

Hepatopulmonary Syndrome among Cirrhotic Patients in Upper Egypt: Prevalence, Clinical Presentations and Laboratory Features

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Abstract

Background: The prevalence of Hepatopulmonary Syndrome (HPS) ranges from 5 to 32% from liver-transplantation centers. Egypt is considered as one of the highest countries in prevalence and incidence of bilharziasis, peri-portal fibrosis and Hepatitis C Virus (HCV) induced liver cirrhosis. Clinical, radiological and laboratory features of HPS were not widely assessed.

Objectives: To determine the prevalence, clinical features and laboratory features of HPS among Egyptian cirrhotic patients.

Patients and Methods: Our study included 570 cirrhotic patients. Arterial blood gases analysis, chest X-ray, pulmonary function tests and transthoracic contrast echocardiography for detection of pulmonary vasodilatation were done for patients with partial pressure of arterial O₂ < 80 mmHg. Also, clinical and laboratory features were assessed. Diagnostic criteria of HPS in cirrhotic patients include arterial hypoxemia and pulmonary vascular dilatation on contrast enhanced echocardiography.

Results: The prevalence of HPS among patients with liver cirrhosis was 4.2%. Patients with HPS had more severe cirrhosis, as determined by advanced Child-Pugh Grade. The presence of dyspnea, platypnea, clubbing, and orthodeoxia was significantly higher in patients with HPS when compared to cirrhotic patients (P value < 0.001). In HPS, right pleural effusion and bilateral basal shadows were the commonest radiological findings (20.8% while chest X-ray of most patients with liver cirrhosis was normal (85%) (P value < 0.05). There was a significant decrease in PaO₂ and O₂ saturation (P < 0.001 for each) but a significant increase in P (A-a) O₂ in patients with HPS versus cirrhotic patients (P < 0.001). Patients with HPS showed a restrictive dysfunction in 59.3%.

Conclusion: The prevalence of HPS among cirrhotic patients was 4.2%. The presence of dyspnea, platypnea, clubbing, orthodeoxia and arterial hypoxemia were the commonest feature. Right pleural effusion and bilateral basal shadows were the commonest radiological findings.

Keywords: Liver cirrhosis; Hepatopulmonary syndrome; Prevalence; Clinical feature

Introduction

Hepatopulmonary Syndrome (HPS) is defined as an arterial oxygenation defect induced by pulmonary vascular dilatation in the setting of liver disease [1]. HPS have been occurred with both acute and chronic liver diseases and most commonly presents in patients with chronic liver diseases resulting in cirrhosis [2]. The prevalence of the hepatopulmonary syndrome ranges from 5 to 32% from liver-transplantation centers [3]. Pulmonary features of HPS include cyanosis, dyspnea, platypnea and orthodeoxia and digital clubbing. Platypnea and orthodeoxia are defined as dyspnea and arterial deoxygenation induced by the upright position and relieved by recumbency [4].

Egypt has a very high prevalence of HCV and a high morbidity and mortality from chronic liver disease and cirrhosis. Approximately 20% of Egyptian blood donors are anti-HCV positive [5]. Chronic hepatitis B is an important medical problem in Egypt. The prevalence of HBs Ag in Egypt is intermediate (2%-7%) [6]. Little data is available about HPS in Egyptian cirrhotic patients.

Aim of the study

The aim of this study was to clarify the prevalence of HPS in cirrhotic patients in Upper Egypt and to determine its main clinical and laboratory features.

Patients and Methods

This study included 570 patients with liver cirrhosis who were consecutively admitted in Tropical Medicine Department. Patients with tense ascites underwent large volume paracentesis before their recruitment in the study.

- Selection criteria for patients with liver cirrhosis:
 1. Clinical criteria of liver cirrhosis.
 2. Ultrasonographic confirmation of liver cirrhosis (course liver, irregular surface ± reduced size).
 3. Biochemical confirmation of liver cirrhosis.
- Exclusion criteria:

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Received May 10, 2012; Accepted July 15, 2012; Published July 20, 2012

Citation: Makhlouf NA, Azeem AA, Makhlouf HA, Moustafa EA, Ghany MA (2012) Hepatopulmonary Syndrome among Cirrhotic Patients in Upper Egypt: Prevalence, Clinical Presentations and Laboratory Features. J Liver 1:108. doi:10.4172/2167-0889.1000108

