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The Utility of Follow-up Transthoracic Echocardiogram to Screen for Severe Portopulmonary Hypertension (POPH) in Patients Granted POPH Model for End-stage Liver Disease (MELD) Exceptions

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Background. The current model for end-stage liver disease (MELD) exception policies for portopulmonary hypertension (POPH) require serial right heart catheterizations (RHCs) every 3 mo to maintain exception points. RHC is necessary for the initial diagnosis of POPH, but the utility of serial catheterizations has not been studied. In patients with POPH MELD exceptions, we sought to compare noninvasive and invasive hemodynamics and determine the sensitivity of echocardiography for the detection of hemodynamically severe POPH that would preclude liver transplant. **Methods.** We performed a single-center retrospective cohort study of patients with POPH MELD exceptions who underwent liver transplant from December 2008 to January 2024. Results were validated at an external center. Echocardiograms and RHCs performed within 1 mo were compared. Pearson correlation coefficient and Bland-Altman plots assessed the association between echocardiogram and RHC variables. We examined varied echocardiographic parameters to optimize sensitivity for the detection of hemodynamically severe POPH. **Results.** Twenty-two individuals underwent 60 follow-up RHCs with paired echocardiograms. Right ventricular systolic pressure (RVSP) and cardiac index estimated with echocardiogram were not strongly correlated with RHC measurements at follow-up (RVSP and RHC pulmonary artery systolic pressure: $R = 0.30$, $P = 0.02$; cardiac index: $R = 0.17$, $P = 0.21$). However, echocardiograms with RVSP ≥48 mm Hg had 100% sensitivity for detecting hemodynamically severe POPH, with 100% negative predictive value. In external validation of 13 paired echocardiograms and RHCs, our algorithm had 64% specificity and 100% negative predictive value. **Conclusions.** Although echocardiogram and RHC hemodynamic estimates were not strongly correlated, these results could potentially negate the current requirement for repeat RHC every 3 mo to maintain POPH MELD exception.

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Portopulmonary hypertension (POPH) describes pulmonary arterial hypertension (PAH) that develops in the setting of portal hypertension. POPH affects approximately 5%–6% of patients with advanced liver disease and has significant implications for liver transplant (LT) candidacy and outcomes.[1] Like other forms of group 1 pulmonary hypertension (PH), POPH is hemodynamically defined by precapillary PH.[2,3]

Much of the clinical importance of POPH has been described in the setting of LT evaluation and outcomes, with

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early studies demonstrating significantly increased post-LT cardiovascular mortality risk in patients with moderate (mean pulmonary artery pressure [mPAP] > 35 mm Hg)[4] to severe (mPAP > 50 mm Hg) POPH.[5] Since 2006, patients with POPH have been eligible for model for end-stage liver disease (MELD) exception points to account for their additional cardiovascular and overall mortality risk, which is not otherwise captured within the MELD laboratory score. The POPH MELD exception policy was intended to appropriately prioritize these patients for LT so they could ideally undergo transplant before their PH progressed in severity and led to right ventricular (RV) failure.[6] The POPH MELD exception criteria, most recently updated in February 2021, include the following: (1) portal hypertension, (2) diagnosis of POPH via hemodynamic data collected from right heart catheterization (RHC) demonstrating mPAP >35 mm Hg, elevated pulmonary vascular resistance (PVR), and normal pulmonary arterial wedge pressure and (3) documented treatment with US Food and Drug Administration–approved PAH therapy for a minimum of 12 wk with (4) posttreatment RHC demonstrating either mPAP <35 mm Hg and posttreatment PVR <400 dyne·s/cm^5 (5 Wood units [WU]) or mPAP 35–45 mm Hg with a posttreatment PVR <240 dyne·s/cm^5 (3 WU). Furthermore, the current POPH MELD exception policies require repeat RHC every 3 mo while awaiting LT to ensure hemodynamic stability and maintain and accrue exception points.

