Journal of Cardiovascular Disease Research

ISSN:0975 -3583,0976-2833 VOL 15, ISSUE 10, 2024

Original Research Article

PULMONARY HYPERTENSION ASSOCIATED WITH LIVER CIRRHOSIS- AN ECHOCARDIOGRAPHIC STUDY

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Received: 23-07-2024 / Revised: 01-08-2024 / Accepted: 10-09-2024

ABSTRACT

INTRODUCTION

Pulmonary hypertension is a well known complication of liver cirrhosis. The aim of present study is to evaluate pulmonary arterial pressure among patients of liver cirrhosis using echocardiography.

MATERIAL & METHODS

The prospective research was conducted among 175 liver cirrhosis patients suspicious of pulmonary hypertension admitted to department of internal medicine of a tertiary care centre during the study period of one year. The degree of liver disease in all individuals was evaluated using Child's classification & Pugh's score. All the patients were then subjected to 2D Echocardiography (PASP) >30mm Hg.

RESULTS

The most common etiology of liver cirrhosis was alcohol (60). 98 (56%) patients had stage A, 47 (26.8%) had stage B & 30 (17.2%) had stage C. Echographics signs were seen in 70 (40%) patients & endoscopic varices grade 1-2 was seen in 46 (51.1%) patients whereas grade 3-4 was seen in 44 (48.8%). In 20% patients PASP exceeded 30 mm Hg, 24% had between 16-29 mmHg & 56% had below 15 mm Hg. Prevalence of pulmonary hypertension was 20%.

CONCLUSION

The echocardiographic noninvasive method for assessing & monitoring PAP is advantageous for cirrhotic patients with well-compensated liver disease.

KEYWORDS

Echocardiography, Liver Cirrhosis, Portal Hypertension, Pulmonary Hypertension.

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INTRODUCTION

Liver cirrhosis may correlate with hemodynamic alterations in the pulmonary vasculature. Two clinically significant disorders have been identified: Portopulmonary hypertension (PPH) & hepatopulmonary syndrome (HPS).[1-3] The two entities are pathophysiologically & clinically diverse; specifically, HPS is marked by vasodilation & gas-exchange impairment leading to severe hypoxemia, while PPH is defined by vasoconstriction & hemodynamic compromise resulting in right heart failure. HPS is a somewhat prevalent illness, affecting roughly 30% of cirrhotic individuals with advanced disease, PPH appears to be quite uncommon.[1-3]

Portopulmonary hypertension (PoPH) is characterized by the onset of PAH associated with portal hypertension, with or without the presence of severe hepatic illness.[4-7] The splanchnic vessels produces vasoconstrictive chemicals, such as endothelin, which are then transported to the pulmonary arteries via porto-systemic shunts. It is hypothesised that pulmonary hypertension results from decreased hepatic clearance of these molecules. Increased shear stresses in the pulmonary artery caused by a hyperdynamic circulatory condition in cirrhosis patients result in vascular remodelling & pulmonary arterial hypertension. (PAH).[8]

PH was initially documented in 0.25% to 0.75% of individuals with portal hypertension, albeit these estimates were derived solely from retrospective, clinical, or postmortem research.[9-12] Subsequently, hemodynamic investigations of systemic & splanchnic circulation in individuals with portal hypertension indicated a heightened incidence of pulmonary hypertension, approximately 2%.[13,14] Recently, increased PAP values (mean > 25 mm Hg) have been reported as a very prevalent occurrence in cirrhotic patients with advanced liver disease, which has clinical implications for OLT. -20%.[15]

Primary biliary cirrhosis without portal hypertension, extrahepatic portal hypertension, & non-advanced liver disease (such as chronic hepatitis) have also been linked to elevated pulmonary arterial pressure.[16-18] The aim of present study is to evaluate pulmonary arterial pressure among patients of liver cirrhosis using echocardiography.

MATERIAL & METHODS

The current prospective study was carried out over the course of a year among patients with liver cirrhosis who were admitted to the internal medicine department of a tertiary care facility. Before the study started, institutional ethics committee approval was obtained. Patients were given a thorough explanation of the study's methodology before being requested to sign an informed consent form.

Through consecutive sampling a total of 175 patients of liver cirrhosis suspicious of pulmonary hypertension were selected on the basis of inclusion & exclusion criteria.

