Original Article

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Pulmonary Functions and Respiratory Efficiency in Patients with Cirrhosis and Portal Hypertension

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Abstract

Background: Decreased number of hepatocytes in cirrhosis may cause synthetic or metabolic dysfunction. Ascites due to cirrhosis and portal hypertension can cause restriction to lung expansion. Intrapulmonary shunting through collaterals and opening up of arteriovenous shunts are the main determinants of impaired gas exchange and can even develop without ascites. Cirrhosis also causes hypoproteinemia and decreased muscular efficiency.

Aim: The aim of this study was to evaluate lung function together with respiratory efficiency in patients without severe ascites.

Materials and Methods: In this case – control study, the following parameters were determined with a computerized spirometer "Super Spiro." Respiratory efficiency tests, expiratory blast test, endurance test, and breath holding time were performed.

Results: There was a statistically significant fall in all the lung parameters studied. There was no change in the forced expiratory volume in 1 sec/forced vital capacity ratio. There was a statistically significant decrease in the respiratory efficiency tests in patients with cirrhosis and portal hypertension. There was a restrictive pattern of lung disease and also obstructive changes in small and mid-airways.

Conclusion: Liver cirrhosis and portal hypertension are associated with pulmonary complication. Early detection of pulmonary complication may lead to good prognosis of the disease.

Key words: Cirrhosis, Lung parameters, Portal hypertension, Respiratory efficiency

INTRODUCTION

Pulmonary function tests are very useful for the evaluation of lung function in respiratory disorders. The liver plays a key role in critical metabolic pathways and systemic functions. It is strategically suited to perform this diverse function by being the first organ to receive the nutrient-enriched blood from the portal system, the unique vascular structure of the live. Cirrhosis of the liver is characterized by the replacement of normal liver tissue with collagen and fatty tissue as the number of hepatocytes decreases.[1-5]

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There is an impairment of all physiologic functions of liver in patients with liver cirrhosis. Cirrhotic patients are at a risk for specific abnormalities of pulmonary mechanics, hemodynamics, and ventilation perfusion mismatch.[6-8] Ascites occurring secondary to both cirrhosis and portal hypertension can cause restriction to lung expansion with characteristic decrease in the mean lung volume including total lung capacity (TLC) and residual volume (RV). Intrapulmonary shunting through collaterals and opening up of arteriovenous shunts are the main determinants of impaired gas exchange and can even develop without ascites. This study attempts to evaluate lung function together with respiratory efficiency in patients without severe ascites.[9-12]

Aim

The aim of the study was to compare the changes in pulmonary functions in patients with liver cirrhosis and portal hypertension.

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MATERIALS AND METHODS

A case–control study was done in a tertiary care hospital. Ethical clearance and patient's informed consent were obtained. Patients who were in the age range from 20 to 50 years were included in the study. Healthy volunteers in both genders with no history of smoking and alcohol were included in the control group. Patients with cirrhosis, portal hypertension, and mild or moderate ascites were included; patients with other comorbidities were excluded from the case group. The following parameters were determined with a computerized spirometer "Super Spiro." This has a volume transducer, which measures expired air directly at body temperature and pressure with saturated water vapor, forced expiratory volume in 1 sec (FEV1), forced vital capacity (FVC), FEV1%, peak expiratory flow (PEF), mid expiratory flow (MEF) 75, MEF 50, MEF 25, maximal mid expiratory flow (MMEF), and maximum voluntary ventilation (MVV). Respiratory efficiency tests, expiratory blast test, endurance test, and breath holding time were performed. All the values for control and study group were analyzed. The mean and standard deviation and appropriate Student's *t*-test were performed.

RESULTS

There is a highly significant difference between FEV, FVC, PEF, MEF 75, MEF 50, MEF 25, MMEF, and MVV, with $P < 0.0001$ (Table 1). There is no significant difference in FEV1/FVC with $P = 0.632$ (Figure 1). Expiratory blast test and endurance test showed highly significant difference between case and control group (Figures 2 and 3).

