



“PREVALENCE OF INTRAPULMONARY SHUNTS IN PATIENTS OF LIVER FAILURE POSTED FOR LIVER TRANSPLANTATION AT A TERTIARY CARE CENTRE IN RAJASTHAN”

Cardiology

Dr. Aniruddh Pratap Singh

DM Resident, Department of Cardiology, Mahatma Gandhi Medical College, Jaipur

Dr. Bhartesh Sethiya*

DM Resident, Department of Cardiology, Mahatma Gandhi Medical College, Jaipur

*Corresponding Author

Dr. Manas Thakur

DM Resident, Department of Cardiology, Mahatma Gandhi Medical College, Jaipur

Dr. Parth Mashru

DM Resident, Department of Cardiology, Mahatma Gandhi Medical College, Jaipur

Dr. Deepesh Agarwal

Professor, Department of Cardiology, Mahatma Gandhi Medical College, Jaipur

Dr. Harshwardhan

HOD, Department of Cardiology, Mahatma Gandhi Medical College, Jaipur

Dr. Tushar Wadhawan

DM Resident, Department of Cardiology, Mahatma Gandhi Medical College, Jaipur

ABSTRACT

A serious and very common pneumonic vascular intricacy of liver sickness is hepatopulmonary condition (HPS). It is a ternion of liver dysfunction and/or portal hypertension, intrapulmonary vascular dilatations, and raised alveolar-arterial oxygen slope. Prevalence varies according to different studies from 4%-47%. While the most well-known presenting complaint of HPS is dyspnoea, it is generally asymptomatic, and in this manner, all liver transfer applicants ought to be evaluated for its presence. Microbubble transthoracic echocardiography is very sensitive for diagnosis. Microbubble contrast transthoracic echocardiography is an exceptionally simple, safe, painless, and extremely successful strategy for the finding of HPS. The prognosis of HPS is not easily predictable, and there is presently no powerful clinical treatment. The main successful treatment is viewed as liver transplantation. In this study, we assess the burden of HPS, analysed by Microbubble contrast transthoracic echocardiography, during the screening of chronic liver failure patients posted for a liver transplant, over the most recent 3 years in a tertiary consideration emergency clinic in Rajasthan. **Materials And Methods:** All adult patients who were posted for liver transplant at Mahatma Gandhi Hospital, Jaipur in previous 3 years will be included in the study. Bubble contrast echocardiography of all these patients will be collected and prevalence of intrapulmonary shunting will be calculated.

KEYWORDS

Hepatopulmonary syndrome; Liver transplantation; microbubble transthoracic echocardiography.

INTRODUCTION

There are three fundamental pneumonic circumstances in patients with liver diseases as well as portal hypertension. These are hepatopulmonary disorder (HPS), portopulmonary hypertension (PoPH), and hepatic hydrothorax. Portopulmonary hypertension is a pulmonary hypertension that occurs because of portal hypertension regardless of liver problem. Hepatic hydrothorax is a pleural effusion in a cirrhotic patient without cardiovascular, pulmonary, or pleural illness. HPS is brought about by hyperdynamic flow, intrapulmonary shunts, and pulmonary vasodilatation. It is a set of three of liver dysfunction or potentially portal hypertension, intrapulmonary vascular dilatations, and expanded alveolar-blood vessel oxygen slope (> 15 mm Hg or > 20 mmHg when age > 65 years).¹ Fluckiger previously portrayed a connection among HPS and cirrhosis on a female patient with cyanosis and cirrhosis in 1884, but Kennedy and Knudson named the circumstance as "Hepatopulmonary Disorder" in 1977.² HPS is normally connected with portal hypertension regardless of cirrhosis, however it can happen likewise in patients with acute or chronic hepatitis,^{3,5} hypoxic hepatitis,⁶ extrahepatic obstruction, Budd-Chiari Syndrome,⁷ and Cavo pulmonary shunts.⁸ The occurrence of HPS is higher than PoPH, and the two of them are related with fundamentally raised morbidity and mortality. Their pathophysiologies, be that as it may, are totally unique. HPS is portrayed by vasodilatation and intrapulmonary shunts, while PoPH is brought about by pulmonary vasoconstriction and raised pulmonary vascular resistance (PVR). Regardless of this distinction, both can be visible in a same patient, with PoPH developing commonly after HPS.^{9,10} One hypothesis for this system includes dysregulation of the normal vascular flagging pathway for pneumonic dilatations and pulmonary arterial remodeling.¹⁰ Another hypothesis includes contrasts in the outflow of the endothelin-1 (ET1) receptor A, which causes vasoconstriction and raised PVR, and receptor B, which prompts upregulation of endogenous nitric oxide synthase and expanded nitric oxide creation, coming about in pulmonary vasodilation.¹¹ Clinical treatment can't fix these circumstances, so both have serious ramifications for liver transplantation (LT). After LT, HPS

