ORIGINAL RESEARCH PAPER
Volume - 10 | Issue - 03 | March - 2021 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

RIGHT SIDED HEART DISEASES CAUSED BY CHRONIC OBSTRUCTIVE PULMONARY DISEASE.

Medicine

Dr Kirti Ladukar* PG 3rd year, Department of General medicine, National Institute of Medical Sciences and Research, Jaipur, Rajasthan. *Correspondig Author

Dr Ganpat Devpura Professor and Head, Department of General Medicine, National Institute of Medical Sciences & Research, Jaipur, Rajasthan.

ABSTRACT

Introduction: Chronic obstructive pulmonary disease defined by GOLD as preventable and treatable disease with some significant extra pulmonary effects, it is a very clinical entity in clinical practice. Though C.O.P.D is basically a benign disease, the prognosis is so poor that the mortality rate is like some malignant disease. Depending on the disease severity the 5year mortality rate varies from 40-70%. In this study we will try to assess the right-side cardiac changes in C.O.P.D patients which may improve the quality of further management both in preventive and therapeutic aspect.

Methodology: This was a prospective study conducted at the OPD and IPD department of General Medicine and Chest Medicine of NIMS tertiary care teaching hospital Jaipur, Rajasthan (India). Patients with a clinical history of chronic obstructive pulmonary disease (COPD) were included in the study. A detailed clinical history, physical examination of all the participants were taken with all the investigations like ECG, 2D echo and PFT. Patients data was analyzed by SPSS v23.

Result: Total number of 100 cases were included in the study. The disease mostly affects the male population then female population of society (59% males and 34% females). Males were significantly affected (39.39%) in age groups 51 to 60 years and females were affected in 41 to 50 years of age groups. The mean age of the study population was 55.39±10.86. We got P-pulmonale in 53% of our cases. R.V.I.D(d) is significantly increased [p2x S.E(d)] in 15 patients (50%) of our study group. Every patient was having mild to moderate degree of Pulmonary Arterial Hypertension (P.A.H) compared to control group who were completely devoid of P.A.H. We have also measured Tricuspid Regurgitation gradient (47.4919.74 mm of Hg). A significant difference is noted from control group (20.2112.04 mm of Hg). Its relationship with Right ventricular diameter was not always very much linear. Right ventricular free wall thickness (R.V.F.W.T) was also increased in almost all patients.

Conclusion: Cardiac changes in C.O.P.D patients is mainly due to hypoxia resulting in pulmonary hypertension. We could not rule out the possibility of Ischaemic Heart Disease, only by history, ECG. Further studies are required to exclude these possibilities to confirm the association of R.V.H purely due to C.O.P.D.

KEYWORDS

C.O.P.D, R.V.H, 2D echo, Heart disease, ECG

INTRODUCTION

Chronic obstructive pulmonary disease defined by GOLD as preventable and treatable disease with some significant extra pulmonary effects, it is a very clinical entity in clinical practice. Chronic obstructive pulmonary disease is a leading cause of death and disability worldwide. According to World Bank data it is expected to move from its status in 2000 as the 4th and 12th most frequent cause of mortality and morbidity respectively, to the 3rd and 5th leading cause of mortality and morbidity respectively in 2020.

Though C.O.P.D is basically a benign disease, the prognosis is so poor that the mortality rate is similar to some malignant disease. Depending on the disease severity the 5year mortality rate varies from 40-70%. One study(1) reported the factors related to survival are–(a) FEV_1, (b) $PaCO_2$, (c) $Pa0_2$, (d) age, (e) Gender, (f) Body weight and Co-morbidity.

