right heart catheterization [6,7].

This study was undertaken to clinical study on the cardiac changes in COPD patients with different grades of severity of the disease, as assessed clinically and through pulmonary function testing.

Materials & methods

Source of data

- Patients admitted with signs and symptoms of COPD diagnosed clinically, both male and female, over a period of one year.

Sample size

- 50 cases were selected randomly over 1 year. Samples size were calculated by taking every 5th case of the average of COPD cases admitted in PMCH, Patna over the last 1years June 2021 to June 2022.

Selection Criteria

Inclusion Criteria

- Patients with persistent respiratory symptoms and airflow limitation, who after spirometry test satisfying the GOLD criteria of COPD & falling in GOLD 2-GOLD 4 and within age group of 35-75 were included in

the study.

Exclusion criteria

- Patients with pulmonary pathology like bronchial asthma, bronchiectasis, COPD GOLD 1, tuber culosis, phenomoconiosis, restrictive lung disease like kyphoscohosis etc.
- Patients with Rheumatic, Congenital or ischaemic heart disease and hypertension.

Result and discussion

Age distribution

I used the lower age of thirty years to calculate my estimate because this was the most frequently available cut off age in the reported studies. Moreover, the disease is very rare below this age.

It is found that obstructive air way disease is more common in the middle and old age. This isbecause

- Mostly, the patients tend to ignore the initial symptoms and with increasing age, the symptoms worsen and they report to the hospital only at this juncture.
- With improving medical care, the life expectancy tends to increase and with it the problem of COPD will also increase with advancing age.

Sex distribution

In my study, the male is to female ratio is 2.12: 1 Sex distribution of COPD in adults (> 30 years of age) in different parts of the country is generally similar. Wig et al M: F was 1.32: 1, Viswanathan M: F was1.59: 1.

The prevalence was uniformly higher among male in all reported studies. The male: female ratio vary from 1.32: 1 to 2.60: 1 with the median ratio at 1.60: 1.

From the two south Indian studies which were carried out mainly in Madras region at different times, (1977 and 1995) the male female ratio was fairly constant maintaining at around 1.6: 1 which is that of the national average.

The male female ratio tends to be much higher in case of urban population. Of several possible reasons which might account for a higher prevalence among males, the most important is the habit of smoking of tobacco.

Smoking pattern

The reported smoker: non-smoker prevalence ratio ranged from 61.6 to 91.1 % in ten different population studies. The median value was around 82.3%.

In my study, the ratio is about 66% of smokers, the majority of non – smokers in my study were female population. The greater the pack years, the greater the prevalence of COPD.

Tobacco was introduced in India by the Portugese 400 years ago. Since then, tobacco consumption continued to rise in India. It has been estimated that there are 1.1 billion smokers worldwide and 182 million (16.6 %) of them live in India.

It has been predicted by WHO that more than 500 million people alive today will be killed by tobacco by 2030. Tobacco is used for smoking as well as in smokeless forms in India. Among the tobacco smokers, beedi smokers constitute 40%, cigarette smokers 20% and those using smokeless forms 40%.

The prevalence of tobacco use during 1993 to 1994 was 23.2% in male (any age) and 4% in female in urban areas, 33.6 % in male and 8.8 % in female in rural areas.

Smokers suffer an irreversible FEV1 loss of 4.4 to 10.4 ml per pack year smoked. Cigarette smoking also retards the normal increase in expiratory flow that occurs during growth in childhood or adolescence. The duration and intensity are of equal importance in determining these effects.

Smoking cessation is associated with a small improvement in lung function, decrease in coughing and sputum expectoration and normalization of the rate of annual decline in lung function.

Pulmonary function test

The primary problem in obstructive lung disease is an increased airway resistance. For each measurement of pulmonary function there is a normal value and range of normal limits.

A common method of comparison is to compute a per centage of predicted normal values according to the equation.

% predicted is equal to:

Measured value/Predicted normal value x 100

Predicted values

Predicted values vary as per age, sex, height and ethnic groups, are obtained by large scale studies in the community and are readily available for use. Values above 80 % of predicted are generally considered as normal. For patients with deformity of the thoracic cage such as Kyphoscoliosis, the arm span from fingertip to fingertip can be used as an estimate of height.

