RESEARCH ARTICLE
Liver function tests in patients with chronic obstructive pulmonary disease in a tertiary care hospital

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ABSTRACT
Background: Cases of chronic obstructive pulmonary disease (COPD) increasing day by day. Smoking is the major causative agent for COPD. The present study undertaken to estimate the deterioration of liver functions in COPD patients due to smoking. Aims and Objectives: This study aims to investigate various liver function tests (LFTs) such as total bilirubin, alanine amino transferase/serum glutamic pyruvic transaminase (SGPT), aspartate amino transferase/SGPT, alkaline phosphatase (ALP), serum ammonia, and serum albumin among COPD patients. To correlate LFTs with forced expiratory volume at end of 1 second (FEV₁) and FEV₁/FVC ratio. Materials and Methods: In our present study, we selected 86 cases of COPD in the age group of 41–60 years. All subjects were smokers and having FEV₁ <80% and FEV₁/FVC <70%. A control group consists of 55 healthy individuals. LFTs were studied. FEV₁ and FEV₁/FVC ratio were correlated with LFTs. Statistical analysis done using SPSS software 20.0 Windows version. Results: Mean level of SGPT among cases was 117 ± 10.23 IU/L against controls who had mean SGPT 11.16 ± 8.32 which was statistically significant (P < 0.05). Likewise, mean level of s. ammonia among cases was 154 ± 10.22 mcg/dl as compare to controls who had mean 24.43 ± 7.97 mcg/dl (P < 0.05). However, no significant difference was found in rest of the LFTs. Furthermore, there was no correlation between altered LFTs and severity of COPD. Conclusion: LFTs are significantly altered in COPD patients due to smoking. Corticosteroids treatment must not be initiated without investigating LFTs among COPD patients.

KEY WORDS: Liver Function Tests; Chronic Obstructive Pulmonary Disease; Forced Expiratory Volume 1; Forced Expiratory Volume 1/Forced Vital Capacity

INTRODUCTION
Chronic obstructive pulmonary disease (COPD) is a common non-communicable disease (NCD) which can be prevented. Usually, it is characterized by air hunger secondary to and airflow limitation, pathology lies in the airway and/or alveoli.[1] Cases of COPD in India are rising continuously and it is the second most common cause of NCD-related deaths in India. Above 30 years of age population have high prevalence of COPD.[2] Growth of India, in terms of urbanization and pollution, has put its people at risk of having COPD. COPD is diagnosed with the help of spirometry. Spirometry shows chronic airflow limitation even after giving the bronchodilators to COPD patients. COPD is now considered as multiorgan disease and liver damage is also likely. In recent times, deterioration of liver functions among COPD patients has been observed by many clinicians. It has been seen that COPD patients are more likely to have non-alcoholic fatty liver disease.
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Liver disease (NAFLD). In patients of NAFLD, liver accumulates excessive triglycerides in absence of alcohol consumption or viral hepatitis. If a patient is having COPD with NAFLD, he/she is at very high risk of cardiovascular-related mortality. Management of COPD patients must be through integrated approach keeping in mind likely multiorgan involvement. Another aspect for having COPD is chronic smoking. Smoking has variety of hazardous effects and organs not in direct contact of smoke such as liver also damaged over the period of time.

In our present study, we decided to assess various liver function tests (LFTs) such as total bilirubin, alanine amino transferase-serum glutamic pyruvic transaminase (ALT/SGPT), aspartate amino transferase (AST/SGPT), alkaline phosphatase (ALP), serum ammonia, and serum albumin among COPD patients. We also want to know whether there is any relation between severity of COPD and liver function deterioration. Answer of these research questions will help in holistic and integrated management of COPD patients.

MATERIALS AND METHODS

In this observational cross-sectional study, 86 male patients either admitted with signs and symptoms of acute exacerbation of COPD or coming for outpatient consultation evaluated for their liver health. Approval of ethics committee of N.H.L. Municipal Medical College, Ahmedabad, was obtained.

Consent form from all study participants was also obtained beforehand.

Inclusion Criteria

The following criteria were included in the study:

• Male patients with age 41–60 years and forced expiratory volume in 1 s (FEV₁) <80% and FEV₁/forced vital capacity (FVC) <0.7 of predicted value

• Chronic smoker with 20–30 pack-years of duration of exposure.

Exclusion Criteria

The following criteria were excluded from the study:

• History of asthma

• Chronic kidney disease

• Chronic liver disease

• Congestive cardiac failure

• Fluid overload

• Patient taking sympathomimetic or hepatotoxic drugs

• Not willing to take part

• Any other coexisting disease

• Obesity

• Alcoholics.