According to a previous study (unpublished) conducted in M M Medical College and Hospital in June 2002, showing total number of Indoor patients in Medicine ward in the month of June 2002 was 689. Number of C.O.P.D patients, admitted on that month was 61 (8.8%), of which male 45 (9.3% of total male admitted), and female 16 (7.8% of total female admitted). Age distribution – 31-40 years – 6 patients, 41-50 years – 8 patients, 51-60 years – 17 patients, 61-70 years– 17 patients, > 70 years – 13 patients. Total number of deaths in C.O.P.D – 17 (27.8% of C.O.P.D patients) of which Male 11 (24% of total male death), Female 6 (37.5% of total female death). Age distribution of death patients, 31-40 years – 3 patients (33% of C.O.P.D patients admitted in this age group), 41-50 years – 2 patients (37% of C.O.P.D patients admitted in this age group), 51-60 years – 4 patients (23% of patients admitted in this age group), 61-70 years – 5patients (29% of C.O.P.D patients admitted in this age group), more than 70 years – 3 patients (23% of C.O.P.D patients admitted in this age group). Number of total deaths in admitted patients 147 (21% of admitted), of which number of male deaths 93 (19% of total male admitted), number of female deaths 54 (26% of total female admitted). It is noted that C.O.P.D patient has significant higher number of admission and death (both in female and male but more in female) than average hospital death and ranked 2nd only after patients of Cerebra Vascular Accident (C.V.A)(2).

In other study suggests that socio-economic factors operating from early in life affect the adult risk of developing C.O.P.D independently of smoking in both males and females. Applying mechanical ventilation to C.O.P.D patients treated with long term 0_2 therapy carries a high mortality and cost. On the other hand early identification and intervention via smoking cessation and possibly by augmentation of antioxidant defense's in the lung offer hope to prevent and forestall premature morbidity and mortality(3).

It is also observed that a large portion of these patient's morbidity and mortality is related to the changes in cardiac function.

When chronic hypoxemia develops the consequent Pulmonary Artery Hypertension (P.A.H) is associated with Right Ventricular Hypertrophy (R.V.H). Prevalence of R.V.H in a large European study of patients with lung disease was 8.9% and increased as airflow limitation worsened in patient with C.O.P.D being present in 70% when $F.E.V_1$ had fallen to 0.6 L. Almost ½ of all patients who died due to cor-pulmonale in cases of C.O.P.D also have Left Ventricular Hypertrophy (L.V.H) on post-mortem examination.(4)

In this study we will try to assess the right cardiac changes in C.O.P.D patients which may improve the quality of further management both in preventive and therapeutic aspect.

Study Methodology

Study Design

This study was conducted at the OPD and IPD department of General Medicine and Chest Medicine at NIMS tertiary care teaching hospital, Jaipur, Rajasthan, India.

Selection of patients

Inclusion criteria

Variable -

A. History: - Patients with a positive history of chronic progressive symptoms including wheeze with breathlessness or cough with or without production of mucoid sputum for 3 consecutive months at least for more than 2 years, long lasting significant smoking history, working in a heavily polluted environment, history of peripheral

oedema or other features of Right heart failure.

B. Examination: - Presence of wheeze, Ronchi, coarse crepitations during exacerbation, features of hyperinflation of lung i.e., hyper-resonant percussion note, obliteration of cardiac and hepatic dullness, decreased breath sound, epigastric pulsation, parasternal heave, palpable P_2, diffuse Right ventricular type of apex.

C. Radiological features: - Features of hyperinflation – i.e., increased translucency, flattening of diaphragm, decreased peripheral vascularity, enlarged Pulmonary artery and its branches with peripheral pruning, cardiac enlargement.

a. Electrocardiographic evaluation: - P-pulmonale, Right axis deviation, clockwise rotation, low voltage complex.

b. Constant

c. Pulmonary function test: - $F.E.V_1$ less than 80% of predicted and $F.E.V_1$/F.V.C less than 70% predicted (except in some cases where some restrictive changes are present, we may get FEV_1/FVC ratio normal or increased).

d. Echocardiography: - Definite enlargement of Right ventricle with or without hypertrophy and consequent enlargement of right atrium.