DISCUSSION

Pulmonary function tests are very useful for the evaluation of lung function in respiratory disorders. Being physiological tests, they can only indicate whether the disease process has caused the impairment of function. They may not be detecting the early stage of the disease. They also cannot make a specific clinical diagnosis. However, they can give an objective assessment of the

Table 1: Demographic and PFT results

Variables	Mean±SD		*P* value*
	Control	Case	
Age	38.6±9.23	42.4±9.92	0.131
Male	26 (86.7)	26 (86.7)	n/a
Female	4 (13.3)	4 (13.3)	n/a
Height	160.8±5.44	162.4±5.48	0.242
Weight	65.83±5.35	62.4±8.64	0.069
PEF	406.6±57.02	235.93±35.27	<0.0001
MEF75	6.64±0.81	3.87±0.62	<0.0001
MEF50	4.86±0.64	2.63±0.50	<0.0001
MEF25	2.77±0.50	1.16±0.30	<0.0001
MMEF	4.50±0.60	2.25±0.35	<0.0001
MVV	114.80±12.97	55.73±7.90	<0.0001

*Student t test, PEF: Peak expiratory flow, MF: Mid expiratory flow, MMEF: Maximal mid expiratory flow, MVV: Maximum voluntary ventilation, PFT: Pulmonary function tests