Caucasians have the largest FEV1 and FVC and of the various ethnic groups, Polynesians areamong the lowest. There is little difference in PEF between ethnic groups. The values for black Africans are 10 to 15 % lower than the Caucasians of similar age, sex and height because for a given standing height, their thorax is shorter. The Chinese have been found to have an FVC about 20 % lower and Indians about 10 % lower than matched Caucasians.

Approximate conversion factors for adjusting European reference values of FVC and FEV1 for Indians are 0.9 for North Indians and 0.87 for south Indians.

Spirometry and copd

- Spirometry is needed to make a firm diagnosis of COPD.
- Together with the presence of symptoms, spirometry helps in staging COPD severityand can be a guide to specific treatment steps.

The lower the % predicted FEV1 the worse the subsequent prognosis

- FEV1 declines over time and faster in COPD than in healthy subjects.
- Spirometry can be used to monitor disease progression, buto to be reliable, the intervalsbetween the measurements must be at least 12 months.

Staging

Once diagnosed, there are no widely accepted staging or severity scoring systems for patients with COPD. At present, we grade the disease based on a single objective physiologic measuresuch as FEV1.

FEV1 as a % of its predicted value is the best single correlate of mortality in COPD. However, it is not until values fall to < 50% of predicted that mortality begins to increase. It follows that there is a need for a more comprehensive staging system that includes age, FEV1, ABG, body mass index, time walked distance, possible bio markers and genetic markers.

Chest x-ray

The main utility of the chest x-ray lies in excluding or suggesting alternate diagnoses that could cause a patient's respiratory symptoms.

In patients with severe emphysema, the chest x-ray may reveal bilateral lung hyperinflation with flattened diaphragms or the presence of bullae, characterized by thin arcuate lines circumscribing areas of radiolucency. It is important to bear in mind, how-ever that a normal chest x-ray does not exclude the presence

of emphysema. In one study, spirometry and HRCTwere per formed on individuals with more than thirty pack years of smoking and normal chest x- rays. It was found that 58% of these individuals had evidence of significant emphysema.

In chronic bronchitis, the principal ab normalities are bronchial wall thickening and an increase in lung markings which is sometimes termed “dirty chest”, refers to a general accentuation of linear markings throughout the lungs associated with loss of definition of vascular markings. Bronchial wall thickening may be manifested as ring shadows end on or as tubular shadowed face (tram tracks).

The chest x-ray may also reveal radiographic changes of pulmonary hypertension in COPD. Patients can have right main pulmonary artery >16 mm in diameter and left main pulmonary prominence below aortic knuckle with pruning or poorly visualized peripheral vasculature. In lateral view right ventricular encroachment into the retrosternal air space can be seen.

Arterial blood gas analysis

In my study, hypoxemia was found in 22 patients and hypercapnia. The hypercapnia was found only in patients having severe or very severe COPD.

Arterial blood gases are commonly abnormal; as a rule, the more the severe the disease, the frequent the hypoxemia and hypercapnia. Arterial hypoxemia is the result of alveolar hypo ventilation and ventilation – per fusion mismatching.

In COPD of mild to moderate severity, hypoxemia exits without hypercapnia. Although the V/Q inequality impairs both the uptake of oxygen and elimination of carbon dioxide, the tendency for elevation of PaCO2 is overcome by an increase in alveolar ventilation to well perfused units. However, the increase in ventilation cannot correct the hypoxemia because of the nonlinear shape of the oxygen dissociation curve.

An increase in arterial PaCO2 does not generally occur until FEV1 is less than approximately

1.2 litres, and the pressure of hypercapnia in a patient with FEV1 > 1.5 litres should raise the possibility of central hypoventilation or obstructive sleep apnea.

Patients with COPD may experience episodic arterial desaturation during sleep, being more severe in patients categorized as blue bloaters than pink puffers. The desaturation is more during the REM sleep, that is partly due to the phasic inhibition of intercostal inspiratory muscle tone that is characteristic of REM sleep.