Like all forms of PH, RHC is necessary for the initial diagnosis of POPH, but the utility of serial follow-up catheterizations in these patients has not been studied. Additionally, RHC is an invasive procedure with associated risk and clinical burden to patients, particularly patients with liver disease who may have an increased risk of bleeding related to thrombocytopenia and/or coagulopathy. Routine follow-up RHC every few months is not considered standard of care in other forms of PAH; instead, RHC is generally performed on the basis of clinical indications such as worsening symptoms or concern for inadequate treatment response. Transthoracic echocardiogram (TTE) is an excellent screening tool for POPH. It may also be helpful to guide the need for follow-up invasive hemodynamic assessment in patients with POPH MELD exceptions.[7,8] Therefore, we sought to compare noninvasive (specifically, TTE) and invasive (RHC) hemodynamic and operability assessments in patients with treated POPH granted POPH MELD exceptions. Our goal was to provide helpful insight that may inform future policies and best practices within this patient population. Specifically, we wanted to address the current Organ Procurement and Transplantation Network policy need/requirement for repeat RHC every 3 mo to maintain POPH MELD exception.

MATERIALS AND METHODS

Study Design

We performed a retrospective cohort study at Mayo Clinic Rochester, Arizona, and Florida to address the above aims and to develop an echocardiographic algorithm for screening for severe POPH that would preclude the safety of LT. We then performed an external validation of this algorithm at the University of Wisconsin.

Subjects and Data Collection

We included patients with POPH who were granted POPH MELD exceptions and underwent LT between December 2008 and January 2024. Patient demographics, MELD exception and LT information, and hemodynamic and echocardiographic data were collected via chart review from the electronic health record. Initial pretreatment diagnostic RHC and TTE were reported with baseline patient characteristics. Follow-up RHCs performed while patients were on PAH therapy in the pretransplant period were compared with TTE performed within 1 mo of RHC. We chose to focus on follow-up RHC and TTE in our analyses as we sought to develop an algorithm for patients with established, treated POPH and approved MELD exceptions. If an individual patient underwent multiple repeat RHCs in the pretransplant period to maintain MELD exception points, all follow-up RHCs were included in the analysis. Posttransplant RHC and TTE data were not included. Per current POPH MELD exception criteria, patients were treated with Food and Drug Administration–approved therapy(-ies) for PAH, of which POPH is a subset. These include phosphodiesterase 5 inhibitors (eg, sildenafil, tadalafil), endothelin receptor antagonists (eg, macitentan, ambrisentan), soluble guanylate cyclase (sGC) stimulators (riociguat), and/or prostacyclin pathway agents (eg, treprostinil). Time on the LT waitlist was extracted from the electronic health record using the date of the United Network for Organ Sharing listing and the date of the transplant.

External Validation

We also sought to validate the algorithm in an external cohort of patients from a second tertiary academic center (University of Wisconsin). The same inclusion criteria were used to identify patients.

Statistical Methods

Data were summarized using median (interquartile range) or number (%). Corresponding variables from TTE and RHCs performed within 1 mo were compared using Pearson correlation coefficients and Bland-Altman plots. We examined varied thresholds to optimize the sensitivity for detection of hemodynamically severe POPH, defined as (1) mPAP of 35–45 mm Hg with PVR ≥ 240 dyne·s/cm^5, (2) mPAP >45 mm Hg, or (3) PVR >400 dyne·s/cm^5 in accordance with current MELD exception criteria. Notably, LT is typically contraindicated and/or deferred when hemodynamically severe POPH is present. The study was approved by the Institutional Review Board (Mayo Clinic, IRB 20-007953).

