

# Improved hospitalization rates in a specialty center for heart failure with preserved ejection fraction and pulmonary hypertension

Chad M. Kosanovich<sup>1</sup> | Hongyang Pi<sup>2</sup> | Adam Handen<sup>3</sup> | Erin Schikowski<sup>4</sup> |  
Yimin Chen<sup>4</sup> | Floyd W. Thoma<sup>1</sup> | Suresh Mulukutla<sup>1,5</sup> | Steve Koscomb<sup>5</sup> |  
Mehdi Nouraie<sup>6</sup> | Stephen Y. Chan<sup>1,3,4</sup>

<sup>1</sup>Department of Medicine, Division of Cardiology, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania, USA

<sup>2</sup>Department of Medicine, Division of Pulmonary, Critical Care and Sleep Medicine, University of Washington Medical Center, Seattle, Washington, USA

<sup>3</sup>Center for Pulmonary Vascular Biology and Medicine; Pittsburgh Heart, Lung, Blood, and Vascular Medicine Institute, Pittsburgh, Pennsylvania, USA

<sup>4</sup>Department of Medicine, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania, USA

<sup>5</sup>Clinical Analytics, University of Pittsburgh Medical Center, Pittsburgh, Pennsylvania, USA

<sup>6</sup>Department of Medicine, Division of Pulmonary, Allergy and Critical Care Medicine, University of Pittsburgh, Pittsburgh, Pennsylvania, USA

## Correspondence

Stephen Y. Chan, Center for Pulmonary Vascular Biology and Medicine, Pittsburgh Heart, Lung, Blood, and Vascular Medicine Institute, Division of Cardiology, Department of Medicine, University of Pittsburgh Medical Center, 200 Lothrop St BST E1240, Pittsburgh, PA 15261, USA.  
Email: [chansy@pitt.edu](mailto:chansy@pitt.edu)

## Funding information

National Heart, Lung, and Blood Institute, Grant/Award Numbers: HL122596, HL124021, HL138437; American Heart Association, Grant/Award Number: 18EIA33900027

## Abstract

Heart failure with preserved ejection fraction can be complicated by pulmonary hypertension. We designed a retrospective study to provide supporting evidence for referral to specialty care centers. Specialty care centers improved hospitalizations but not mortality—in part due to more aggressive medication management and guideline-directed monitoring.

## KEYWORDS

combined post- and precapillary PH, heart failure with preserved ejection fraction, pulmonary hypertension, specialty care center

**Abbreviations:** Cpc-PH, combined post- and precapillary PH; HFpEF, heart failure with preserved ejection fraction; HC, hazard ratio; Ipc-PH, isolated postcapillary PH; mPAP, mean pulmonary arterial pressure; PH, pulmonary hypertension; RHC, right heart catheterization; SCC, specialty care centers; UPMC, University of Pittsburgh Medical Center.

Chad M. Kosanovich, Hongyang Pi, Mehdi Nouraie and Stephen Y. Chan contributed equally to this study.

This is an open access article under the terms of the Creative Commons Attribution License, which permits use, distribution and reproduction in any medium, provided the original work is properly cited.

© 2021 The Authors. *Pulmonary Circulation* published by Wiley Periodicals LLC on behalf of the Pulmonary Vascular Research Institute.

## BACKGROUND

Heart failure with preserved ejection fraction (HFpEF) accounts for nearly half of hospitalizations for heart failure in the United States.<sup>1</sup> Chronic passive venous congestion raises postcapillary pulmonary pressures and can induce pulmonary hypertension (PH) with worsened prognosis.<sup>2,3</sup> In HFpEF registries, the prevalence of PH-HFpEF is noted up to 83%.<sup>2</sup>

Passive transmission of venous congestion can cause isolated postcapillary PH (Ipc-PH).<sup>4</sup> As pulmonary vascular disease becomes disproportionate to left ventricular disease, a combined post- and precapillary PH (Cpc-PH) phenotype develops.<sup>5</sup> Cpc-PH resembles pulmonary arterial hypertension with respect to genetics, hemodynamics, and survival, but comorbidities mimic Ipc-PH.<sup>4,5</sup>

Unlike pulmonary arterial hypertension and more similar to the management of HFpEF, PH-HFpEF lacks guideline-directed vasodilator therapy, and management focuses on the optimization of comorbidities and volume status.<sup>6,7</sup> In the United States, the Pulmonary Hypertension Association has increased referral efforts to specialty care centers (SCC) with mortality and hospitalization benefits for pulmonary arterial hypertension.<sup>8,9</sup> Current guidelines recommend referral to SCC when a Cpc-PH phenotype develops, but guidelines lack supporting data with IIIc level of evidence.<sup>6</sup>