typically settles, however, the reaction of PoPH is not predictable.¹ All patients on the LT waiting list ought to be evaluated for HPS and PPH on the grounds that both are routinely asymptomatic. In this survey, we give an outline of the predominance of HPS in the beyond 3 years in a tertiary consideration clinic in Rajasthan.

At the point when there is a patient with liver illness who shows clinical signs of HPS, blood vessel oxygenation, and pneumonic dilatation ought to be surveyed. Pulmonary function tests and imaging ought to be done to research associative lung sickness since HPS can coincide with other pneumonic or heart infections and compound side effects and hypoxemia.¹² HPS is typically asymptomatic, so all liver transplant centers ought to regularly evaluate patients for HPS.

Microbubble transthoracic echocardiography (MTTE), atomic lung filtering (99m-Techetium-marked full-scale totaled egg whites examination, MAA check), and seldom pulmonary angiography and high-resolution computed tomography (CT) can be utilized for the location of IPVD. MTTE is considered for the diagnosis of HPS.^{1,13,14} For MTTE, agitated saline with microbubbles with a diameter > 10 mm are infused into the peripheral vein in the arm.^{15,16} Ordinarily, these microbubbles are consumed by alveoli and caught by the pulmonary capillary bed (8-15 mm). In HPS, bubbles can't remain in the lung, and they cross over to the lung flow and enter the left atrium. If microbubbles are found in the left atrium in three to six cardiovascular cycles after they are found in the right atrium, it is characteristic of intrapulmonary shunting; but in the event that it occurs inside three heart cycles, this demonstrates intracardiac shunting. In our study we compiled the data of all the patients undergoing liver transplants in our institute in the past 3 years and collected data on microbubble transthoracic echocardiography in those patients. We then calculated the prevalence of intra-pulmonary shunting in these patients with specific distribution according to the age and sex of the patients.

DISCUSSION AND RESULTS

Total number of patients in our study group were 161. These all were posted for liver transplantation in our institute in the past 3 years. Microbubble transthoracic echocardiography was done in all patients prior to the liver transplant.

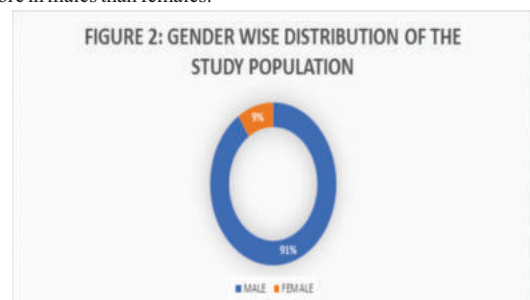
1. Age Wise Distribution Of Study Population:

In our study group, out of total 161 patients, 144 were of the age <60, and 17 were of the age ≥ 60 years. This suggests that majority of liver failure patients are of the younger age group.



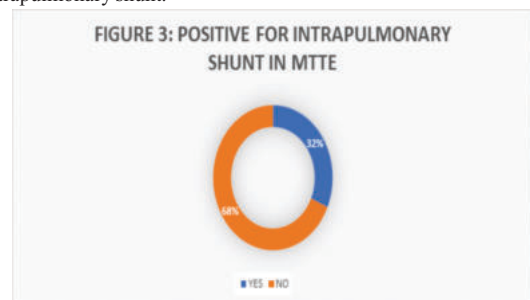
2. Gender Wise Distribution Of The Study Population:

In our study group, out of total 161 patients, 146 were males, and 15 were female patients. This suggests that prevalence of liver failure is more in males than females.