II. **Exclusion criteria**

A. These are based on factors that are known to produce changes in any portion of heart anatomically or functionally. Conventionally exclusion should be started from the very beginning i.e. history taking, clinical examination and various investigations.
B. History suggestive of valvular heart disease, congenital heart disease or coronary artery disease i.e., Angina pectoris, old myocardial infarction, paroxysmal nocturnal dyspnoea, history of rheumatic fever and its sequele and any evidence of the above-mentioned diseases on examination.
C. History suggestive of pericardial heart disease – presentation with swelling of abdomen, pain, or heaviness in chest and suggestive in clinical examination.
D. Excluding the other systemic disease affecting cardiac and pulmonary functions – i.e., hypertension, diabetes mellitus, hypothyroidism, chronic renal failure, hyper or hypoadrenalism, dyslipidaemias.
E. History suggestive of connective tissue diseases – arthritis, oral ulcer and any suggestion on clinical examination.
F. History of addiction to alcohol or any other drugs that can independently cause cardiac changes or via pulmonary changes by it.
G. A number of specific causes of chronic airway obstruction are not included in the term of C.O.P.D i.e. cystic fibrosis, bronchiectasis, bronchiolitis obliterans, suggested on history, clinical examination or radiology and are excluded from our study.
H. ECG evidence of ischaemia.
I. Echocardiographic evidence of regional ventricular wall motion abnormality, akinesia, hypokinesia, dyskinesia or evidence of cardiomyopathy.

Selection of control

This difficult job done from all patients attending O.P.D or Indoor of General Medicine and Chest Medicine Department of NIMS hospital, JAIPUR.

Having no history of smoking, bronchial asthma, atopy, occupational exposure, no clinical symptoms, and signs of C.O.P.D and without any change suggestive of C.O.P.D in chest X-ray, pulmonary function test. We have matched age, sex and socio-economic status of control and study groups.

METHODS: -

All patients under our study went through –

a. Clinical examination including history taking.
b. Chest X-ray Postero Anterior and Lateral view.
c. Pulmonary function test.
d. Resting 12 leads ECG examination with long lead II taken in deep inspiration and expiration.
e. Echocardiographic examination with 2-Dimensional and 'M' mode study. Echo- Doppler study.

Statistics-

Data collected from patients was entered and analysed through SPSS v23. The distribution of parametric data variables was presented as mean and SD. The standard error of difference was presented as SE. Independent student t-test was used to compare the means of the parametric continuous variables.

RESULT

Total number of 100 cases were included in the study from the outpatient and indoor department of general medicine and respiratory department of NIMS hospital Rajasthan Jaipur. All the Cases were selected according to the Inclusion and exclusion criteria as mentioned previously.

In C.O.P.D patients there are several ECG changes and ST-T changes and abnormal appearance of q waves mimicking old infarction or I.H.D and for that the following criteria for I.H.D in presence of C.O.P.D will help to differentiate between the two.

One patients of our study group have definite P-pulmonale and other features of chronic cor-pulmonale, but have some of the features of coronary heart disease (i.e. Q in lead I>0.04 sec., qR in lead aVL with T inversion, T inversion in lead V_6, T inversion in lead I). This patient was screened extensively with normal resting echocardiography and some regional wall motion abnormality was seen. So, he was excluded from our study group.

Table 1:- ECG findings of the cases

SL. NO.	ECG FINDINGS	NUMBER OF PATIENTS	PERCENTAGE
1.	**P-PULMONALE**	53	53%
2.	RIGHT AXIS DEVIATION	33	33%
3.	CLOCKWISE ROTATION OR PERSISTENT S WAVE IN LEAD V_6	40	40%
4.	qR IN LEAD aVR	13	13%
5.	R/S RATIO >1 IN LEAD V_1	33	33%
6.	rSR, qR, Rsr PATTERN IN LEAD V_1	7	7%
7.	R.V.H	33	33%
8.	LOW VOLTAGE COMPLEX	20	20%
9.	ST-T WAVE CHANGES IN RIGHT PRECORDIAL LEADS	16	16%
10.	ARRYTHMIA	0	0
11.	L.V.H	3	3%
12.	NORMAL	13	13%

We got P-pulmonale in 53% of our cases. It indicates that it is not very much sensitive in C.O.P.D patients though most common. Low voltage complex seen in 20% patients, due to hyperinflation. One patient in our study group having features of L.V.H. 2 patients of our study showed rSR□ pattern in lead V1. 4 patients 13% of our study group showed ECG within normal limit (table 1).