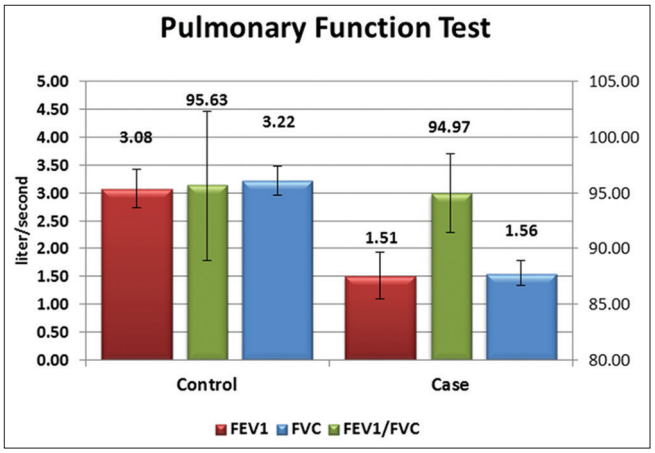

Figure 1: Comparison of case and control pulmonary function test results

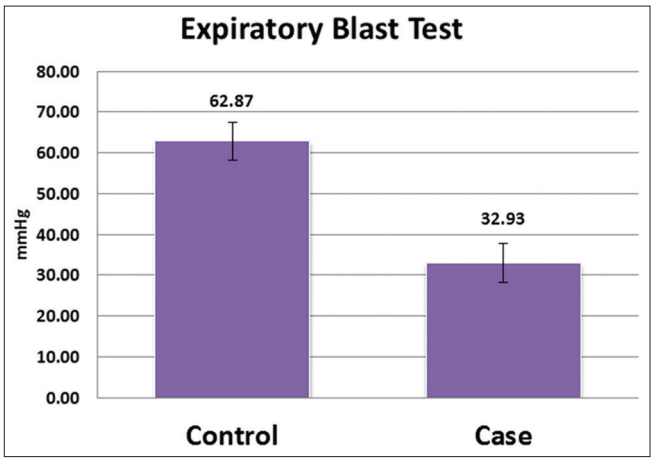

Figure 2: Comparison of case and control expiratory blast test results

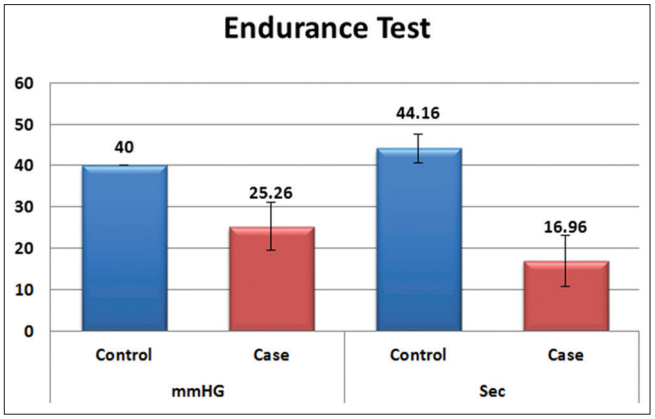

Figure 3: Comparison of case and control endurance test results

functional status of the respiratory system and indicate the nature and extent of functional disturbance in disorders associated with pulmonary impairment and disability. Serial measurements are useful in the following course of the disease, evaluating therapy and determining prognosis. Edison[13] studied the pulmonary function in patients with cirrhotic ascites and compared with normal controls. The vital capacity and MVV were significantly affected in patients with ascites. The functional residual capacity and TLC were low. He noticed that pulmonary function changes were restrictive in nature at the early stages; as the disease progressed, there was an obstructive type of change in the larger airways. He explained that the cause of interstitial edema in cirrhosis included decreased plasma colloid osmotic pressure and the portopulmonary shunt that developed impairment of the metabolizing function resulting in elevated endotoxins and histamine activity. This causes increased pulmonary capillary permeability. It further leads the lymph entering extrathoracic lymphatic vessels. Caruso *et al.*[8] studied 27 patients with liver cirrhosis and 36 control subjects. They showed a decrease in peak expiratory flow rate (PEFR) and FEF 25. A restrictive disorder was observed only in patients with child's C with hyperbilirubinemia and lower albumin levels. Patients with esophageal varices had a decrease in FEF 25-75. In 1998, Behra *et al.*[5] also identified low FVC and FEV, with normal ratio in patients with portal hypertension, and a low PEFR was also reduced in patients. The present study also correlated with this observation of a normal FEV1/FVC ratio. Hara *et al.*[14] also reported that a fall in FEF 50 and FEF 75 explains similar to our study which may be due to mechanical compression on small airways by interstitial edema induced by the presence of vasoactive substance and endotoxins in the circulating blood. One of the studies reported the pulmonary functions in portal hypertension of different etiologies with various grades of ascites. They found that in cirrhotic patients without ascites, FVC, RV, TLC, and functional residual capacity were lower than the predicted values. In patients with ascites, FVC, FEV, and FEF (25-75) were lower than the predicted values. Patients with non-cirrhotic portal hypertension had normal pulmonary function. Another study explained that thoracic cavity plays an important role in the mechanical ventilatory function, and the alteration of some of its structures due to decompensate cirrhosis and the presence of ascites and muscle atrophy directly influence its normal function. They showed that in cirrhotic patients, a decreased ventilatory function of a restrictive type and decreased respiratory muscular tension which improved after the resolution of ascites. The study group had decreased values of VC, TLC, and RV. The diffusion capacity was altered. He also compared pulmonary function with albumin levels. They found that reduced albumin levels showed a decrease in VC, TLC, and diffusing capacity of lung for carbon monoxide levels. In our study, there was a significant decrease in almost all the parameters studied, namely, FVC, PEFR, MMEF, MEF 25, MEF 50, and MEF 75 except FEV1%. This relates well with the study conducted by Abelmann *et al.*[1] who studied the lung function in various cases of abdominal distension such as ascites, congestive cardiac failure, and pregnancy. They demonstrated the restrictive pattern in all the groups, especially a significant decrease in FVC. The increase in the rigidity of the abdominal wall and subsequent muscle atrophy directly influences the respiratory muscle function resulting in restrictive ventilatory defect and increases the work of breathing. In our study, this has been very well evidenced. There was a significant fall in MVV of 40 mmHg, endurance test, and expiratory blast test. Although ascites contributed to a major extent, there were other pathophysiological changes occurring in the lungs and also in the muscles which brought a huge significant fall in all the lung parameters studied. Although this fall leads to a restrictive pattern, there were also obstructive changes more so in the mid and small airways.[15]

CONCLUSION

There was a significant fall in all the lung parameters except FEV1/FVC in the study group compared to the control group. There was a significant change in the respiratory efficiency also. There was a huge fall in the expiratory flow rate. This explains the restrictive pattern of lung disease and also obstructive changes in small and mid-airways. Liver cirrhosis and portal hypertension can cause pulmonary dysfunction. Early detection of pulmonary dysfunction may improve the respiratory functions in patients with cirrhosis and portal hypertension.

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