InclusionCriteria

1. Patients with age above 18 years.
2. Patients diagnosed with liver cirrhosis (Ultrasonogram (USG) revealing altered echotexture & reduced liver size) & exhibiting portal hypertension (evidenced by at least two of the following: portal vein diameter exceeding 13 mm, ascites with splenomegaly, esophageal varices or portosystemic collateral venous communications on Ultrasonogram identified during Upper GI endoscopy)
3. Patients willing to participate in the study.

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Exclusion Criteria

1. Patients with age less than 18 years.
2. Patients with cirrhosis who also have cardiovascular &/or pulmonary diseases, as well as other medical illnesses such PE, collagen vascular disease, schistosomiasis, human immunodeficiency virus, & oral appetite suppressants that may be linked to pulmonary hypertension.
3. Individuals with severe malnourishment, III-IV grade encephalopathy, & liver cirrhosis linked to hepatocellular cancer were excluded. Wilson's disease, hereditary hemochromatosis, primary biliary cirrhosis, & primary sclerosing cholangitis.
4. Patients not willing to participate in the study.

The degree of liver disease in all individuals was evaluated using Child's classification & Pugh's score. People with liver disease had their pulmonary hypertension evaluated clinically, ultrasonographically, & endoscopically. After fasting for the whole night, each participant was examined in the morning. Doppler ultrasonography was used to perform a thorough evaluation of the liver, spleen, portal, & splanchnic veins in order to evaluate the blood flow in the portal vein, the hepatic & superior mesenteric arteries, collateral veins, spontaneous shunts, & the lack of clinically detectable ascitic fluid. Every patient received a routine fibroscopy endoscopic examination, & Dagradi's classification was used to grade the esophageal varices. The diagnosis of pulmonary hypertension was made using specific endoscopic &/or echographic markers. Serum HBV indicators & alpha-fetoprotein were assessed via radioimmunoassay, HCV antibodies were evaluated using Western blot analysis, & liver function tests were conducted through automated routine procedures.

After that, 2D echocardiography was performed on each patient (pulmonary hypertension is defined as pulmonary systolic artery pressure (PASP) > 30 mm Hg at rest).[19]

Data was exported to SPSS version 25.0 after being entered into Microsoft Excel. Descriptive statistics, such as mean values, standard deviation, & percentages, were used to analyse the data. The relationship between qualitative variables was evaluated using the Chi-Square test. A statistically significant p-value is one that is less than 0.05.

RESULTS

Maximum number of patients were in the age group of 31-40 years (65), followed by 41-50 years (40), <30 years (25), 51-60 years (20), 61-70 years (10) & >70 years (5) as shown in figure 1.

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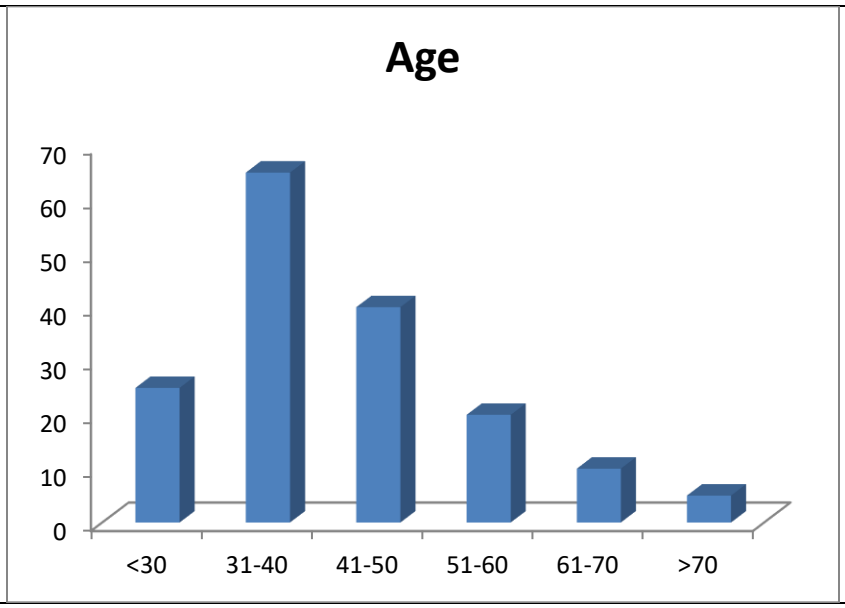

Figure 1: Distribution of patients on the basis of age

Males (105) were higher in number as compared to females (70) as shown in figure 2.