We compared care of PH-HFpEF managed at SCC versus non-SCC locations and hypothesized SCC improved outcomes with respect to mortality and hospitalizations with higher compliance using contemporary guidelines for pulmonary hypertension.<sup>6</sup>

## METHODS

The Institutional Review Board at the University of Pittsburgh Medical Center (UPMC) approved this study (PRO11070366), and this study was performed in accordance with the Helsinki Declaration. By using the Medical Archival Retrieval System, a repository of UPMC health data encompassing 41 network hospitals, patients were retrospectively identified between January 1, 2008, and December 1, 2018, with a left ventricular ejection fraction  $\geq 50\%$  by transthoracic echocardiogram with a mean pulmonary arterial pressure (mPAP)  $\geq 25$  mmHg and pulmonary capillary wedge pressure  $> 15$  mmHg by index right heart catheterization (RHC). Patients were further classified by RHC using pulmonary vascular resistance. Ipc-PH was defined when pulmonary vascular resistance was  $< 3$  woods units. Cpc-PH was defined when pulmonary vascular resistance was  $\geq 3$  woods units. Patients with at least one encounter with a SCC

provider at UPMC (accredited by the Pulmonary Hypertension Association) were included in the SCC cohort. The non-SCC cohort was further divided, based on a hospital size cut-off of 300 beds.

Demographics, comorbidities by International Classification of Disease codes, yearly medications, and frequencies of RHC and transthoracic echocardiogram were included from index RHC. A mixed effect survival model with the random coefficient for each patient and Weibull distribution established time to hospital admission. Cox proportional hazard modeling was used for mortality and presented as hazard ratio (HR) with adjustment for disease severity using mPAP, age, gender, and comorbidities. Medication adjustment was used in subanalysis with 83% coverage in the cohort as well as hospital size. Two-sided statistical tests with a  $p < 0.05$  was defined as significant. All statistical analyses were performed using Stata 17.0 software (StataCorp).

## RESULTS

A total of 2863 patients in the UPMC system met inclusion criteria with 974 managed at SCC and 1889 at non-SCC. SCC patients were younger (median 66 vs. 69 years,  $p < 0.001$ ), more commonly female (60 vs. 51%,  $p < 0.001$ ), followed for longer (5.3 vs. 4.1 years,  $p < 0.001$ ), and had worsened hemodynamic parameters including mPAP and pulmonary vascular resistance ( $p < 0.001$ ). Pulmonary capillary wedge pressure was lower for SCC ( $p < 0.001$ ). Cardiac output was not different. The frequencies of hypertension, congestive heart failure, coronary artery disease, obesity, chronic obstructive pulmonary disease, obstructive sleep apnea, pulmonary fibrosis, end-stage renal disease, cirrhosis, and thromboembolism were significantly higher in SCC. SCC management led to more frequent RHCs (mean: 1.22, 95% CI: 1.16–1.29 vs. 1.06, 95% CI: 1.04–1.07,  $p < 0.001$ ) but not echocardiograms.

Referral and management by SCC were associated with reduced hospital admissions (HR: 0.84, 95% CI: 0.78–0.90,  $p < 0.001$ ) (Table 1), but not mortality. When subdivided for hemodynamic group, Cpc-PH (HR: 0.84, 95% CI: 0.74–0.94,  $p = 0.003$ ) and Ipc-PH (HR: 0.83, 95% CI: 0.76–0.92,  $p < 0.001$ ) demonstrated a significant reduction in hospitalizations without mortality difference. After index RHC, 87 patients in SCC and 40 in non-SCC converted from Ipc-PH to Cpc-PH. In two additional analyses, including completely removing the converts and reclassifying all converters into the Cpc-PH subgroup, these major significant differences in SCC versus non-SCC outcomes were still observed. A secondary analysis to stratify based on mPAP  $\geq 40$  from index RHC