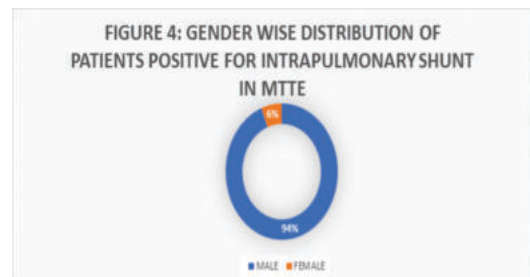
3. Positive For Intrapulmonary Shunting In Mtte:

In our study group, out of a total 161 patients, 51 patients, i.e., 32 % of the total study population, had positive MTTE suggestive of intrapulmonary shunt.



4. Gender Wise Distribution Of Patients Positive For Intrapulmonary Shunting In Mtte:

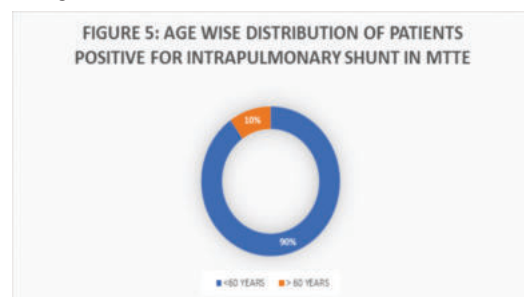
Out of 51 patients, positive for intrapulmonary shunt in MTTE, 48 (94%) were males and only 3(6%) were females. This suggests that the prevalence of intrapulmonary shunt is much higher in males than females.



1. Age Wise Distribution Of Patients Positive For Intrapulmonary Shunting In Mtte:

Out of 51 patients, positive for intrapulmonary shunt in MTTE, 46 (90%) patients were <60 years of age, and 5 (10%) patients were > 60

years of age.



Summary

- In our study group, out of total 161 patients, 144 were of the age <60, and 17 were of the age ≥ 60 years. This suggests that majority of liver failure patients are of the younger age group.
- In our study group, out of total 161 patients, 146 were males, and 15 were female patients. This suggests that prevalence of liver failure is more in males than females.
- In our study group, out of a total of 161 patients, 51 patients, i.e., 32 % of the total study population, had positive MTTE suggestive of intrapulmonary shunt.
- Out of 51 patients, positive for intrapulmonary shunt in MTTE, 48 (94%) were males and only 3(6%) were females.
- Out of 51 patients, positive for intrapulmonary shunt in MTTE, 46 (90%) patients were <60 years of age, and 5 (10%) patients were ≥ 60 years of age.

CONCLUSION

Our study suggests that there is higher incidence of positive MTTE suggesting HPS in younger population than in older population. Also, liver failure as well as HPS is much more common in males than in females. HPS is a severe pulmonary vascular complication of liver disease. As it is usually asymptomatic, all patients should be screened for its presence prior to LT since it can affect survival after LT. There is no medical therapy described to date, and LT is considered to be the only effective therapy. Complete resolution of HPS after LT is usually seen within a year in most HPS patients. Further follow up study of post LT patients must be done to see whether MTTE returns to normal after 1 year of LT or not.

Abbreviations:

ABG, blood vessel blood gas; CBDL, normal bile pipe ligation; CO, carbon monoxide; CT, figured tomography; ET, endothelin; HPS, hepatopulmonary disorder; HO, heme oxygenase; iNOS, inducible NOS; IPVD, intrapulmonary vascular enlargement; L-NAME, N(G)-nitro-L-arginine methyl ester; LT, liver transplantation; MAA examine, full scale amassed egg whites filter; Merge, model endstage liver sickness; MTTE, microbubble transthoracic echocardiography; NO, nitric oxide; NOS, NO synthase; PoPH, portopulmonary hypertension; PVR, pulmonary vascular resistance; TEE, transesophageal echocardiogram;

