Left ventricular assessment in chronic obstructive pulmonary disease

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Left ventricular function has been assessed by measuring the left ventricular hypertrophy and traction shortening through calculation of the LVIDd, LVIDs, IVST and LVPWT during diastole by eachocardiography in (26) patients with severe COPD (group II), five patients with severe COPD and known primary left ventricular disease (group III), (14) healthy age matched subjects were examined as control (group I).

There is statistically significant left ventricular hypertrophy in patients with severe COPD (group II) and patients with severe COPD and known primary left ventricular disease (group III) compared with the control (group I).

While there is apparent reduction in fraction shortening percent in patients with COPD which is not significant there is statistically significant reduction in fraction shortening in patients with COPD and known primary left ventricular disease when they are compared with the control group.

ABBREVIATION L. V.:Left Ventricle. LVIDd: Diastolic left ventricular internal dimension. LVIDs: Systolic left ventricular internal dimension. FS%: Fraction shortening percent. LVPWT: Left ventricular posterior wall thickness during diastole. IVSP: Interventricular septal wall thickness. COPD:Chronic obstructive pulmonary disease. FEV1 %: Forced expiratory volume in one second percent of predictive value. MMEFR: Maximum mid expiratory flow rate. RBS:Random blood sugar.
Introduction

Cigarette smoking is well known to be the leading cause of chronic obstructive pulmonary disease (chronic bronchitis and emphysema) \(^{[1]}\). On other hand, it is an important cause for coronary ischemia and subsequent left ventricular dysfunction.

Several investigators had found that left ventricular dysfunction occurs secondarily to COPD and right ventricular hypertrophy independent to the presence of coronary ischemia or other disease \(^{[2,3]}\), and this is attributed to several factors including chronic hypoxia, hypercapnea, increase plasma volume, polycythemia and increase cardiac output \(^{[4]}\).

However most of the investigators, specifically the recent, had demonstrated a completely different picture, majority of patients with COPD have normal left ventricular function once other causes are excluded \(^{[5,6,7,8]}\).

Detection of left ventricular hypertrophy and left ventricular dysfunction in a proportion of patients without evidence of clinical myocardial ischemia may be attributed to an occult coronary ischemia, an expected problem in elderly chronic smokers \(^{[9]}\).

The recognition of left ventricular dysfunction is crucial in patients with COPD. Clinical signs may be completely unreliable such as dyspnea, orthopnea, oedema, gallops and cardiomegaly as these signs may be seen in pulmonary disease without left ventricular dysfunction.
The present study is designed to assess the change of left ventricular wall thickness and contractility. Echocardiography is used to assess these changes.

The purpose of this study is to find whether investigation for left ventricular function is mandatory for patients with COPD as left ventricular failure is an important cause of dyspnea.

**Patients and Methods**

This study had been conducted in Saddam teaching hospital in Najaf at the period from December 1999 till September 2000.

Fourteen Normal subjects were considered as control group (group 1), and 31 inpatients with severe COPD were evaluated during their admission. 26 of them have severe COPD with out evidence of other disease and consider as group 2. The remainder five patients have severe COPD and primary LV disease represent (group 3). Each member in the 3 groups examined clinically and send for the following investigations:

1. Serum cholesterol and RBS.
2. Electrocardiography.
3. Pulmonary function lest. Had been done by the same person Using (Autospiror DISCOM.14) model. We measured the FEV1% and MMEFR.
4. Echocardiography: had been done by the same person using M mode and two dimension echocardiography using (Voluson 530 D, Kretz technik Korea data system) model. We measured the LVIDd, LVIDs, LVPWT during diastole, IVST and FS% which was calculated from the following equation:

\[
\text{FS} = \frac{\text{LVIDd} - \text{LVIDs}}{\text{LVIDd}} \times 100\%
\]

**Group 1:**

These individuals have the following characteristics:

1) No history of previous cigarette smoking or prior cardiopulmonary
disease
2) No history of medication.
3) Normal blood pressure, serum cholesterol, RBS and electrocardiograph).
4) Pulmonary function test:
   a. Their mean FEV1% more than 90%.
   b. Their mean MMFER 2.7 L/Sec.