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1. Patients with cardiopulmonary diseases and those with current smoking were excluded from the study.
2. The presence of right pleural effusion is not an excluding criterion.
 - All patients were subjected to:
 1. History and physical examination.
 2. Liver function test.
 3. Abdominal ultrasonography.
 4. Chest X-ray, posteroanterior and lateral views.
 5. Measurement of O₂ saturation by pulse Oxymeter as initial screening test.
6. Arterial blood gas analysis: Those with O₂ saturation less than 95% on pulse Oxymeter had undergone arterial blood gas analysis in recumbent position and after standing to detect orthodeoxia. Arterial blood gases on room air were obtained by blood sample from radial artery and analyzed using blood gases analyzer (Rapid lab 850; CHIRON/Diagnostics; critical care systems) for determining pH, arterial O₂ tension (PaO₂), arterial CO₂ tension (PaCO₂) and O₂ saturation. Orthodeoxia was considered when there was decrease in PaO₂ ≥ 5% or ≥ 4 mmHg from the supine to upright position [7].
7. Spirometry was done for patients with arterial O₂ saturation less than 95%. Spirometer (D 97723; Zan 300, Oberthulba, Germany) was used to determine Forced Expiratory Volume in 1st second (FEV1), the Forced Vital Capacity (FVC), and the Tiffeneau value (FEV1/FVC %) and forced expiratory flow from 25-75%. Results were expressed in liters and liters/second and as percentage of normal value for gender, age, and height (percent predicted).
8. In patients with hypoxemia (partial pressure of arterial O₂<80 mm Hg), contrast enhanced echocardiography was performed by using agitated saline which creates a stream of microbubbles after intravenous injection [2]. Pulmonary vascular dilatation is diagnosed by contrast-enhanced transthoracic echocardiography (HD Sonos 5000) with saline (shaken to produce microbubbles>10 μm in diameter) [3,4]. Under normal circumstances, these microbubbles, 60 to 90 μm in diameter, opacify only the right heart chambers because they are filtered in the pulmonary capillary bed and do not appear in the left side of the heart. In an intrapulmonary shunt, the microbubbles appear in the left-heart chambers 4 to 6 heart beats after their initial visualization in the right side of the heart [8].
 1. Diagnostic criteria for HPS [3,9]:
 2. Presence of liver disease: portal hypertension (most common) with or without cirrhosis.
 3. Oxygenation Defect: Partial pressure of O₂<80 mmHg or alveolar-arterial oxygen gradient P (A-a) O₂ ≥ 15 mmHg while breathing ambient air.
 4. Pulmonary vascular dilatation: Positive finding on contrast enhanced echocardiography.
 - Ethical Aspects:

This study was approved by the Ethical Committee of Assiut

University Hospital, and a written informed consent was obtained from all enrolled patients.

- Statistical analysis:

Statistical analysis was performed using Statistical Package for the Social Sciences (SPSS- version 17). The results were expressed as mean ± standard deviation. For statistical evaluation, Student T test was used to compare continuous variables for the 2 groups. Proportions were compared with chi-square tests. When the number of patients in the study groups is 5 or less, comparison of proportion is performed by Fisher's Exact Test. P values of less than 0.05 were considered significant. Graphics was performed using Microsoft excel.

Results

This study included 570 patients with liver cirrhosis (mean age was 53.80 ± 10.13 years; 382 (67.02%) were males and 188 (32.98%) were females) who were consecutively admitted in Tropical Medicine Department. The etiology of liver cirrhosis was hepatitis C virus (HCV) infection in 553 (97%) patients, hepatitis B infection (HBV) in 12 (2.1%) patients and HCV-HBV co-infection in 3 patients (0.5%), autoimmune hepatitis in one patient (0.2%) and Wilson disease in one patient (0.2%).

Among 570 patients with liver cirrhosis, 75 patients were found to have O₂ saturation less than 95% by pulse oxymeter. Sixty four of them had PaO₂ less than 80 mmHg. Among those 64 patients, only 24 cases met the diagnostic criteria of hepatopulmonary syndrome. Therefore, the prevalence of HPS in our study was 4.2% among total cirrhotic patients. However, among those (64 hypoxemic patients) underwent contrast echocardiography, the prevalence of HPS was 37.5% (24 out of 64 patients) (Figure 1). We compare cases with HPS (24 cases) with contrast echo negative cirrhotic cases (40 cases). Patients in these 2 groups were HCV related cirrhosis.

