Prevalence of Portal Hypertension in Patients with Group 1 and 4 Pulmonary Hypertension

Jeffery Turley MD, Jacob Newman DO, Justin Canakis DO, Lama Raddaoui MD, Jeremy Payano MS, Jalil Ahari MD, Ameer Abutaleb MD, Mardi Gomberg-Maitland MD
George Washington University Hospital, Washington, DC

Background

Pulmonary hypertension (PH) increases the risk of liver dysfunction secondary to congestive hepatopathy. Markers of liver dysfunction include thrombocytopenia, transaminitis, and direct bilirubinemia.

Objective

This study aims to define the prevalence of PHT in patients with Group 1 and 4 PH.

Methods

A case control study from a single tertiary institution analyzed 69 patients with a confirmed diagnosis of Group 1 or 4 PH. To assess PHT, the primary outcome measure was a platelet count < 150ug/ml. Secondary outcome measures included AST to platelet ratio index (APRI) scoring as marker of PHT. Patients with missing data on any of the key variables, current decompensated heart failure, or prior diagnoses of other hematologic or hepatic diseases were excluded from analyses. Fisher's exact tests were employed to assess the association between PH and PHT, with further stratification by PH pharmacologic treatment class.

Results

Table 1: Baseline Characteristics

Variable	(Mean, SD)	(n, %)
Sex		
Male		(23, 32.9%)
Female		(47, 67.1%)
Age	(57.3, 15.03)	
Race		
Asian		(1, 1.4%)
Black		(35, 50.0%)
Hispanic		(4, 5.7%)
Unknown		(3, 4.3%)
White		(27, 38.6%)
Platelet	(229.2, 76.59)	
<150		(10, 14.9%)
≥150		(59, 85.1%)
APRI	(.354, .634)	
APRI ≥.7		(5, 7.5%)
APRI ≤.5		(64, 88.1%)
FIB-4	(2.10, 4.38)	
<1.45		(38, 55.2%)
1.34-3.25		(26, 38.9%
>3.25		(4, 6.0%)

- 10 patients had a platelet count < 150ug/ml (OR 0.836, CI 0.163-5.648).
- 3 patients had significant fibrosis without a platelet count < 150ug/ml.
- 5 patients had significant fibrosis by APRI scores > 0.7 (OR 4.569, CI 0.477-59.578) (Table 1).
- There was no significant effect on platelet level dependent on PH therapy (Table 2).

Table 2: Prevalence of Portal Hypertension by Pulmonary Hypertension Pharmacologic Treatment Class

Class	Prevalence	Odds Ratio	p-value
Endothelin Receptor Antagonists	(36, 51.4%)	1.106	1.000
Prostacyclin/Prostanoid Agonists	(20, 28.6%)	0.945	1.000
Phosphodiesterase Inhibitors/Soluble GMP Stimulators	(60, 85.7%)	0.621	1.000

Conclusion

Among individuals with Group 1 or 4 PH, there were no statistically significant differences in the odds of developing PHT based upon platelet count and APRI scores. PH pharmacologic treatment class had no significant effect on the prevalence of PHT. Larger-scale studies, including transient elastography, are required to understand the link between PH and PHT.

Acknowledgements

We would like to acknowledge The George Washington University Hospital and The GW Medical Faculty Associates.

Contact: Jeffery Turley (jeffturleyjr@gwu.edu)