Table 2:- D-Echo finding of the cases

Echocardiographic findings in study and control group –

SL. NO.	ECHOCARDIOGRAPHIC FINDINGS	RESULTS	
		STUDY GROUP (n=30)	CONTROL (n=20)
1.	RIGHT VENTRICULAR FUNCTION –		
	a) R.V.I.D (d) cm/B.S.A	2.001±0.66 [SD-0.33]	0.94±0.481 [SD-0.24]
	b) R.V.F.W.T cm/B.S.A	1.180±0.26 [SD-0.131]	0.86±0.28 [SD-0.14]
	c) I.V.S (s) cm/B.S.A	1.2±0.28 [SD-0.14]	0.91±0.32 [SD-0.16]
	d) IVS (d) cm/B.S.A	1.0±0.48 [SD-0.24]	0.86±0.22 [SD-0.11]
	e) Pulmonary Artery pressure gradient during diastole	+	–
	f) Tricuspid Regurgitation gradient	47.49±19.74 mm Hg [SD-9.87]	20.21±12.04 mm Hg [SD-6.02]
2.	LEFT VENTRICULAR FUNCTION -		
	a) Systolic Function		
	i) Ejection Fraction (EF%)	58.75±14.20 [SD-7.10]	56.4±16.434 [SD-8.217]

	ii) Fractional shortening (FS%)	32.16±12.96 [SD-6.48]	34.66±11.70 [SD-5.85]

SL. NO.	ECHOCARDIOGRAPHIC FINDINGS	RESULTS	
		STUDY GROUP (n=30)	CONTROL (n=20)
	b) Diastolic Function		
	i) E-peak velocity cm/sec	59.86±10.56 [SD-5.28]	69.25±17.52 [SD-8.76]
	ii) A-peak velocity cm/sec	65.26±16.30 [SD-8.15]	52.75±14.08 [SD-7.04]
	iii) E/A ratio	0.896±0.20 [SD-0.10]	1.32±0.22 [SD-0.11]
	c) Left Ventricular cavity dimension		
	i) L.V.I.D (d) cm/B.S.A	4.13±1.58 [SD-0.79]	3.7±1.0 [SD-0.5]
	ii) L.V.I.D (s) cm/B.S.A	2.81±1.56 [SD-0.78]	2.3±0.62 [SD-0.31]
	iii) L.V.P.W.T (s) cm/B.S.A	1.186±0.22 [SD-011]	0.96±0.42 [SD-0.21]
	iv) L.V.P.W.T (d) cm/B.S.A	1.01±0.30 [SD-0.15]	0.93±0.42 [SD-0.21]
	v) LV Mass g/m^2	106.84±60 [SD-30]	74.82±61.92 [SD-30.96]

Averages of 2-Dimensional, M-mode and Doppler echocardiographic values are tabulated in table XVI and compared with values obtained from normal individuals maintaining more or less similar age and sex distribution without any cardiac disease or symptoms, who volunteered spontaneously. It was seen that R.V.I.D(d) is significantly increased [p2x S.E(d)] in 15 patients (50%) of our study group. Every patients was having mild to moderate degree of Pulmonary Arterial Hypertension (P.A.H) compared to control group who were completely devoid of P.A.H. We have also measured Tricuspid Regurgitation gradient (47.4919.74 mm of Hg). A significant difference is noted from control group (20.2112.04 mm of Hg). Its relation with Right ventricular diameter was not always very much linear. Right ventricular free wall thickness (R.V.F.W.T) was also increased in almost all patients. The mean R.V.F.W.T was 1.1800.26 cm in study group and 0.860.28 cm in control group [P1.1 cm. At the same time calculated Left Ventricular Mass by using Deverenx regression equation and body surface area, Left Ventricular Mass should be >135 gm/m2 in male and 111 gm/m2 in female to establish them as Left Ventricular hypertrophy (L.V.H). In our study 5 (16%) patients had L.V.H. All these 5 patients had increase in both I.V.S and L.V.P.W.D. There were also a number of cases having increased I.V.S and L.V.P.W.D but do not exceed the limit of L.V.H through Deverenx regression formula. Regarding Systolic Function of Left Ventricle we measured Ejection Fraction (E.F) and Fractional Shortening (F.S). Mean Ejection Fraction (58.7514.20) was slightly elevated than control (56.416.43) and the difference was not statistically significant. [S.E(d) (2.903) and Mean difference (2.35) which is less than twice of S.E(d), so P not less than 0.05]. Earlier study of different investigator by Radionuclide Ventriculography, also have shown a normal to supernormal Ejection Fraction in chronic cor-pulmonale patient due to C.O.P.D . An impairment of Left Ventricular Diastolic Function was noted in our study. By using Doppler method we can accurately measure E and A velocity around Mitral valve. Most of them showed decrease in E velocity and the mean E velocity (59.8610.56) was significantly decreased from control (69.2517.52) value. A velocity was also noted to be increased (65.2616.30) significantly than control (52.7514.08) value. E/A ratio was less than 1 in most cases (excepting 3 patients, though reduced to a great extend). In the study group E/A ratio (0.8960.20) also significantly lowered than control (1.320.22) value. We also examined the status of different valves to exclude abnormal shunt or valvular heart disease.