Patients with HPS were 15 males and 9 females (62.5%/37.5%) with mean age of 51.67±13.88 years. Patients with liver cirrhosis were 27 males and 13 females (67.5%/32.5%) with mean age 55.08 ± 8.22 years. Among patients with HPS, 15 individuals (62.5%) were non-smokers while 9 individuals (37.5%) were smokers (passive smoker or ex- smokers) while in cirrhotic patients, 16 individuals (40%) were non-smokers and 24 individuals (60%) were smokers (passive smoker or ex- smokers), (Table 1).

Most patients with HPS felt dyspneic (95.8%) whereas dyspnea was noted in 10 cirrhotic patients (25%) (P value<0.001), also the percentage

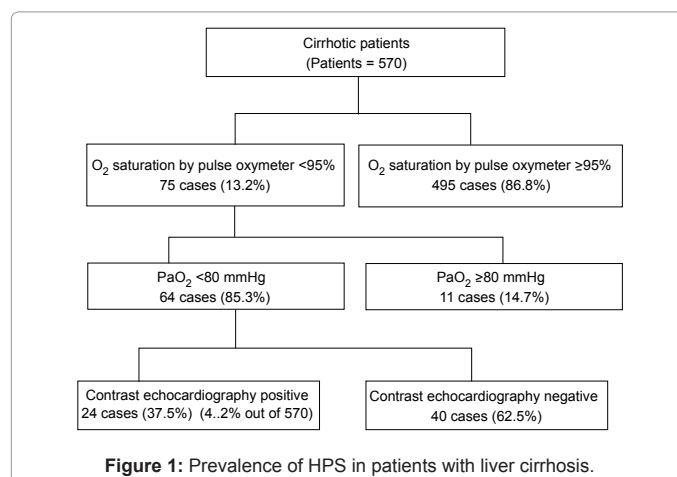


Figure 1: Prevalence of HPS in patients with liver cirrhosis.

of platypnea (91.7%) was significantly higher in HPS when compared to patients with liver cirrhosis (7.5%, P value<0.001). Clubbing was found in 14 patients with HPS (58.3%) while only 6 (15%) patients with liver cirrhosis had clubbing (P value<0.01). The percentage of orthodoxy (70.8%) was significantly higher in HPS when compared to patients with liver cirrhosis (5%) (P value<0.001), also, the percentage of encephalopathy (75%) was significantly higher in HPS when compared to patients with liver cirrhosis (47.5%) (P value<0.05). About 83.3% of patients with HPS were Child grade C, however only 57.5% patients with liver cirrhosis were Child grade C (<0.05). As regards, the presence of ascites and lower limb edema, there were no significant difference between patients with liver cirrhosis and patients with HPS (P value>0.05 for each). As regard the presence of portal hypertension, there was no significant difference between patients with liver cirrhosis and patients with HPS (P value>0.05) (Table 2).

In HPS, the presence of right pleural effusion was noticed in 5 patients (20.8%) also, bilateral basal shadows were noticed in 5 patients (20.8%). Raised right copula was present in one patient (4.2%) with HPS while the remaining 13 patients had no abnormality in chest X-ray (54.2%). However, chest X- ray of most patients with liver cirrhosis was normal (85%, P value<0.05) (Table 3).

The mean values of pH, PaO₂, PaCO₂, P (A-a) O₂, and O₂ saturation were recorded in (Table 4). The presence of hypoxemia and widening of alveolar arterial oxygen fraction was the presenting feature in HPS. There was a significant decrease in PaO₂ and O₂ saturation (P<0.001 for each) but a significant increase in P (A-a) O₂ in patients with HPS versus patients with liver cirrhosis (P<0.001). There was no significant difference in pH and PaCO₂ in HPS compared to patients with liver cirrhosis (P>0.05 for each).

Variable	Liver cirrhosis	HPS
Number	40	24
Male/female	27/13 (67.5%/32.5%)	15/9 (62.5%/37.5%)
Smoking*	24 (60%)	9 (37.5%)
Mean age± (yrs)	55.08 ± 8.22	51.67 ± 13.88

Data expressed as number (%) or mean ±SD

* Passive smoker or ex- smokers

Table 1: Demographic data of study groups.

