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Respiratory and cardiovascular complications in patients with liver cirrhosis due to hepatitis C virus and its impact on quality of life

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ABSTRACT

Objective: To assess the respiratory and cardiovascular complications in patients with liver cirrhosis due to hepatitis C virus and its impact on quality of life.

Subjects and methods: The study was carried out on 100 patients with liver cirrhosis and 50 healthy control volunteer without liver cirrhosis where respiratory and cardiovascular complications were assessed by arterial blood gas (ABG) analysis (PaO₂, PaCO₂ and pH), pulmonary function tests (PFTs) [forced vital capacity (FVC%), forced expiratory volume in one second (FEV1%), FEV1/FVC ratio and forced expiratory flow in 25% to 75% of FVC (FEF 25–75%)] and echocardiography [pulmonary artery systolic pressure (PASP), left atrial (LA) area, diastolic dysfunction (DD), and pulse rate].

Results: The pulmonary artery systolic pressure (PASP) measured by echo was significantly higher (48.23 mmHg) in cirrhotic patients than that of the control non cirrhotic group (28.02 mmHg) ($P < 0.001$). By ABG analysis, PaO₂ was significantly lower in cirrhotic patients (52.29 mmHg) than in the control group (79.34 mmHg) ($P < 0.001$) with a significant negative correlation between PASP and PaO₂. On the other hand, the differences between both groups regarding PaCO₂ and pH were insignificant ($P > 0.05$). There was a highly significant positive correlation between portal vein diameter and the level of PASP. High mortality rate 41% was reported among patients with high PASP. Regarding the PFTs, the differences between both groups regarding the FVC%, FEV1%, FEV1/FVC and FEF 25–75% were insignificant ($P > 0.05$).

Conclusion: Respiratory and cardiovascular complications (elevated PASP, hypoxemia, increased LA area DD and/or tachycardia) are common complications in hepatic patients and should be assessed for control of symptoms, quality of life and survival.

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Introduction

Pulmonary hypertension and hypoxemia were addressed in many researches as complications of liver cirrhosis. Portopulmonary hypertension (POPH) is a form of pulmonary arterial hypertension (PAH) associated with portal hypertension with or without underlying chronic liver disease. POPH is increasingly recognized and recent evidence suggests that it is one of the leading

causes of PAH [1]. The pathophysiology of POPH is poorly understood although the pathological changes in pulmonary vasculature in advanced POPH are similar to those seen in idiopathic pulmonary hypertension (PH) [2]. A hyperdynamic systemic circulation with increased cardiac output and reduced systemic vascular resistance is present in roughly half of patients with end-stage liver disease [3,4].

Aim of the study

To study the incidence of respiratory and cardiovascular complications among patients with liver cirrhosis and its impact on the disease outcome

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Subjects and methods

The study was carried out during the period from June 2014 to June 2016 on 100 patients with liver cirrhosis due to hepatitis c virus referred from the Tropical Department, Menoufia University Hospitals, Egypt. They were 59 males and 41 females with a mean age of 53 ± 6.49 years. Also, the study included 50 age and sex matched healthy controls without liver disease. They were 32 males and 18 females with a mean age of 51 ± 6.48 years. The study was approved by the ethical committee of Menoufia University Hospitals and informed consents were taken from all subjects before enrollment in the study.

Thorough medical history taking then general and local clinical examination were done. Laboratory and radiological examinations were done including abdominal ultrasound. The Child-Pugh score (sometimes the Child-Turcotte-Pugh score) was used to assess the prognosis of chronic liver disease, mainly cirrhosis. It includes five clinical measures of liver disease, each measure is scored 1–3, with 3 indicating most severe derangement (Fig. 1) [5].

Arterial blood gases were taken from all patients under complete aseptic conditions from the radial artery on adequately heparinized syringe under anaerobic conditions then the levels of PaO₂, SaO₂, PaCO₂ and pH were taken [6]. Pulmonary functions (FVC%, FEV1%, FEV1/FVC, FEF 25–75) were measured for all patients and controls using Quark PFTs – Italy.

A complete echocardiographic study, including chamber quantification and complete flow doppler was performed using a vivid 9 system (General electric, made in Norway). The frequency of the transducer was 2.5 MHz.

In lateral decubitus, parasternal M-mode images were used to measure left ventricular (LV) and left atrial (LA). By echo, we estimated PASP using tricuspid regurge (TR) velocity. PASP was determined from the peak TR jet velocity, using the simplified Bernoulli equation and combining this value with an estimate of the RA pressure: PASP = 4(V)² + RA pressure, where V is the peak velocity (in

meters per second) of the TR jet, and RA pressure was estimated from inferior vena cava (IVC) diameter and respiratory changes [7].

The normal cutoff value for invasively measured mean PA pressure (by right ventricular catheter) is 25 mmHg at rest. In the echocardiography laboratory, PASP is more commonly measured and reported. Normal resting values are usually defined as a peak PASP of 35 or 36 mmHg, assuming a RA pressure of 3 to 5 mmHg. The American College of Cardiology Foundation and American Heart Association expert consensus document considers estimated PASP greater than 40 mmHg as indicative of pulmonary hypertension and recommends further evaluation of these patients with right heart catheter [7]. Dyspnea was assessed and scored by mMRC score as follows (Fig. 2) [8].

The symptoms were assessed by New York Heart Association (NYHA) as follows [9]

- Class 1: Asymptomatic.
- Class II: Symptoms with moderate activity.
- Class III: Symptoms with mild activity.
- Class IV: Symptoms at rest.

Results

In Table 1: The PASP was significantly higher in cirrhotic patients (48.23 mmHg) than that of control group (28.02 mmHg). By ABG analysis, the PaO₂ was significantly lower in cirrhotic patients (52.29 mmHg) than in the control group (79.34 mmHg). Also, the LA area was higher in cirrhotic patients (3.84 mm) in comparison to that in the control group (2.99 mm) and the same for pulse rate which was higher in the cirrhotic group (89.91 bpm) than in the control group (67.78 bpm). Regarding the PaCO₂ and pH, there was no significant differences between both groups. Also, PFTs (FVC%, FEV1%, FEV1/FVC and FEF 25–75%)

Clinical and Lab Criteria	Points		
	1	2	3
Encephalopathy **	(grade 0)	(grade 1 or 2)	(grade 3 or 4)
Ascites *	None	Mild to moderate (diuretic responsive)	Severe (diuretic refractory)
Bilirubin (mg/dL)	< 2	2-3	>3
Albumin (g/dL)	> 3.5	2.8-3.5	<2.8
Prothrombin time			
Seconds prolonged	<4	4-6	>6
International normalized ratio	<1.7	1.7-2.3	>2.3

GRADE ²	DESCRIPTION	POINTS
(A)	Mild; well-compensated disease	5-6
(B)	Moderate; significant functional compromise	7-9
(C)	Severe; decompensated disease	10-15

Figure 1. The Child-Pugh score [5].

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