RESULTS

Patient Demographics

We included 22 individuals who underwent a total of 60 follow-up RHCs with paired TTE. Seven of 22 individuals were women (32%) with an average age of 58 y at the time of LT. The most common causes of liver disease in the cohort were hepatitis C (6/22, 27.3%) and alcohol (6/22, 27.3%). The median calculated laboratory MELD-Na score at the time of listing was 16. The majority of the cohort had severely elevated right ventricular systolic pressure (RVSP; median 79 mm Hg; interquartile range [IQR], 67–86) on baseline TTE, with 36.8% (7/19) demonstrating mild RV dysfunction and 36.8% (7/19) with moderate or moderate-severe RV dysfunction. The median baseline hemodynamics on initial (diagnostic) RHC were consistent with severe POPH: mPAP 52 mm Hg (IQR, 41–58) and PVR 6.4 WU (IQR, 4.4–9.9). Among the

cohort, the median waitlist time was 318 d (IQR, 86–674), with a mean of 819 d (SD, 1225). Just before transplant, a majority of patients were treated with an endothelin receptor antagonist (17/22; 77.3%) and combination PAH therapy (15/22; 68.2%). Further details regarding baseline characteristics can be found in Table 1.

Paired RHCs and TTEs at Follow-up

A total of 60 paired follow-up TTEs and RHCs were included in the analysis. The exact number of specific parameter pairs varied slightly based on available data reported on echocardiogram; for example, RVSP was not reported for all echocardiograms, most often due to the inability to measure tricuspid regurgitation velocity. Echocardiograms were performed within a median 2 d (IQR, 1–12) of RHC. At baseline, TTE RVSP and RHC pulmonary artery systolic pressure (PASP) were moderately but not significantly correlated (R = 0.41, P = 0.07) and were weakly correlated at follow-up (n = 58 paired, R = 0.30, P = 0.02; Table 2). Similarly, follow-up TTE and RHC CI measurements were not strongly correlated (n = 54 paired, R = 0.17, P = 0.21). Correlation data are reported in Table 2. Bland-Altman plots are depicted in Figure 1.

The median difference between follow-up TTE RVSP and RHC PASP among the cohort was 8 mm Hg (IQR, 5–12), with the majority (62%) having a difference of <10 mm Hg between the modalities. The median difference between cardiac index (CI) as measured by TTE and RHC was 0.7 L/min/m^2 (IQR, 0.4–2.0).

In total, individual patients underwent as many as 11 total RHCs while awaiting LT (range, 2–11), with a median of 5 (IQR, 1–8) RHCs while awaiting LT.

TABLE 1.

Baseline characteristics (N = 22)

Baseline characteristics	
Female Sex	7/22 (32%)
Age at time of LT, y	58 (50–60)
Race (self-identified)	
White	19/22 (86.4%)
American Indian	2/22 (9.1%)
Hispanic	1/22 (4.5%)
Cause of liver disease	
Alcohol	6/22 (27.3%)
Nonalcoholic fatty liver disease	3/22 (13.6%)
Hepatitis C	6/22 (27.3%)
Cryptogenic cirrhosis	3/22 (13.6%)
Other	4/22 (18.2%
Laboratory values (MELD) (n = 20)	
Na	140 (137-141)
Creatinine	1.04 (0.9-1.5)
Total bilirubin	2.3 (1.5-3.4)
INR	1.4 (1.3-1.8)
Calculated MELD-Na (n = 20)	16 (14–20)
Echocardiogram at diagnosis	
Estimated right atrial pressure (n = 18), mm Hg	10 (5–14)
Estimated right ventricular systolic pressure (n = 19), mm Hg	79 (67–86)
Estimated cardiac output (n = 16), L/min	6.4 (5.9–7.6)
RV function: n = 19	
Normal	5/19 (26.3%)
Mild dysfunction	7/19 (36.8%)
Moderate dysfunction	4/19 (21%)
Moderate-severe dysfunction	3/19 (15.8%)
Pulmonary hemodynamics at diagnosis	
Right atrial pressure (n = 21), mm Hg	11 (7–18)
Pulmonary artery systolic pressure (n = 21), mm Hg	87 (65–95)
Mean pulmonary artery pressure (n = 22), mm Hg	52 (41–58)
Pulmonary artery wedge pressure (n = 22), mm Hg	11 (9–15)
Cardiac output (n = 22), L/min	5.8 (4.7–6.8)
Cardiac index (n = 21), L/min/m^2	2.8 (2.5–3.2)
Pulmonary vascular resistance (n = 19), Wood unit	6.4 (4.4–9.9)
PAH treatment	
ERA	17/22 (77.3%)
PDE5i	11/22 (50%)
Inhaled treprostinil	4/22 (18.2%)
Intravenous or subcutaneous prostacyclin	8/22 (36.4%)
sGC stimulator	1/22 (4.5%)
Monotherapy (1 PAH medication)	7/22 (31.2%)
Combination therapy (>1 PAH medication)	15/22 68.2%)