Variable	Liver cirrhosis (n =40)	HPS (n =24)	P value*
Dyspnea	10 (25%)	23 (95.8%)	< 0.001
Platypnea	3 (7.5%)	22 (91.7%)	< 0.001
Cyanosis	11 (27.5%)	24 (100 %)	< 0.001
Clubbing	6 (15%)	14 (58.3%)	< 0.01
Lower limb odema	15 (37.5%)	14 (58.3%)	> 0.05
Orthodoxy	2 (5%)	17 (70.8%)	< 0.001
Encephalopathy	19 (47.5%)	18 (75%)	< 0.05
Mild to moderate	13 (32.5%)	7 (29.1%)	
Severe to coma	6 (15%)	11 (45.9%)	
Ascites	32 (80%)	16 (66.7%)	> 0.05
Mild	8 (20%)	3 (12.5%)	
Moderate	18 (45%)	11 (45.8%)	
Tense	6 (15%)	2 (8.4%)	
Child grade			< 0.05
A or B	17 (42.5%)	4 (16.7%) 20	
C	23 (57.5%)	(83.3%)	
Portal hypertension #	30 (75%)	21(87.5%)	> 0.05

* HPS versus liver cirrhosis Data expressed as number (%) # Portal hypertension was diagnosed by the presence of splenomegaly and esophageal varices

Table 2: Clinical features of study groups.

Variable	Liver cirrhosis (n =40)	HPS (n =24)	P Value
Abnormality	6 (15%)	11 (45.8%)	< 0.05
Right pleural effusion*	4 (10%)	5 (20.8%)	
Bilateral basal interstitial shadows	1 (2.5%)	5 (20.8%)	
Raised right copula	1 (2.5%)	1 (4.2%)	

Data expressed as number (%)

* Mild Pleural effusion

Table 3: Chest X ray of study groups.

Variable	Liver cirrhosis (n =40)	HPS (n =24)	P Value
pH	7.48 ± 0.05	7.47 ± 0.06	NS P > 0.05
PaO ₂	73.37 ± 6	49.70 ± 9.24	S P < 0.001
PaCO ₂	29.71 ± 4.07	29.72 ± 7.87	NS P > 0.05
P (A-a) O ₂	10.52 ± 6.13	53.64 ± 21.82	S P < 0.001
O ₂ saturation	93.43 ± 1.06	83.45 ± 8.48	S P < 0.001

Data expressed as mean ±SD

PaO₂, PaCO₂= arterial partial pressures of oxygen and carbon dioxide

P (A-a)O₂ = Alveolo-arterial oxygen gradient

S =significant

NS =non significant

Table 4: Arterial blood gases of study groups.

Variable	Liver cirrhosis (n =40)	HPS (n =24)	P Value
FEV1 L/S	1.94 ± 0.61	1.72 ± 0.67	NS P >0.05
FEV1 % predicted	71.2 ± 23.2	63.42 ± 18.66	NS P >0.05
FVC L/S	2.51 ± 0.75	2.13 ± 0.90	NS P >0.05
FVC % predicted	69.18 ± 17.88	61.46 ± 21.60	NS P >0.05
FEV1/FVC%	78.90 ± 8.17	82.92 ± 8.03	NS P >0.05
FEF 25-75 L/S	1.91 ± 0.81	1.67 ± 0.60	NS P >0.05
FEF25-75% predicted	61.88 ± 28.24	56.17 ± 15.79	NS P >0.05

Data expressed as mean ±SD

FEV₁ = forced expiratory volume in 1 s ; FVC= forced vital capacity; FEV1/FVC%

= Ratio of FEV1 /FVC ; FEF = Forced expiratory flow

S =significant

NS =non significant

Table 5: Pulmonary function tests of study groups.

Regarding pulmonary function tests, there was no significant difference between patients with HPS and patients with liver cirrhosis in all parameters of pulmonary function tests (P>0.05 for each) (Table 5). Patients with HPS showed a restrictive dysfunction in 59.3% (Figure 2).

