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Editorial

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Issues on Estimated Pulmonary Artery Pressure in Liver Transplant Candidates

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▸ See the article "Correlation between Echocardiographic Pulmonary Artery Pressure Estimates and Right Heart Catheterization Measurement in Liver Transplant Candidates" in volume 26 on page 75.

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Conflict of Interest
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Estimating pulmonary artery systolic pressure (PASP) by Doppler echocardiography has been widely used in clinical practice since it is known to be correlated with invasively measured PASP, easy to perform, and stratifies a clinical outcome in various cardiovascular disease.[1-4] Importantly, PASP changes according to left ventricular filling pressure, stroke volume and pulmonary vascular resistance.[1,2] Therefore, it has dynamic nature based on the loading conditions at the time of Doppler evaluation or exercise.

In patients with advanced chronic liver disease, who are candidates for liver transplantation, pulmonary vascular disorders including portopulmonary hypertension (PoPHT) are common.[5] Because PoPHT has prognostic significance, echocardiography is mandatory in the evaluation of patients with advanced liver disease prior to liver transplant even when the patients are asymptomatic.[6] Although right heart catheterization is the gold standard for diagnosis of pulmonary hypertension, it has been used in selected cases to confirm or grade pulmonary hypertension. Particularly, patients with advanced liver disease commonly have low platelet counts and impaired immune system therefore are prone to have procedure related complications including hematoma or infection. Therefore, understanding of diagnostic performance of Doppler estimated PASP for diagnosing pulmonary hypertension is crucial.

In this issue of the Journal, Habash et al.[7] report the results of their study of 31 patients being evaluated for liver transplant compared with control subjects without liver disease. They analysed Doppler-estimated PASP and invasive PASP directly measured by right heart catheterization. They demonstrated that the correlation between two parameters was only modest and an estimated PASP > 47 mmHg was only 59% sensitive and 78% specific in diagnosing pulmonary arterial hypertension defined as mean pulmonary arterial pressure > 25 mmHg in patients with advanced liver disease, while estimated PASP > 43 mmHg was 91% sensitive and 100% specific in patients without liver disease.

Although this paper contains of the accuracy and cut-off values of a very important hemodynamic parameter in diagnosing pulmonary hypertension, some limitations should be keep in mind before interpreting the results and applying the conclusion to our practice. First, patients with advance liver disease may change their intracardiac volume status

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according to presence of hypoalbuminemia, ascites, and/or the use of diuretics for 3 months. Since this study was retrospectively performed, the non-invasive hemodynamic results may influence the patient's treatment including medications. Second, the recognition of PoPHT in patients with advanced liver disease requires high clinical suspicion because symptoms may not be overt. Therefore, the most important role of non-invasive assessment of PASP is not on the accuracy and specificity but on sensitivity as a screening tool for pulmonary hypertension. In the present study, the sensitivity of ePASP > 47 mmHg was only 59%. Third, the differences in baseline characteristics between two groups seems to be prominent. The control group showed a higher prevalence of co-morbidities and most importantly had a higher PASP reflecting a more advanced status of pulmonary hypertension. Although this original investigation has a few intrinsic limitations, a relatively poor diagnostic performance of ePASP for pulmonary hypertension in candidates for liver transplantation may provide us scientific stimuli for the simultaneous hemodynamic comparisons in patients with advanced liver disease.

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