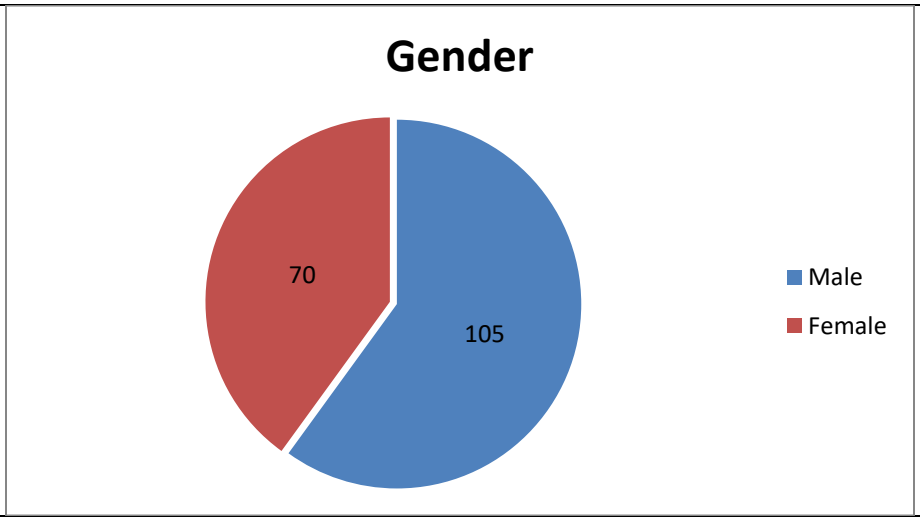

Figure 2 Distribution of patients on the basis of gender

The most common etiology of liver cirrhosis was Alcohol (60) followed by HBV (37), HCV (35), HCV+ alcohol (25) & cryptogenic (18) as shown in figure 3.

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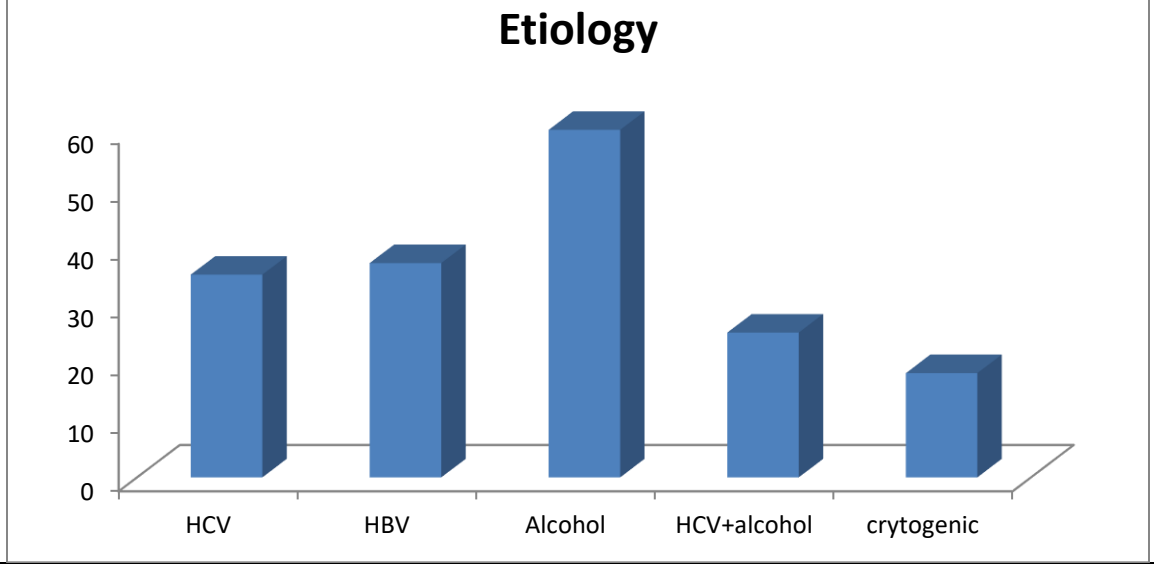

Figure 3: Etiology of liver cirrhosis

Out of all the 175 patients 98 (56%) patients had stage A, 47 (26.8%) had stage B & 30 (17.2%) had stage C as shown in table 1.