ERA, endothelin receptor antagonist; INR, international normalized ratio; LT, liver transplant; MELD, model for end-stage liver disease; PAH, pulmonary arterial hypertension; PDE5i, phosphodiesterase 5 inhibitor; RV, right ventricular; sGC, soluble guanylate cyclase.

TABLE 2.

Pearson correlation between pulmonary pressure and CI measured by TTE vs RHC

	TTE RVSP/RHC PASP	TTE CI/RHC CI
Baseline	N = 20	N = 17
	R = 0.41, P = 0.07	R = 0.44, P = 0.07
Follow-up	N = 58	N = 54
	R = 0.30, P = 0.02	R = 0.17, P = 0.21

CI, cardiac index; PASP, pulmonary artery systolic pressure; RHC, right heart catheterization; RVSP, right ventricular systolic pressure; TTE, transthoracic echocardiography.

Assessing for Hemodynamically Severe POPH

Sixteen follow-up RHCs (16/60; 26.7%) demonstrated hemodynamically severe POPH that would preclude the safety of LT and accrual of MELD exception points. The majority of TTEs (58/60; 96.7%) had RVSP reported. Follow-up echocardiograms with an RVSP ≥50 mm Hg had 93% sensitivity for detecting hemodynamically severe POPH, whereas echocardiograms with RVSP ≥48 mm Hg had 100% sensitivity for detection of hemodynamically severe POPH with a specificity of 28% and 100% negative predictive value (NPV). See Table 3 for details.

External Validation

In our external validation cohort, 13 paired echocardiograms and RHCs within 40 d were identified among patients with POPH MELD exceptions. None of the RHCs met the criteria for hemodynamically severe POPH. RVSP ≥48 mm Hg had 69% specificity and 100% NPV for hemodynamically severe POPH. Since no RHCs met the criteria for hemodynamically severe POPH, none of these 13 procedures changed MELD exception eligibility.

DISCUSSION

In our retrospective analysis of 22 patients granted POPH MELD exceptions who underwent LT between December 2008 and January 2024, we evaluated 60 paired follow-up TTEs and RHCs. The overall cohort had significantly