Approximately 20% of patients with COPD and normal awake arterial Oxygen tension have nocturnal, non-apneic oxygen desaturation. Exertional oxygen desaturation is also common. These episodes are ameliorated with supplemental oxygen.

In a recent prospective study of 43 patients with Pulmonary arterial hypertension, with normal resting oxygenation, it was found that 70% had evidence of nocturnal hypoxemia.

Electro cardio graphic findings

The ECG signs satisfying the right ventricular hyper trophy (RVH) criteria was found in 28 cases.

Diagnostic criteria for RVH for persons older than 30 years of age

- Right axis deviation > + 110o
- Tall R wave in V1 > or = 7 mm, S wave in V1 < or = 2 mm
- R/S ratio in V1 > 1, R/S ratio in V5 or V6 or = 1
- S wave V5 or V6 > 2 mm
- qR pattern in V1 (increases specificity)
- Most important is scrutiny for right atrial

enlargement, peaked P waves with anamplitude in V1, V2 or V3 > 1.5 mm or > 2.5 mm in II, III, a VF.

- More than or equal to two criteria are required for the diagnosis of RVH.

In an analysis of the contribution of individual signs in defining the long-term prognosis of COPD patients, it was found that P wave axis of more than or equal to + 90o (a sign of severe right atrial over load) and the SI, SII, SIII pattern are independent negative prognostic predictors for survival. In the patients presenting with one or both of these signs, had a three-year survival rate of 44 and 14 % respectively versus 50 and 61% for patients having other or no use ECG signs of chronic corpulmonale. The remaining signs of chronic corpulmonale like RBBB, low voltage QRS, SIQ3 pattern were less consistently associated with poor survival.

A P wave axis > or = 700 qualifies as the ECG hallmark of, and thus a screening criterion for COPD. A P wave axis more than or equal to 900 probably identifies the stage of lung hyperinflation corresponding to very severe or almost terminal illness. In the two studies assessing this sign, its prevalence was known to be 24 % and 15% respectively. Although highly specific, ECG is generally insensitive in detecting pulmonary arterial hypertension (PAH). Kilcoyn et al evaluated the ECG of 200 patients with COPD andCorpulmonale and noted at least one of the following changes:

A rightward shift of the mean QRS axis 30 degrees or more from its previous position

- Inverted biphasic or flattened T waves in precordial leads
- ST depression in II, III, AVF
- Right bundle branch block (RBBB)

In calzi et al reported that an SI, SII, SIII pattern, right atrial overload and alveolar – arterial oxygen gradient more than 48 mmhg during oxygen therapy were the strongest predictors of death.

SI, SII, SIII pattern is a relatively uncommon finding not highly specific for COPD. It reflects an abnormal wave front rightward and superiorly oriented and opposed to the electrical formsof ventricular free wall. Low voltage QRS is frequently associated with chronic corpulmonale from COPD, but not associated with corpulmonale from other pulmonary diseases.

Kok – Jensen studied the ECG of 228 patients between 40 to 69 years of age with COPD. According to him, the survival was very poor in the groups of patients with an ECG showing a QRS axis + 900 to 1800 and a PII amplitude of 0.20 mv or more. Only 37% and 42% of the patients with these changes were alive after four years. Patients with changes only in standardleads had a significantly better survival than those with changes in precordial leads as well.

Echocardiographic findings

In my study, 21 patients out of 28 who had evidence of right ventricular hypertrophy had elevated pulmonary artery pressure by Doppler echo cardio graphy (> 30 mmhg systolic or > 20 mmhg mean pulmonary artery pressure). Echo cardio graphic screening for pulmonary hypertension is based on identification of the tricuspid regurgitant jet (TR), absent in normal individuals. Measurements of TR velocity (m / sec) provides an estimate of the back flow between the right ventricle and the right atrium. The systolic Pulmonary artery pressure is estimated by the modified Bernoulli equation which is:

P=4V2 where V is the velocity of the tricuspid regurgitant jet. By adding this pressure gradient to an

estimate of the right atrial pressure, the right ventricle peak systolic pressure is determined. The right ventricle peak systolic pressure approximates pulmonary artery systolic pressure (PSAP) obtained by right heart catheterization.