Pulmonary function test (PFT) is the major tool for diagnosis of C.O.P.D patients. In early stages of the disease, this test may be normal, but the history and chest x-ray may suggest the presence of the disease (Table 3)

Table 3:- pulmonary function test finding of the cases

Average values of different parameters in Pulmonary Function Test (P.F.T)

SL. NO.	PARAMETERS	RESULTS OBTAINED IN STUDY GROUP (n=30)	RESULTS OBTAINED IN CONTROL GROUP (n=20)
1.	F.E.V_1 IN MEN	1.217±0.78 L/S [SD-0.39]	2.52±0.16 L/S [SD-0.08]
2.	F.E.V_1 IN WOMEN	1.107±0.66 L/S [SD-0.33]	2.00±0.11 L/S [SD-0.055]
3.	F.V.C IN MEN	1.88±0.72 L [SD-0.36]	3.56±0.54 L [SD-0.27]
4.	F.V.C IN WOMEN	1.514±0.50 L [SD-0.25]	2.60±0.24 L [SD-0.12]
5.	F.E.V_1/F.V.C IN MEN	65.98±29.26% [SD-14.63]	74.02±8.01% [SD-4.0]
6.	F.E.V_1/F.V.C IN WOMEN	69.9±34.24% [SD-17.12]	79.03±6.07% [SD-3.03]

Patients are divided according to type of lung function

SL. NO.	LUNG FUNCTION INFERENCE	NUMBER OF PATIENTS	PERCENTAGE
1.	OBSTRUCTIVE	21	66%
2.	OBSTRUCTIVE+RESTRICTIVE	9	33%

Both obstructive and combined (obstructive + restrictive) type of lung disease have been found in our study.

We have found a significant reduction of F.E.V1, F.V.C, F.E.V1/F.V.C ratio, P.E.F.R and V.C in our study group than control. F.E.V1 in men 1.217 0.16 L/S [P<0.05, as SE(d) 0.112 and mean difference 1.30 which is more than twice of SE(d)]. F.E.V1 in women 1.1070.66 L/S and control 2.000.11 L/S, [P<0.05, as SE (d) 0.069 and Mean difference 0.90]. F.V.C in men 1.880.71 L and control 3.560.52 L [P<0.05, as SE(d) 0.214 and mean difference 1.68]. F.V.C in women 1.5140.50 L and control 2.600.24 L [P<0.05, as SE (d) 0.1185 and Mean difference 1.086]. F.E.V1/F.V.C in men 65.9814.63% and control 74.028.01% [P<0.05, as SE(d) 3.68 and Mean difference 8.04]. F.E.V1/F.V.C in women 69.617.12% and control 79.036.07% [P<0.05, as SE(d) 3.66 and Mean difference 9.13].

9(~ 30%) of our patients (3 Female and 6 Male) showed restrictive pattern along with predominant obstructive pattern of pulmonary function test. Though we are not able to show definite relationship of severity or type of PFT with the severity of cardiac changes.