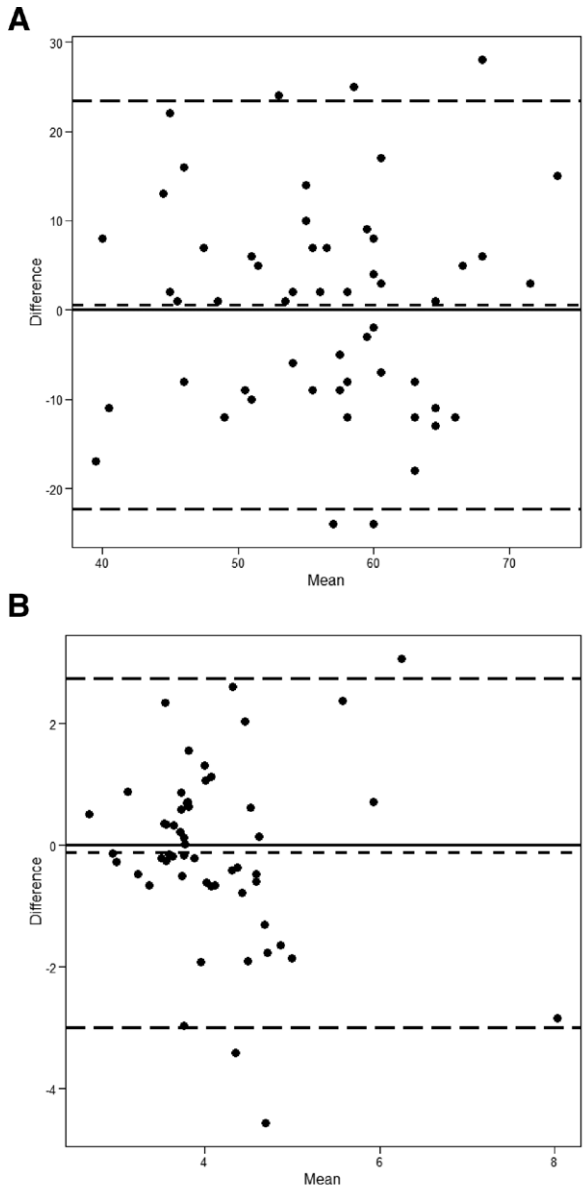

FIGURE 1. Bland-Altman plots. A, TTE and RHC PASP at follow-up. B, TTE and RHC CI at follow-up. CI, cardiac index; PASP, pulmonary artery systolic pressure; RHC, right heart catheterization; TTE, transthoracic echocardiogram.

elevated pulmonary pressures (median mPAP 52 mm Hg, PVR 6.4 WU) on initial diagnostic RHC, with only about one-quarter (26.3%) having normal RV function at baseline. Neither RVSP nor CI, as assessed by follow-up TTE, were strongly correlated with invasive pulmonary hemodynamics as measured by RHC. Nonetheless, TTE provided valuable noninvasive information for the detection of hemodynamically severe POPH: RVSP ≥48 mm Hg was 100% sensitive, with a high NPV in the detection of severe PH that would preclude the safety of LT. In an external validation patient cohort from another academic center, NPV was also 100% with a specificity of 69%, although none of the follow-up RHCs demonstrated severe POPH and therefore did not affect MELD eligibility. In summary, follow-up TTE was not a good surrogate for RHC in assessing exact hemodynamics but was helpful in assessing high-risk hemodynamics and

TABLE 3.

TTE screening performance for the detection of severe PH precluding LT

N = 58 paired TTE + RHC, 15/58 severe PH	RVSP ≥48 mm Hg	RVSP ≥50 mm Hg
Sensitivity	100%	93%
Specificity	28%	28%
Positive predictive value	33%	31%
Negative predictive value	100%	92%
N used for calculations		
True positive	15	14
False negative	0	1
False positive	31	31
True negative	12	12

LT, liver transplant; PH, pulmonary hypertension; RHC, right heart catheterization; RVSP, right ventricular systolic pressure; TTE, transthoracic echocardiography.

determining overall LT eligibility. Our findings may have important implications for current MELD exception policies, which require serial follow-up RHC every 3 mo for patients granted POPH MELD exceptions. Our study results suggest that noninvasive assessment with TTE is valuable and has excellent NPV for ruling out hemodynamically severe POPH that would preclude the safety of LT and accrual of MELD exception points. Importantly, this analysis suggests that we may preclude the need for repeat RHC every 3 mo, which incurs both risk and expense, to maintain the POPH MELD exception.