5) Their mean age 58 years (range 40-63) 13 male and 1 female. Group 2:

These patients have the following characteristics:
1- History of chronic cigarette smoking with cigarette index of at least 30 pack per years.
2- History of chronic cough with or without sputum.
3- No history of chronic cardiac disease including ischemic heart disease, hypertensive heart disease, valvular heart disease or cardiomyopathy.
4- No medication a part from drugs for COPD (Bronchodilators, steroids, antibiotics ...etc).
5- Pulmonary function test:
   a) Their mean FEV1% (46).
   b) Their mean MMFR 0.42 L/Sec.
6- Normal RBS, serum cholesterol, blood pressure and electrocardiography.
7- Their mean age 58 years (range 50-70), 2 of them were female.

Group 3:

They have the same characteristics of group 2 but they have history of ischemic heart disease and heart failure documented by electrocardiographic changes and chest X-ray finding. All of them are taking medication for this problem.

RESULTS

The control subjects (group 1) had the following values:
1. The mean FEV1% (91.4 ± 3) and the mean MMFR (2.7 ± 0.15 L/Sec). Table (1).
2. The mean LViDd (44.5 ± 3.7 mm). Table (2).
3. The mean LViDs (30 ± 3.4mm). Table (2).
4. The mean LV PW thickness during diastole (10.5 ±2.4 mm). Table 2.
5. The mean L VST (10.2 ± 1.7 mm). Table 2.
6. The mean of their FS% (34.6 ± 4.9). Table 2.
7. All the echocardiographic results of this group lie with the normal range
The patients of group 2 had the following results:

1. There was a marked reduction in their FEVi% and MMEFR (47.7 ±1.9), (0.42 = 0.12 L /Sec) respectively, Table (1).
2. The mean LVIDd was (50 ± 6.2 mm) which is significantly higher than group 1 subjects in 18 patients (69%), P. value <0.005, Table (2).
3. The mean LVIDs was (34.5 ± 6.7mm) which is significantly higher than group 1 subjects in 17 patients (65%), P. value <0.005, Table (2).
4. The mean EVPW thickness during diastole was (16.9±4.6mm) which is also significantly higher than group 1 in 19 patients (73%), P. value <0.005, Table (2).
5. The mean IVST was (14.3 ±2.6mm) which is significantly higher than group 1 in 22 patients (84%), P. value <0.005, Table (2).
6. The mean of their FS% was (30 ± 6.6) which is lower than group 1 in 8 patients (30%) but statistically not significant, P. value <0.025, Table (2).

The patients in group 3 had the following results:

1. There was a marked reduction in their FEVi% and MMEFR (41.8 ± 2.1 ), (0.35 - 0.05 L /sec) respectively, Table (1).
2. The mean LVIDd was (48 ± 7.8 mm) which is significantly higher than group 1 subjects in 4 patients (80%), P. value <0.005, Table (2).
3. The mean LVIDs was (47 ± 8 mm) which is significantly higher than group 1 subjects in (100%), P. value <0.005, Table (2).
4. The mean LVPW thickness during diastole was (14.8 ± 3mm) which is significantly higher than group 1 in all patients (100%), P. value <0.005, Table (2).
5. The mean IVST was (16.2 ± 2.6mm) which is significantly higher than subjects in group 1 in 4 patients (80%), P. value <0.005, Table (2).
6. The mean FS % was (15 ± 3.9) which is significantly lower than group 2 subjects in all patients (100%), P. value <0.005, and also significantly lower than group 2, P. value 0.005, Table (2).

Table ((1))
Results of the pulmonary function test

<table>
<thead>
<tr>
<th></th>
<th>Group I</th>
<th>Group II</th>
<th>Group III</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV1%</td>
<td>91.4±3</td>
<td>47.7±7.9</td>
<td>41.8±2.1</td>
</tr>
<tr>
<td>MMEFR L/Sec</td>
<td>2.71±0.15</td>
<td>0.42±0.12</td>
<td>0.35±0.05</td>
</tr>
</tbody>
</table>

MMEFR: maximum mid expiratory flow rate. 
FEV1 %: Forced expiratory Volume in one second percent of predictive value. 
Group I: group (1) control subjects. 
Group II: group (2) patients with severe COPD. 
Group III: group (3) patients with severe COPD and known primary LV disease.