Child Stage	N (%)
A	98 (56)
B	47 (26.8)
C	30 (17.2)

Table 1 Distribution of patients on the basis of Child's classification & Pugh's score

Echographics signs were seen in 70 (40%) patients & endoscopic varices grade 1-2 was seen in 46 (51.1%) patients whereas grade 3-4 was seen in 44 (48.8%) patients as seen in table 2.

Characteristics		N (%)
Echographic signs		70 (40)
Endoscopic varices	Grade 1-2	46 (51.1)
	Grade 3-4	44 (48.8)

Table 2 Characteristics of portal hypertension

The Doppler echocardiographic findings of the right heart & pulmonary artery systolic pressure in cirrhotic patients are shown in table 3. In 35 out of 175 patients (20%), pulmonary artery systolic pressure (PASP) exceeded 30 mm Hg, indicating the presence of pulmonary hypertension. In 42 patients (24%), the pulmonary pressure levels exceeded 15 but remained below 30 mm Hg. The pulmonary systolic pressure was less than 15 mm Hg in 98 individuals (56%). The right ventricle & right atrium (both longitudinal & transverse diameters) were statistically significantly larger in liver disease patients with elevated PASP, & the E/A ratio was reversed in cirrhotic patients without echocardiographic signs of pulmonary hypertension ($p < 0.05$).

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Table 3 Right Heart Doppler Echocardiographic Findings & Pulmonary Artery Systolic Pressure in patients

PASP (mmHg)	N (%)	RVDD (mm)	RALD (mm)	RATD (mm)	E/A
≤15	98 (56)	22±2.9	36±4.3	34±4.1	0.89±0.1
16-29	42 (24)	27±3.1	38±2.5	38±2.1	0.81±0.4
≥30	35 (20)	33±5.2	44±3.1	41±2.8	0.63±0.2

Table 3: Patients' right heart Doppler echocardiogram results & pulmonary artery systolic pressure

Patients with PASP ≥30 mmHg were considered as having pulmonary hypertension 35 (20%) as shown in figure 4.

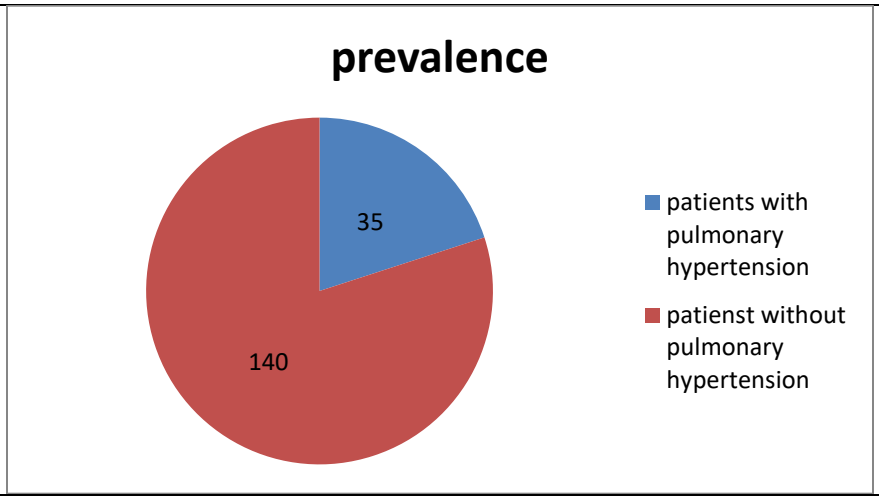

Figure 4: Prevalence of pulmonary hypertension

As indicated in Table 4, the mean PASP for patients undergoing 2D ECHO to detect pulmonary hypertension was 26.56±10.2 in Class A, 25.89±11.1 in Class B, & 29.34±9.0 in Class C.