In this patient cohort, median baseline pulmonary hemodynamics (mPAP 52 mm Hg, PVR 6.4 WU) were consistent with severe PH, which, per current guidelines, would preclude LT due to prohibitive perioperative risk.[1,9] Treatment of POPH with PAH therapy is indicated to improve pulmonary hemodynamics and facilitate the safety of LT. These are the types of patients for whom the POPH MELD exception criteria were intended to identify and prioritize appropriately for transplant if they achieve acceptable hemodynamics. As prior studies have demonstrated, TTE is an excellent screening tool for the initial detection of POPH and is included as such in pre-LT screening guidelines.[1,10] Raevens et al[7] previously assessed different RVSP (referred to as systolic pulmonary artery pressure [sPAP] in their study) cutoff values on TTE for initial POPH screening in potential LT candidates. They demonstrated that sPAP of 38 mm Hg had an excellent (100%) sensitivity and NPV for detecting POPH (confirmed via RHC) with an 82% specificity.[7] A retrospective study from Habash et al[11] assessed the correlation between TTE and RHC measurements of sPAP among patients being evaluated for LT and a control group without liver disease. Although estimated PASP per TTE and as measured by RHC were modestly correlated (R= 0.58, P = 0.006) in the potential LT group, they were more strongly correlated (R= 0.74, P < 0.001) in the control group, although the difference in correlation was ultimately not statistically significant.[11] Their findings suggest that echocardiographic estimates of pulmonary pressures may be somewhat less reliable among patients with significant liver disease, as also demonstrated in our findings. This is likely multifactorial as estimates of RVSP are heavily influenced by the pathophysiologic disturbances related to hepatic dysfunction, including volume

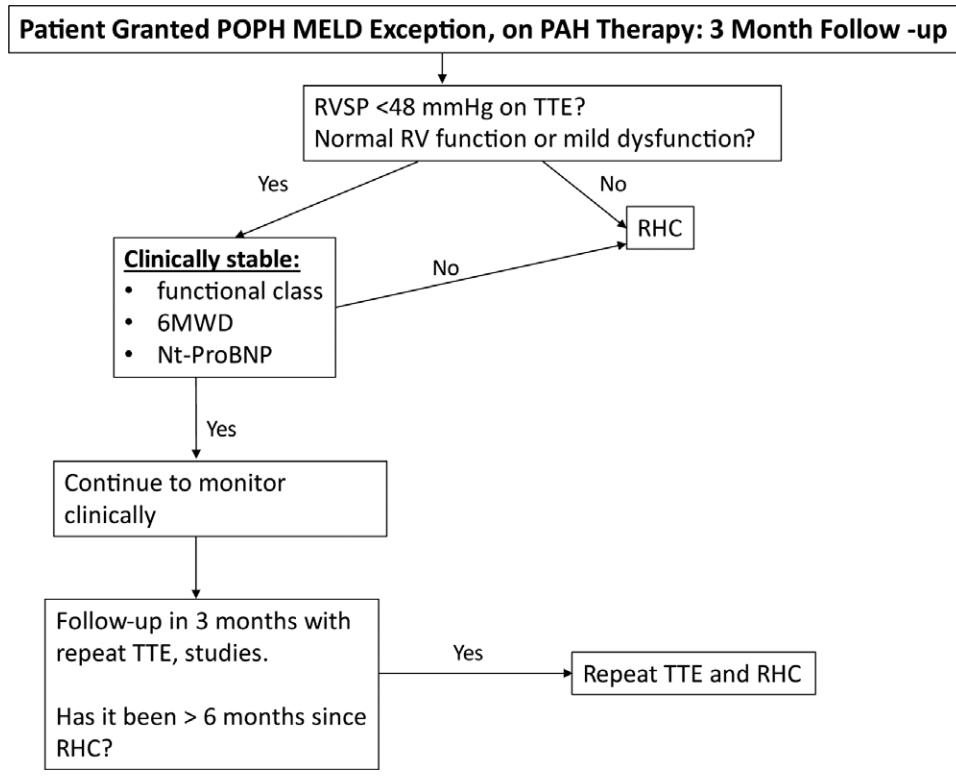

FIGURE 2. Proposed alternative follow-up clinical algorithm for patients granted POPH MELD exceptions. MELD, model for end-stage liver disease; POPH, portopulmonary hypertension.

shifts and a tendency toward a high cardiac output state, among others. Our study's findings indicate that such echocardiographic limitations continue to affect the accuracy of TTE estimates once patients are treated with PAH therapy. Furthermore, TTE can have its own limitations, including operator dependence and sometimes limited windows, among others.