Table (2)

Echocardiographic results of the three groups

<table>
<thead>
<tr>
<th></th>
<th>Group 1</th>
<th>Group 2</th>
<th>Group 3</th>
<th>P. Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVIDd</td>
<td>44.5±3.7</td>
<td>50±6.2</td>
<td>48±2.8</td>
<td>*&lt;0.005</td>
</tr>
<tr>
<td></td>
<td>**&lt;0.005</td>
<td>***NS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVIDs</td>
<td>30±3.4</td>
<td>34.5±6.7</td>
<td>47±8</td>
<td>*&lt;0.005</td>
</tr>
<tr>
<td></td>
<td>**&lt;0.005</td>
<td>***&lt;0.005</td>
<td>***NS</td>
<td></td>
</tr>
<tr>
<td>LVPWT</td>
<td>10.5±2.4</td>
<td>16.9±4.6</td>
<td>14.8±3</td>
<td>*&lt;0.005</td>
</tr>
<tr>
<td></td>
<td>**&lt;0.005</td>
<td>***NS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IVST</td>
<td>10.2±1.7</td>
<td>14.3±2.6</td>
<td>16.2±2.6</td>
<td>*&lt;0.005</td>
</tr>
<tr>
<td></td>
<td>**&lt;0.005</td>
<td>***NS</td>
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</tbody>
</table>
**DISCUSSION**

The echocardiographic value in detection of left ventricular abnormalities has some limitations in patients with emphysematus lung changes, however it's an easily reproducible, noninvasive and non time consuming test. This study and several investigators are in agreement at applying echocardiography for this purpose and the results are informative and conclusive.

Left ventricular hypertrophy is found to be prominent in patients with COPD irrespective of the presence of clinically detectable left ventricular disease as there is an increase in LVPWT and IVST in both groups II and III which is significant comparatively with group I, this result is agreed by most of the investigators \(^{(23,6,7,12,13)}\). An increase in LV preload is suggested to be the essential cause and this is attributed to polycythemia, increased plasma volume and hypoxia. This concept is highly suggested in this study as there is frank elevation in LVIDd as compared to minor elevation in LVIDs. The minor systolic dysfunction was reflected in mild decrease in fraction shortening which is statistically not different from control group, only eight patients had mild to moderate reduction in FS% in group II patients comparing this result with that of group III; primary LV disease in important in causing left ventricular dysfunction, this result suggests that the cause of LV dysfunction in these eight patients is probably due to the an occult coronary vascular disease which cannot be ruled out without further invasive procedures like angiography \(^{(5,6,7,13,14)}\) on the other hand the clinical features of

| FS%  | 34.6 ±4.9 | 30 ±6.6 | 15 ±3.9 | **NS**  
|------|-----------|---------|---------|--------
|      | **<0.005** | ***<0.005** |         |        |

Mean ±SD

*P. Value between group I and group II.
**P. Value between group I and group III.
***P. Value between group II and group III.

FS% : Fraction shortening percent.
LVIDd : Diastolic left ventricle internal dimension.
LVIDs : Systolic left ventricle internal dimension.
LVPWT : Left ventricular posterior wall thickness during diastole.
IVSPT : Interventricular septal wall thickness.
Group 1 : Control subjects.
Group 2 : Patients with severe COPD.
Group 3 : Patient with severe COPD and known primary LV disease.
left ventricular dysfunction in patients with COPD may be misleading,\(^{(5,7,9)}\) and the unjustified treatment for left ventricular failure can be hazardous in these patients, most notably the increased, digitalis intoxication and the adverse effect of excess diuretics and venodilator.

Conclusively, Assessment of left ventricular function by echocardiography is mandatory for proper management specially in severely ill patients.

REFERENCES

10. McDonald, LG, Fegenbium, H, Chang, S. Analysis of left ventricular wall motion by reflected ultrasound application to assessment of myocardial function.