The mean values of bilirubin, albumin, AST, ALT and prothrombin time were shown in (Table 6). There was no significant difference in serum bilirubin, albumin and ALT or prothrombin time in patients with HPS versus patients with liver cirrhosis (P>0.05 for each). However, there was a significant increase in AST in patients with HPS when compared with patients with liver cirrhosis (P<0.05).

As regard the echocardiographic finding, all patients with HPS had intrapulmonary shunt (100%) as shown in figure (3) and (4). Patients with HPS had more impairment of diastolic function than patients with liver cirrhosis (83.3% versus 40%) as shown in (Table 7).

Discussion

Hepatopulmonary syndrome is defined as the clinical triad of liver disease, arterial deoxygenation and intrapulmonary vascular dilatation [1,7]. The prevalence of the hepatopulmonary syndrome ranges from 5 to 32%. This wide range in prevalence is most probably due to

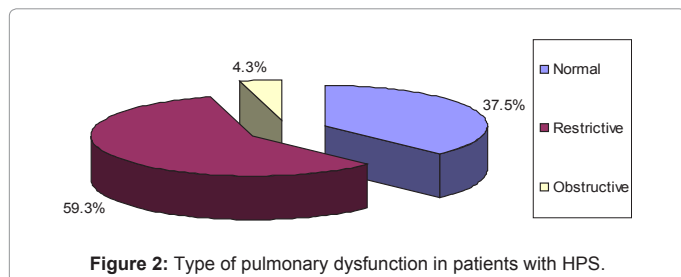


Figure 2: Type of pulmonary dysfunction in patients with HPS.

Variable	Liver cirrhosis (n =40)	HPS (n =24)	P Value
Bilirubin μ mol/l	58.78 \pm 51.61	77.54 \pm 73.36	NS P >0.05
Albumin g/L	23.20 \pm 6.91	23.65 \pm 5.14	NS P >0.05
AST U/L	68.24 \pm 40.51	106.54 \pm 85.07	S P <0.05
ALT U/L	64.94 \pm 33.39	86.26 \pm 168.53	NS P >0.05
Prothrombin time(Second)	19.15 \pm 6.52	21.22 \pm 5.45	NS P >0.05

Data expressed as mean \pm SD
S =significant
NS =non significant

Table 6: Liver function tests of study groups.

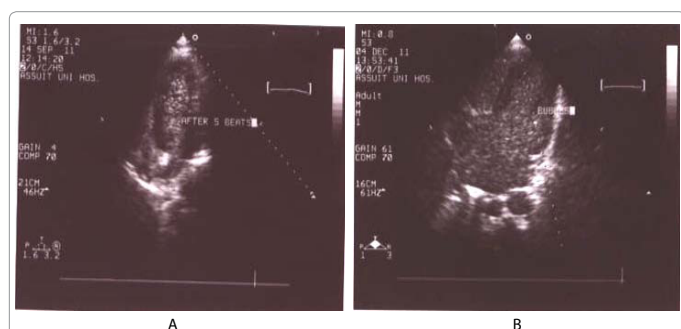


Figure 3: Intrapulmonary Shunt by contrast enhanced echocardiography. A. Contrast enhanced echocardiography in 49 year old man shows microbubbles appear in the left-heart chambers 5 heart beats after their initial visualization in the right side of the heart. B. Contrast enhanced echocardiography in 45 year old man shows microbubbles appear in the left-heart chambers.

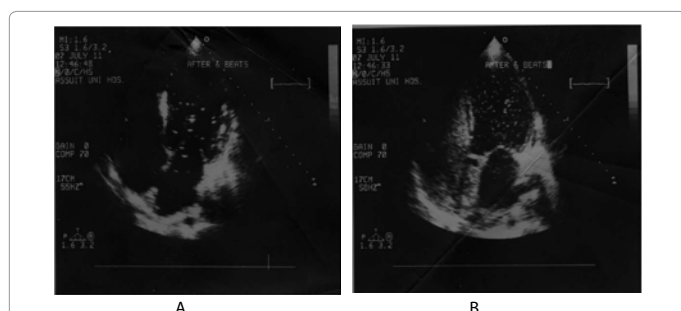


Figure 4: Intrapulmonary Shunt by contrast enhanced echocardiography. A. Contrast enhanced echocardiography in 50 year old man shows beginning of microbubbles in the left-heart chambers 6 heart beats after their initial visualization in the right side of the heart. B. Contrast enhanced echocardiography in the same 50 year old man show left-heart chambers full of microbubbles.

varying cutoffs for the partial pressure of arterial oxygen and abnormal alveolar-arterial gradient which are used to define gas-exchange abnormalities in HPS [3].