Numerous studies have examined the correlation bet ween right ventricular systolic pressure (RVSP) as estimated by Doppler echocardiography and RVSP as directly measured during right heart catheterization, and most of the studies reported a relatively tight correlation (the r value ranged from 0.57 – 0.95). In a study by Hinder liter and colleagues, systolic pulmonary artery pressure was underestimated by at least 20 mmhg in 31% of patients. Other studies have demonstrated that the concordance between Doppler echocardiography and direct measurement via right heart catheterization worsens as the pressure rises, with poorer correlation when the systolic pulmonary arterial pressure is over 100 mmhg. P Sahoo and Misra et al observed that in their ECG and echocardiographic evaluation of 50 cases, ECG ab normalities were found in 24 cases. Out of these, 20 cases had echo cardio graphic evidence of raised Pulmonary artery pressure. In addition, 2-dimensional echocardiography can be used to assess RV dimensions and wall thickness. Pulmonary artery hypertension can also be assessed with pulsed Doppler echo cardiography from the sub – xiphoid region using a general-purpose ultrasound device.

Conclusion

In my study of 50 cases of COPD, the conclusions are the following

- COPD is commonly seen in persons above 40 years of age I e., in the middle and old aged people.
- It is more common among males.
- It is associated with the smoking pattern of more than 20 pack years. Its severity increases with increasing age and duration of smoking.
- ECG abnormalities suggestive of pulmonary hypertension (Right ventricular hypertrophy) was found in more than half of these cases. Poor progression with R/S is < 1 was found in 12 cases, right ventricular strain patterns like ST depression in II, III AVF and T wave inversion was found in 7 and 6 cases respectively, low voltage QRS in 5 cases. Other rare ECG abnormalities like lead I sign and SI, S II, S III was found in 2 and 1 cases respectively. Rhythm abnormalities like ventricularectopics, RBBB are found in 2 cases and multi focal atrial tachycardia in 1 case.
- The presence of raised pulmonary artery pressure was confirmed using the Doppler Echo in 75 % of the patients showing ECG evidence of right ventricular hypertrophy.

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Table 1: smoking pattern

S. No	Pack years	Predominantbronchitis	Predominantemphysema	Total	Percentage
1.	> 30	12	6	18	36
2.	20–30	8	3	11	22
3.	< 20	3	1	4	8
4.	Non smoker	12	5	17	34

Table 2: pulmonary function testfev1 /FVC %

S. No	FEV1 /FVC %	Predominantbronchitis	Predominantemphysema	Total	Percentage
1.	30–40	3	1	4	8
2.	41–50	20	4	24	48
3.	51-60	11	8	19	38
4.	61–70	1	2	3	6

Table 3: pulmonary function test Post Bronchodilator FEV1%

S. No	Post bronchodilatorFEV1%	Predominantbronchitis	Predominantemphysema	Total	Percentage
1.	Mild (> 80%)	1	2	3	6
2.	Moderate (50 – 80 %)	16	8	24	48
3.	Severe (30 – 50 %)	14	3	17	34
4.	Very severe (< 30) or (< 50% with respiratory failure)	4	2	6	12

Table 4: chest x-ray findings

S. No	CXR findings	Predominantbronchitis	Predominantemphysema	Total	Percentage
1.	Emphysematous changes	4	12	16	32
2.	Increased broncho vascular markings	24	3	27	54
3.	Cardiomegaly	4	2	6	12
4.	Evidence of pulmonary hypertension	6	-	6	12

Table 5: measurement of pulmonary artery pressure by echo-doppler

S. No	Systolic pulmonaryartery pressure (mmhg)	Predominantbronchitis	Predominantemphysema	Total	Percentage
1.	31–40	8	2	10	20
2.	41–50	6	1	7	14
3.	51–60	2	-	2	4
4.	61–70	1	-	1	2
5.	71–80	1	-	1	2