Still, we can safely say that restricted pattern usually seen in patients with prolonged disease process having moderate to severe P.A.H or Tricuspid Regurgitation gradient. It is mainly due to fibrosis by repeated infections, plural thickening, and decreased excursion of diaphragm due to flattering and fatiguability.

DISCUSSION

The classical view of the development of cardiac changes in patients with C.O.P.D is that, hypoxia, mainly by inducing vasoconstriction, structural changes of pulmonary vessels, leads to Pulmonary Arterial Hypertension (P.A.H). This P.A.H imposes increased work (after load) on the Right ventricle, leading to Right Ventricular Hypertrophy (R.V.H) and eventually its dilatation. The severity of P.A.H, the rapidity with which it becomes severe and the possible eventual onset of Right ventricular Failure (R.V.F) are influenced by – alteration of ventilatory function causing alveolar pressure changes, alternation of gas exchange with more or less severe hypoxemia, hypercapnoea and acidosis, alternation in volume load. At some stage myocardium is unable to function against this high pressure load, dilates and fails. It is seen that R.V.F usually occur relatively early in C.O.P.D patients compared to Interstitial Lung Disease (I.L.D) patient probably because of sustained hypoxemia and hypercapnoea.

Right ventricular changes in C.O.P.D patients is a sine qua non of co1r–pulmonale. It is already mentioned that recurrent or persistent hypoxia resulting in P.A.H and is leading to R.V.H and ultimately Right Ventricular dilatation by imposing increased work (after load) on

Right Ventricle. The degree of R.V.H varies greatly and closely correlates with the progression of underlying disease process. But one study of non invasive assessment of cardiac function in patients with C.O.P.D by Bagnato G.F et al(5) suggested that early involvement of Right Heart have been observed even if they had clinical stable condition and no Pulmonary hypertension.

We got cardiac enlargement in 6 (20%) of our patients; all of them were due to Right ventricular enlargement as evidenced from Chest X-ray lateral view. We could not get Left ventricular enlargement in any of our patients probably due to increased lung volume in most of our patients. This finding is consistent with earlier studies of Murphy and Adamson and Hutcheson(6).

In our study we found P- pulmonale (53%), Right axis deviation (33%), R.V.H (33%), clockwise rotation and persistent s wave in V_6 in (40%) of our patients. All these features suggest Right ventricular predominance. It is also to be mentioned that 4(13%) patients of our study group showed normal ECG. These findings are consistent with a study(7) where they have concluded that C.O.P.D can be diagnosed by ECG in 78.18% of cases and 95 % by Echocardiography.

Echocardiography is the investigation of choice among the non-invasive procedures to detect cardiac (anatomical and functional) changes. It can be assessed by-

Right Ventricular Internal Diameter in Diastole [R.V.I.D(d)] - In our study we have got a significant increase in Right Ventricular Internal Diameter (2.0010.66) than control (0.940.48) group [P<0.05, determined by detecting Standard Error of difference, S.E (d)]. We got increase in [R.V.I.D(d)] i.e. Right ventricular enlargement in all our study group.

Right Ventricular Free Wall Thickness [R.V.F.W.T] - The mean R.V.F.W.T (1.180 2.62 cm) in our study group is significantly [P< 0.05, determined by S.E(d)] increased in 66% patients of our study group. It indicates R.V.H (i.e. thickness> 1.1cm).

Inter Ventricular Septum [I.V.S] - We have significantly increased [P<0.05 detected by determining S.E(d)] I.V.S thickness both in systole (1.2 .28) and diastole (1 0.48) in our study group than control [I.V.S (s) 0.91 0.16, I.V.S(d) 0.860.14]. 15 out of 30 (50%) of patients have increased thickness of I.V.S. Other 50% of patient did not show any change which indicates that Right ventricular free wall hypertrophy may occurs much more earlier than I.V.S.