REFERENCES

- Rodriguez-Roisin R, Krowka MJ, Herve P, Fallon MB. Pulmonary-hepatic vascular Disorders (PHD). Eur Respir J 2004;24:861-880. doi: 10.1183/09031936.04.00010904.
- Rodriguez-Roisin R, Agusti AG, Roca J. The hepatopulmonary syndrome: newname, old complexities. Thorax 1992;47:897-902. doi: 10.1136/thx.47.11.897.
- De BK, Sen S, Sanyal R. Hepatopulmonary syndrome in non-cirrhotic portal hypertension. Ann Intern Med 2000;132:924. doi: 10.7326/0003-4819-132-11-200006060-00023.
- Regev A, Yeshurun M, Rodriguez M, Sagie A, Neff GW, Molina EG, et al. Transient hepatopulmonary syndrome in a patient with acute hepatitis A. J Viral Hepat 2001;8:83-86. doi: 10.1046/j.1365-2893.2001.00270.x.
- Teuber G, Teupe C, Dietrich CF, Caspary WF, Buhl R, Zeuzem S. Pulmonary dysfunction in non-cirrhotic patients with chronic viral hepatitis. Eur J Intern Med 2002;13:311-318. doi: 10.1016/S0953-6205(02)00066-3.
- Fuhrmann V, Madl C, Mueller C, Holzinger U, Kitzberger R, Funk GC, et al. Hepatopulmonary syndrome in patients with hypoxic hepatitis. Gastroenterology 2006;131:69-75. doi: 10.1053/j.gastro.2006.04.014.
- De BK, Sen S, Biswas PK, Sanyal R, Majumdar D, Biswas J. Hepatopulmonary syndrome in inferior vena cava obstruction responding to cavoplasty. Gastroenterology 2000;118:192-196. doi: 10.1016/S0016-5085(00)70428-8.
- Tercier S, Delarue A, Rouault F, Roman C, Breaud J, Petit P. Congenital portocaval fistula associated with hepatopulmonary syndrome: ligation vs liver transplantation. J Pediatr Surg 2006;41:e1-e3. doi: 10.1016/j.jpedsurg.2005.10.071.
- Ioachimescu OC, Mehta AC, Stoller JK. Hepatopulmonary syndrome following portopulmonary hypertension. Eur Respir J 2007;29:1277-1280. doi: 10.1183/09031936.00140306.
- Zopey R, Susanto I, Barjaktarevic I, Wang T. Transition from hepatopulmonary syndrome to portopulmonary hypertension: A case series of 3 patients. Case Rep Pulmonol 2013;2013:561870. doi: 10.1155/2013/561870.
- Shao D, Park JE, Wort SJ. The role of endothelin-1 in the pathogenesis of pulmonary

- arterial hypertension. *Pharmacol Res* 2011;63:504–511. doi: 10.1016/j.phrs. 2011.03.003.
12. Krowka MJ, Wiseman GA, Burnett OL, Spivey JR, Therneau S, Poraykoet MK, et al. Hepatopulmonary syndrome: a prospective study of relationships between severity of liver disease, Pa,O₂ response to 100% oxygen, and brain uptake after 99 mTc MAA lung scanning. *Chest* 2000;118:615–624. doi: 10.1378/chest.118.3.615.
 13. Abrams GA, Jaffe CC, Hoffer PB, Binder HJ, Fallon MB. Diagnostic utility of contrast echocardiography and lung perfusion scan in patients with hepatopulmonary syndrome. *Gastroenterology* 1995;109:1283–1288. doi: 10. 1016/0016-5085(95)90589-8.
 14. Rollan MJ, Munoz AC, Perez T, Bratos JL. Value of contrast echocardiography for the diagnosis of hepatopulmonary syndrome. *Eur J Echocardiogr* 2007; 8:408–410. doi: 10.1016/j.euje.2006.07.005.
 15. Mimidis KP, Vassilakos PI, Mastorakou AN, Spiropoulos KV, Lambropoulou Karatza CA, Thomopoulos KC, et al. Evaluation of contrast echocardiography and lung perfusion scan in detecting intrapulmonary vascular dilatation in normoxic patients with early liver cirrhosis. *Hepatogastroenterology* 1998;45:2303–2307.
 16. Aller R, Moya JL, Moreira V, Garcia-Lledo A, Sanromán AL, Pains C, et al. Diagnosis and grading of intrapulmonary vascular dilatation in cirrhotic patients with contrast transesophageal echocardiography. *J Hepatol* 1999; 31:1044–1052. doi: 10.1016/S0168-8278(99)80317-1.