In the current study, the prevalence of HPS was 4.2% which is in

agreement with the study of Stoller et al. [4], where HPS was diagnosed in four out of 98 patients (4%). In some literatures the prevalence of HPS in the setting of cirrhosis ranges from 4% to 17% [7,8,10]. However, Vedrinne and colleagues found HPS in 8% of their cirrhotic patients [11]. Zakaria et al. [12] found that 6 patients (10%) met the suggested diagnostic criteria for HPS out of 60 portal hypertension and HCV induced cirrhosis Egyptian patients.

Our results were lower than that of Aller et al. [13] who reported that HPS is present in 61%. In the study of Aller et al. different formula for calculation of alveolar-arterial O_2 gradient may be the reason for the different prevalence values. Also, the contrast agent used may contribute to the different prevalence data [13]. Moreover, our results were lower than that of Schenk et al. [3] who reported that HPS is present in 34%. This could be explained by the higher rate of hypoxaemic patients in the study population of Schenk et al (43% of all patients had PaO_2 values <80 mmHg) compared with those in our study (11.2% of all patients had PaO_2 values <80 mm Hg). However, among 64 hypoxemic patients underwent contrast echocardiography in our study, the prevalence of HPS was 37.5% (24 out of 64 patients). This was in agreement with the study of Abu El Makarem et al. [14] (who reported that HPS was observed in 17 out of 50 patients (34%) underwent contrast echocardiography).

In the present study, most patients with HPS had dyspnea (95.8%) whereas dyspnea was noted in 10 cirrhotic patients (25%), also the percentage of platypnea (91.7%) was significantly higher in HPS when compared to cirrhotic patients with negative contrast enhanced echocardiography (7.5%). The frequent presence of dyspnea and platypnea in patients with HPS in comparison to patients with liver cirrhosis was in concordance with some series that reported high percentage of patients with "clinically significant" HPS felt dyspneic (57%) whereas dyspnoea was seldom noted in the patient groups without HPS (6%) [3]. Also, this data were in concordance with some literatures which reported that, dyspnea was the predominant presenting symptom of HPS [9,13,14].

Most patients with HPS were cyanosed while cyanosis was present in few patients with liver cirrhosis without HPS. This was attributed to the presence of intrapulmonary shunt and arterial hypoxemia in HPS. The presence of spider nevi, digital clubbing, cyanosis, and severe hypoxemia (partial pressure of oxygen, <60 mm Hg) strongly suggests hepatopulmonary syndrome [1,15].

Conflicting data exist in literatures regarding the correlation between HPS and the severity of liver disease. Some studies reported that there was no relationship between the presences or severity of the hepatopulmonary syndrome and the severity of liver disease as assessed on the basis of the Child-Turcotte-Pugh classification or the Model for End-Stage Liver Disease (MELD) [16]. Our study showed that, the prevalence of HPS increased with the severity of liver disease which agree with that of Vachiéry et al. [17] who found that hypoxaemic cirrhotics had a significantly higher Child-Pugh score. However our results disagree with those of Abrams et al. [18] who showed significantly lower PaO_2 values, higher P (A-a) O_2 values, and greater shunt fractions in Child-Pugh A cirrhosis compared with Child-Pugh

Variable	Liver cirrhosis (n =40)	HPS (n =24)	P Value
Shunt	0 (0%)	24 (100%)	< 0.001
Impaired diastolic function	16 (40%)	20 (83.3%)	< 0.001

Data expressed as number (%)

Table 7: Contrast enhanced Echocardiography.

B and C Classes. Also, our study is in concordance with that of Schenk et al [3] and Abu El Makarem et al. [14] who found a clear significant correlation between the severity of HPS and Child- Pugh score. Patients with HPS had more severe cirrhosis.