Child stage	Mean ± SD PASP (mmHg)
A	26.56±10.2
B	25.89±11.1
C	29.34±9.0

Table 4: Patients' mean PASP distribution according to CHILD PUGH stage

DISCUSSION

Vascular alterations in the pulmonary circulation are a well-documented consequence of liver cirrhosis. Patients with cirrhosis & pulmonary arterial hypertension (PAH) may exhibit modest & subclinical cardiac abnormalities. These alterations may result in systolic & diastolic dysfunction, which can be identified using echocardiography.[20]The present prospective study was conducted among 175 liver cirrhosis patients to evaluate pulmonary arterial pressure among using echocardiography. Based on the clinical severity of their liver illness & whether or not they

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had endoscopic or ultrasonographic evidence of portal hypertension, the patients were evaluated using the Child's categorization.

Most of the patients were male with age group between 31-40 years this aligns with a study conducted in Denmark, which indicated a higher incidence of cirrhosis among men[21] in our study the most common etiology was alcohol which was similar to study conducted in Iceland which showed that alcoholic fatty liver disease had a higher mortality & a worse survival compared to other aetiological factors.[22]

In our study the prevalence of pulmonary hypertension is found to be 20%. The data provided herein, utilizing a noninvasive methodology, indicate a high prevalence (-20%) of PPH, comparable to the proportion obtained through catheterization in chosen patients with advanced liver disease who are candidates for liver transplantation.[3] The incidence of pulmonary hypertension in cirrhotic patients varies between 2% & 20%, according to multiple investigations conducted via right cardiac catheterization. The significant diversity underscores the necessity for this investigation.[23,24] The heterogeneity among the study sample & the discordance regarding the diagnosis of portopulmonary hypertension (PPH) may account for these disparities. According to PPH, there are at least three causes: (1) a high flow phenomenon brought on by cirrhosis's hyperdynamic circulation; (2) a volume phenomenon connected to possible cirrhosis cardiac symptoms; & (3) vasoconstriction brought on by a variety of medial hypertrophy, intimal fibrosis, & classic plexogenic arteriopathy.[25] The prevalence of pulmonary hypertension is reduced to 1% to 0.2% when only the third description is included.[15]

Pulmonary hypertension is more common in patients with more severe liver damage & portal hypertension; in particular, the incidence of pulmonary hypertension increased from Child's class A to class C & from patients without portal hypertension to those with evidence of portal hypertension. Thirty percent of pulmonary hypertension patients were categorized as Class A, fifty-two percent as Class B, & nineteen percent as Class C in the study by Gurgheanet al. [23].In this investigation, where 64% of patients were categorized as Child-Pugh B & C, the odds ratio for developing pulmonary hypertension compared to those in class A was statistically negligible (p=0.36), but the severity of hepatic disease may be a determining factor. According to the Child-Pugh method, Auletta M. et al. discovered that patients with more severe liver disease had a higher frequency of portal hypertension..[26]The observations support the concept that ultrasonographic examination in non-decompensated cirrhotic patients can facilitate the early identification of elevated pulmonary pressure. Increased pulmonary pressure may be linked to liver illness in the absence of portal hypertension, despite the definition of PPH inherently suggesting elevated portal pressure.[17,18] This study, along with prior publications, indicates that pulmonary hypertension escalates with the length of liver disease & portal hypertension; however, no definitive correlation with the severity of portal hypertension, hepatic failure, or volume of blood shunted has been established.[13]

An echocardiographic assessment may overstate pulmonary artery pressure; nonetheless, a strong association between right ventricular systolic pressure (RVSP) measured by echocardiogram & pulmonary artery systolic pressure (PASP) obtained via pulmonary artery catheterization has recently been established in candidates for liver transplantation. Conversely, the mechanism of pulmonary vascular disorders in liver patients (HPS & PPH) remains unclear; the interplay between vasoconstrictors & vasodilators, along with the potential influence of growth factors, generates numerous intricate ideas. Likewise, less information exists regarding the clinical progression of these two illnesses & the occurrences affecting people with HPS & PPH.[26]

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CONCLUSION

In conclusion, cirrhotic patients with well-compensated liver disease may benefit from the availability of an echocardiographic, noninvasive technique for detecting & tracking pulmonary arterial pressure. Due to ethical considerations, an invasive investigation cannot be advised for these participants; however, echocardiographic follow-up may reveal certain features of the clinical progression of pulmonary hypertension associated with liver cirrhosis, with catheterization studies reserved for select, more advanced patients.

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