Another important finding is abnormal **Septal Shape and Motion**. End diastolic measurement of septal radius and curvature showed flattening of IVS in all our patient due to diastolic Right ventricular volume overload. This accounts, in part for abnormal septal motion observed in patients with Right ventricular volume overload. And interesting study by Takakura M et al, in Japan(8) in February 1999, in 25 C.O.P.D patients with no significant difference at the Right ventricular end diastolic pressure at rest, from control group, have shown that the pressure exceeded that of Left ventricular pressure in early diastole, coinciding with marked downward ventricular septal motion in early diastole during exercise. They concluded that occult cor-pulmonale can be diagnosed accurately by the appearance of this abnormal septal motion and distorted Left ventricular shape during exercise in C.O.P.D patients. Recently this leftward shift was evidenced to result from a decreased trans- septal pressure gradient and did not require Right Ventricular pressure to exceed Left ventricular diastolic pressure[10]. In our present study 2–Dimensional echocardiography also showed leftward shift of septum and flattening of I.V.S at end systole. It may be speculated that septal shape during systole might well depend in part on respective Left and Right free wall systolic tension, which are determined according to Laplace formula, by the product of the intra vascular pressure by the radius of curvature of free wall. These are abnormally elevated in the Right Heart characteristics of cor-pulmonary. According to Guzman and associates(9), Right ventricular loading could alter the local balance process of the Right ventricular septal junction.

Pulmonary artery pressure gradient – All of our study group had increase in P.A.P and differs from controlled group which did not have increased P.A.P.

Tricuspid Regurgitation gradient - Normally the gradient across Tricuspid valve is 20 mm of Hg. P.A.H resulting in increased after load in right ventricle causes increase in the gradient. We have found increased value in all of our patients, which varies, from mild moderate to severe.

CONCLUSION

Cardiac changes in C.O.P.D patients is mainly due to hypoxia resulting in pulmonary hypertension leading to hypertrophy and enlargement of Right Ventricle with accompanying sign, and symptoms. Pulmonary hypertension is present in all of our patients along with significantly increased Tricuspid Regurgitation gradient. We also found to the effect of chronic cor-pulmonale due to C.O.P.D. on the Left Ventricular function.

We could not rule out the possibility of Ischaemic Heart Disease, only by history, ECG and Echocardiography on rest as we were not supposed to go through Tread Meal Test study, coronary angiography or autopsy study. It is also not possible to rule out the possibility of early hypertrophic cardiomyopathy as there is no generally accepted marker. Further studies are thus necessary to exclude these possibilities to confirm the association of R.V.H purely due to C.O.P.D.

REFERENCES

1. Nishimura K, Tsukino M. Clinical course and prognosis of patients with chronic obstructive pulmonary disease. Current Opinion in Pulmonary Medicine. 2000.
2. Prescott E, Lange P, Vestbo J. Socioeconomic status, lung function and admission to hospital for COPD: Results from the Copenhagen City Heart Study. Eur Respir J. 1999;
3. Petty TL. Definitions, causes, course, and prognosis of chronic obstructive pulmonary disease. Respiratory care clinics of North America. 1998.
4. Eugene Braunwald. Harraison's Principles of Internal Medicine. 15th ed. 2001. 1358 p.
5. Bagnato GF, Mileto A, Gulli S, Piscioneri S, Romano C, Giacobbe O, et al. Non invasive assessment of cardiac function in patients with bronchial asthma (BA) or chronic obstructive pulmonary disease (COPD). Allergol Immunopathol (Madr). 1999;
6. Murphy ML, Boger J, Adamson JS, Rubin S. Evaluation of cardiac size in chronic bronchitis and pulmonary emphysema. Chest. 1977;
7. Putnik M, Povazan D, Vindis-Jesić M. Electrocardiography and echocardiography in the diagnosis of chronic cor pulmonale. Med Pregl. 1998;
8. Takakura M, Harada T, Fukuno H, Okushi H, Taniguchi T, Sawada S, et al. Echocardiographic detection of occult cor pulmonale during exercise in patients with chronic obstructive pulmonary disease. Echocardiography. 1999;
9. Guzman PA, Maughan WL, Yin FCP, Eaton LW, Brinker JA, Weisfeldt ML, et al. Transseptal pressure gradient with leftward septal displacement during the Mueller manoeuvre in man. Br Heart J. 1981;