Although chest radiograph is normal in many patients, bilateral interstitial infiltrate is the frequent findings in HPS. This was compatible with that of Rodriguez-Roisin and Krowka [9] who reported that chest radiograph is frequently nonspecific, perhaps suggesting a mild interstitial pattern in the lower lung that may reflect the existence of diffuse pulmonary vascular dilatation. Also our results was in agreement with the study which found that chest radiograph showed interstitial markings, predominately in the lower lung fields, in seven (21%) and two (3%) patients with and without a positive contrast echocardiography, respectively ($p < 0.01$) [3]. Also, other series support our results and estimated that interstitial markings were observed significantly more often in contrast echo positive patients. They are typical of HPS and are predominately localized in the lower lung fields, and may reflect pulmonary vascular dilatations [19,20]. In our study, the difference in other radiological findings was not significant.

Since intrapulmonary vascular dilatation results in ventilation-perfusion mismatch, impaired oxygenation is the major clinical manifestation of HPS. Impaired oxygenation in HPS varies from a mild increase in the alveolar-arterial oxygen gradient to severe arterial hypoxemia [21]. In this study, all patients with HPS were hypoxemic with widening in the alveolar-arterial gradient and reduced arterial partial pressure of O_2 with statistically significant difference when compared with patients without HPS. This was supported by different series which reported that, the parameters of arterial oxygenation, PaO_2 and $P(A-a)O_2$, were highly significantly different with widened $P(A-a)O_2$ gradient and hypoxemia in the group with a positive contrast echocardiography. $PaCO_2$ was reduced due to hyperventilation, with no difference between the two groups [3,22,23].

Orthodexia is significantly higher in patients with HPS and may be related to ventilation-perfusion mismatch from pooling of blood in the dilated intrapulmonary vasculatures in the lung bases and in 30% of patients with cirrhosis, this blood flow is enhanced by the absence or impairment of hypoxic pulmonary vasoconstriction [7]. In this study orthodexia was present in 70.8% of patients which was in agreement with other series which reported prevalence ranges from 20% to 80% in patients with HPS [7,21]. So it is highly specific for HPS in the setting of liver cirrhosis.

In the current study, patients with HPS showed a restrictive dysfunction in about 60%, however there was no significant difference between patients with HPS and patients without in all parameters of pulmonary function tests. Moreover, only one patient with HPS (4.3%) showed a mid obstructive dysfunction despite the absence of chronic cough or chest X ray abnormality, this obstruction could be explained by the previous history of smoking and the use of fixed FEV1/FVC ratio in our locality to define air flow limitation may result in more frequent diagnosis of obstructive dysfunction in the elderly. These results were reported by some studies which found that lung function values (FEV1 and TLC) were slightly lower in cirrhotic patients with a positive contrast echocardiography compared with those with negative contrast echocardiograms [3,23]. Ventilatory restriction was noted in 25% of severe advanced liver disease and airflow obstruction in only 3% [24,25]. Honrani et al. [24] reported that, in patients with liver cirrhosis, decreased plasma osmotic pressure may cause a cuff of fluid surrounding the terminal airways. This may suggest early small airway abnormalities.

In the present study, there was no significant difference in serum bilirubin, albumin and ALT or prothrombin time in patients with HPS versus patients with liver cirrhosis. However, there was a significant increase in AST in patients with HPS when compared to patients with liver cirrhosis, which may signify more advanced stage of liver disease in HPS. Akhter et al. [26] reported that raised AST/ALT ratio is a useful parameter that can predict the severity of liver diseases. In contrast to this, some literatures found that the mean total bilirubin was significantly higher, and mean values for serum albumin, prothrombin time, and erythrocyte count were significantly lower in cirrhotic patients with a positive contrast echocardiography [3].

In our study, we used contrast-enhanced transthoracic echocardiography which is the most practical method and preferred screening test to detect pulmonary vascular dilatation [3,4,9]. In our study, contrast-enhanced transthoracic echocardiography was positive in all patients with HPS. Most studies investigating HPS have used transthoracic contrast echocardiography for the detection of intrapulmonary vasodilation and all reviews on HPS describe transthoracic contrast echocardiography as the method of assessment of intrapulmonary vasodilation apart from lung perfusion scan [22].

Conclusion

The prevalence of HPS among total cirrhotic patients was 4.2%. However, the prevalence of HPS among hypoxemic cirrhotic patients was 37.5%. Dyspnea, platypnea, clubbing, orthodexia and arterial hypoxemia were the commonest feature. Right pleural effusion and bilateral basal shadows were the commonest radiological findings.

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