Procedure

All the study subjects and 55 controls were sent to the pulmonary function testing (PFT) laboratory after thoroughly taking occupational and smoking history. We explained the entire procedure for PFT beforehand and computerized spirometer (kit micro, COSMED, Srl, Rome Italy) was used. Record of all the flow volume curves also maintained and best of the three results was considered as per American Thoracic Society guidelines. For admitted patients, we took the reading on the day before the discharge.

Five milliliters blood samples were drawn from the cubital vein with proper aseptic precautions and samples sent for LFTs. Total bilirubin, ALT/SGPT, AST/SGPT, ALP, serum ammonia, and serum albumin were analyzed of all the subjects and controls on Siemens automatic analyzer (Dimension EXL 200).

COPD Classification

COPD was defined with modified Global Initiative on Obstructive Lung Disease criteria as follows: Post-bronchodilator FEV₁/FVC <0.70. Mild COPD was defined as FEV₁% predicted ≥80%, moderate as FEV₁% predicted ≥50% and <80%, severe as FEV₁% predicted ≥30% and <50%, and very severe as FEV₁% predicted <30%.

Statistical Analysis

Statistical analysis performed using SPSS software (version 20.0). Cases and controls were compared using “t”-test; to find out any significance of LFT based on severity of COPD, we used analysis of variance test.

RESULTS

Eighty-six patients of COPD and 55 controls were studied [Table 1]. We had taken only male subjects and all the cases were smokers with 20–30 pack-years of exposure. There was no significant difference among cases and controls for baseline characteristics. All subjects’ hemogram, renal functions, blood sugar, and electrocardiogram were normal but all cases had significantly altered pulmonary functions [Table 2]. Table 3 shows comparison of LFTs between cases and controls. We found altered levels (above normal) of s. ammonia, SGPT, and lactic dehydrogenase (LDH). This difference was also statistically significant. Remaining LFTs were within normal range. While comparing the results of altered LFTs according to severity of the COPD [Table 4], we found that there is no significant difference in the levels of s. ammonia, SGPT, and LDH.

DISCUSSION

In our present study, we got significantly altered three laboratory tests measuring liver health, namely, SGPT, s.
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Table 1: Baseline characteristics

<table>
<thead>
<tr>
<th>Variables</th>
<th>COPD patients (Mean±SD), ( n=86 )</th>
<th>Healthy males (Mean±SD), ( n=55 )</th>
<th>( P )-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>49.41±4.49</td>
<td>47.55±4.09</td>
<td>&gt;0.05*</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>68.44±9.18</td>
<td>69.41±14.15</td>
<td>&gt;0.05*</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.67±0.09</td>
<td>1.65±0.07</td>
<td>&gt;0.05*</td>
</tr>
<tr>
<td>BMI</td>
<td>19.8±2.0</td>
<td>19.9±1.96</td>
<td>&gt;0.05*</td>
</tr>
</tbody>
</table>

*No significant difference, BMI: Body mass index, COPD: Chronic obstructive pulmonary disease

Table 2: Spirometry parameters

<table>
<thead>
<tr>
<th>Variables</th>
<th>COPD patients (Mean±SD), ( n=86 )</th>
<th>Healthy males (Mean±SD), ( n=55 )</th>
<th>( P )-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV1</td>
<td>1.39±0.56</td>
<td>3.98±4.09</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>FVC</td>
<td>2.35±0.6</td>
<td>4.35±2.56</td>
<td>&lt;0.001*</td>
</tr>
</tbody>
</table>

*Statistically significant difference, FEV1: Forced expiratory volume at end of 1 s, FVC: Forced vital capacity, COPD: Chronic obstructive pulmonary disease

Table 3: Serum mean concentration of LFTs

<table>
<thead>
<tr>
<th>LFTs</th>
<th>COPD cases (86)</th>
<th>Controls (55)</th>
<th>( P )-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>T. bilirubin</td>
<td>0.8±0.1 (mg/dl)</td>
<td>0.7±0.1 (mg/dl)</td>
<td>&gt;0.05**</td>
</tr>
<tr>
<td>S. ammonia</td>
<td>46.47±10.22 (mcg/dl)</td>
<td>40.43±7.35 (mcg/dl)</td>
<td>&lt;0.05*</td>
</tr>
<tr>
<td>S. albumin</td>
<td>3.9±0.5 (g/dl)</td>
<td>4.2±0.5 (g/dl)</td>
<td>&gt;0.05**</td>
</tr>
<tr>
<td>SGPT</td>
<td>67.18±9.25 (IU/L)</td>
<td>43.2±7.35 (IU/L)</td>
<td>&lt;0.05*</td>
</tr>
<tr>
<td>SGOT</td>
<td>18.5±3.89 (IU/L)</td>
<td>16.39±2.55 (IU/L)</td>
<td>&gt;0.05**</td>
</tr>
<tr>
<td>ALP</td>
<td>69.25±12.52 (IU/L)</td>
<td>50.41±11.19 (IU/L)</td>
<td>&gt;0.05*</td>
</tr>
<tr>
<td>LDH</td>
<td>295.23±28.68 (U/L)</td>
<td>189.67±18.56 (U/L)</td>
<td>&lt;0.05**</td>
</tr>
</tbody>
</table>