To our knowledge, our study is the first to assess TTE screening validity and specific cutoff values among patients with POPH on PAH therapy awaiting LT with validation in an external cohort. Importantly, the high sensitivity of our screening TTE criteria in detecting hemodynamically severe PH suggests that mandated RHC every 3 mo may be unnecessary for all patients. Furthermore, other countries (eg, France) do not have this requirement. Instead, more similarly to standard clinical practice among other forms of PAH, the need for RHC while on therapy should be determined by a combination of clinical status and/or worsening and the results of noninvasive testing, including TTE. Six-minute walk distance, for example, is a commonly used functional assessment in PAH, with a recent meta-analysis identifying a minimal clinically important difference of approximately 33 m.[12] Alternatively, the frequency of serial RHCs could be decreased to every 6 mo rather than every 3 mo, assuming reassuring TTE findings and clinical status. Additionally, in patients with borderline hemodynamics and/or worsening TTE findings (eg, increasing RVSP, declining RV function) who are nearing LT, it is reasonable to repeat RHC to ensure acceptable hemodynamics. That approach would be similar to living donor LT considerations in which potential liver recipients are being treated for POPH.

Within our follow-up cohort data, only 3 of 60 TTEs demonstrated more than mild (ie, mild-moderate, moderate, severe) RV dysfunction, making the inclusion of an RV function threshold not immediately applicable to our screening algorithm. However, we suspect this nonetheless plays a significant role in LT risk assessment and therefore advise maintaining a low threshold to repeat RHC with poor and/or worsening RV function. An example of a potential revised clinical follow-up algorithm can be seen in Figure 2.

Importantly, we do **not** advocate for eliminating RHC for the diagnosis or clinical follow-up evaluation of POPH. Rather, we think serial RHC every 3 mo in patients with treated POPH and MELD exceptions are excessive and often unnecessary. Only 27% of follow-up RHCs changed management in regard to LT eligibility, and TTE was able to detect these in all cases where TTE RVSP was reported (N = 58). The patient-related burden of repeated RHC, including procedural risk, time commitment, and financial burden, etc, should not be underemphasized, particularly when some patients have to undergo up to 11 RHCs in the pre-LT period, as we saw in our cohort. Although RHC is generally considered a low-risk procedure and the actual incidence of complications is unknown, there are numerous published case reports and series detailing (often severe and sometimes fatal) adverse sequelae. Chen et al[13] published a literature review on this topic in 2020, which included complications from 46 articles. Complications from RHC were related to venous access (eg, pseudoaneurysm, vascular perforation, arteriovenous fistula formation) or catheterization itself (eg, arrhythmias, rupture or injury of involved structures [right atrium, tricuspid valve, RV, pulmonary artery, etc], endocarditis).[13] Moreover, it is likely that more common, less severe complications are underrepresented in the available literature. Although RHC is typically safe for the majority of patients, the procedural

risk remains and can be rarely catastrophic. Therefore, consideration should be given to alternative noninvasive assessments such as TTE when effective and feasible. Furthermore, the utilization of healthcare resources with RHC, as with any invasive procedure, can also be significant.

Limitations

The limitations of our study include the retrospective nature of the study and the small sample size. There was also variability in how many paired TTE and RHCs patients underwent, ranging from 2 to 11, making it likely that certain patients' data were overly represented. The cohort was predominantly men (68%) and White (86.4%), with women and other ethnic groups underrepresented. Additionally, our external validation was limited to a single institution that did not include patients with severe hemodynamic POPH measured by RHC, potentially limiting its informative impact.

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