*Statistically significant difference, **statistically not significant, SGPT: Serum glutamic pyruvic transaminase, SGOT: Serum glutamic oxaloacetic transaminase, ALP: Alkaline phosphatase, LDH: Lactic dehydrogenase, COPD: Chronic obstructive pulmonary disease, LFTs: Liver function tests

Table 4: Correlation of LFTs with severity of COPD

<table>
<thead>
<tr>
<th>LFTs</th>
<th>Moderate (FEV1≥50% and 1&lt;80% of predicted) (( n=41 ))</th>
<th>Severe (FEV1≥30% and &lt;50% of predicted) (( n=45 ))</th>
<th>Very severe (FEV1&lt;30% of predicted)</th>
<th>( P )-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>S. ammonia</td>
<td>--</td>
<td>45.98±7.44</td>
<td>46.97±6.39</td>
<td>&gt;0.05*</td>
</tr>
<tr>
<td>SGPT</td>
<td>--</td>
<td>66.55±8.21</td>
<td>67.81±7.29</td>
<td>&gt;0.05*</td>
</tr>
<tr>
<td>LDH</td>
<td>--</td>
<td>294.88±17.19</td>
<td>295.58±13.57</td>
<td>&gt;0.05*</td>
</tr>
</tbody>
</table>

*Statistically not significant, FEV1: Forced expiratory volume at end of 1 s, COPD: Chronic obstructive pulmonary disease, LFTs: Liver function tests

ammonia, and LDH. Values of these tests were above their normal range signifies underlying liver damage. However, we could not establish any relation between severity of COPD and alteration in LFT. There was not any significant difference of altered LFTs among moderate and severe category of COPD patients.

COPD patient number is growing day by day and it is becoming the major public health problem in India. COPD patients loose essence of life at the same time it is a great burden to the nation economically. Smoking nowadays very common among youngsters and it has been proved that almost 50% of those who smoke daily, eventually develop COPD.[10] Heavy smokers are those who have exposure of 20 pack-years or more[10] but even one or two cigarettes are also sufficient to cause COPD in future. Smoking can affect variety of organs such as bladder, pancreas, stomach, kidney, and liver also.[10] It is very important to take smoking history properly of all COPD patients. El-Zayadi[11] mentions direct and indirect way of damage caused by cigarettes on most of the organs including liver. Direct mechanism is oxidative stress and lipid peroxidation which lead to fibrosis of liver while indirect damage exerted by tissue hypoxia, secondary polycythemia, and iron overload.[11] Our findings are consistent with a study done by Viglino et al.[13] where they find high chances of having NAFLD among COPD patients. Possible reasons of this are fat inflammation, release of cytokines including tumor necrosis factor-alpha, and last but not least insulin resistance. Another study by Divo et al.[12] prospectively followed 1664 patients with COPD with a systematic record of 79 comorbidities. Liver cirrhosis was reported in 2.5% of patients and associated with 2-fold higher mortality. However, in this study, liver cirrhosis might have developed because of alcohol and smoking, possibility...
of underlying metabolic syndrome can’t be ruled out. COPD and NAFLD both are disease of common etiology where systemic inflammation plays a key role ultimately these two lead to metabolic syndrome. Another study by Viglino et al.[4] confirms increased cardiovascular mortality due to COPD and NAFLD. Many a times, biochemical alterations starts earlier than clinical manifestation of altered liver function manifestations so it is important that clinician detects it early and prescribes medicines accordingly. Most of the time, COPD patients are not evaluated for liver health and put directly on corticosteroid and methylxanthines. These can deteriorate liver functions further. Our study strongly recommends evaluation of liver function in all COPD patients.

We could not get mild and very severe category patients of COPD since these patients were underreported. This actually did not allow us in achieving our objective of establishing correlation between severity of COPD and alteration in LFT to the fullest. Further studies in this regard with larger sample size can be carried out.

CONCLUSION

Smoking significantly affects LFTs in patients of COPD in the form of raised SGPT, s. ammonia, and LDH. We advise regular checkup and raise a caution before putting patient on corticosteroids – keeping liver damage in mind.

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REFERENCES


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