**TABLE 1** Effects of SCC on clinical outcomes including hospitalization and mortality

	Effect on time to admission (HR) <sup>d</sup>	Effect on mortality (HR) <sup>d</sup>	Effect on time to admission (HR) <sup>c</sup>	Effect on mortality (HR) <sup>c</sup>
1a: SCC vs. non-SCC centers (large and small)				
All <sup>a</sup>				
(N = 2602) <sup>d</sup>	<b>0.84 (0.78–0.90)</b>	1.09 (0.95–1.24)	<b>0.89 (0.82–0.97)</b>	1.08 (0.93–1.25)
(N = 2153) <sup>c</sup>	<b>p &lt; 0.001</b>	p = 0.21	<b>p = 0.007</b>	p = 0.33
Cpc-PH				
(N = 1009) <sup>d</sup>	<b>0.84 (0.74–0.94)</b>	1.08 (0.89–1.31)	<b>0.87 (0.76–0.99)</b>	1.09 (0.88–1.35)
(N = 870) <sup>c</sup>	<b>p = 0.003</b>	p = 0.44	<b>p = 0.030</b>	p = 0.43
Ipc-PH				
(N = 1593) <sup>d</sup>	<b>0.83 (0.76–0.92)</b>	1.06 (0.88–1.29)	0.91 (0.82–1.01)	1.05 (0.84–1.30)
(N = 1283) <sup>c</sup>	<b>p &lt; 0.001</b>	p = 0.52	p = 0.08	p = 0.70
1b: SCC vs. large non-SCC centers				
All <sup>b</sup>				
(N = 2123) <sup>d</sup>	<b>0.84 (0.77–0.91)</b>	1.09 (0.94–1.25)	<b>0.86 (0.79–0.94)</b>	1.05 (0.90–1.23)
(N = 1893) <sup>c</sup>	<b>p &lt; 0.001</b>	p = 0.25	<b>p &lt; 0.001</b>	p = 0.51
Cpc-PH				
(N = 849) <sup>d</sup>	<b>0.84 (0.74–0.95)</b>	1.15 (0.93–1.42)	<b>0.81 (0.71–0.93)</b>	1.05 (0.84–1.31)
(N = 772) <sup>c</sup>	<b>p = 0.005</b>	p = 0.19	<b>p = 0.003</b>	p = 0.667
Ipc-PH				
(N = 1274) <sup>d</sup>	<b>0.84 (0.76–0.93)</b>	1.0 (0.82–1.22)	<b>0.89 (0.80–0.98)</b>	–
(N = 1121) <sup>c</sup>	<b>p &lt; 0.001</b>	2.0 p = 0.98	<b>p = 0.024</b>	p = 0.83

Note: Bold values denote  $p < 0.05$ .

<sup>a</sup>Adjusted for age, gender, mean pulmonary arterial pressure, and baseline comorbidities including congestive heart failure, chronic obstructive pulmonary disease, coronary artery disease/coronary artery bypass grafting, obstructive sleep apnea, pulmonary fibrosis, end-stage renal disease, cirrhosis, deep vein thrombosis/pulmonary embolism.

<sup>b</sup>Adjustment as above including medications: loop diuretics, spironolactone, vasodilators, antihypertensives, antiplatelets, statins, anticoagulation, diabetes treatment, obstructive airway treatment.

<sup>c</sup>Interaction between center and group:  $p = 0.56$  for time to admission,  $p = 0.55$  for mortality when compared to all hospitals (1a).

<sup>d</sup>Interaction between center and group:  $p = 0.40$  for time to admission,  $p = 0.70$  for mortality when compared to large hospitals (1b).

demonstrated a more robust reduction in hospitalization for SCC overall (HR: 0.77, 95% CI: 0.66–0.91,  $p = 0.002$ ) and when subdividing for Cpc-PH (HR: 0.78, 95% CI: 0.64–0.94,  $p = 0.012$ ) and Ipc-PH (HR: 0.64, 95% CI: 0.50–0.84,  $p = 0.001$ ) specifically.

Medication use differed among cohorts, especially with respect to loop diuretics, spironolactone, and vasodilator therapy. SCC patients were more commonly prescribed loop diuretics (47% vs. 40%,  $p = 0.001$ ) and spironolactone (18% vs. 10%,  $p < 0.001$ ), and significant for each hemodynamic group for loop diuretics (Cpc-PH:

51% vs. 45%,  $p < 0.001$  and Ipc-PH: 48% vs. 42%,  $p < 0.001$ ), and spironolactone (Cpc-PH: 20% vs. 10%,  $p < 0.001$  and Ipc-PH: 16% vs. 11%,  $p < 0.001$ ). Vasodilator use was more common in SCC (14% vs. 1.5%,  $p < 0.001$ ) and most evidently in Cpc-PH (21% vs. 2.7%,  $p < 0.001$ ). There was low frequency use in Ipc-PH (3.8% vs. 0.9%,  $p < 0.001$ ). Of SCC patients, 12 Ipc-PH patients were on vasodilators, with most vasodilators being phosphodiesterase inhibitors prescribed for erectile dysfunction (four patients) or for Cpc-PH hemodynamics that appeared later and with documented improvement

in symptoms (eight patients). Alternatively, 10 patients in non-SCC Ipc-PH subgroup were prescribed vasodilators, with the majority started on vasodilator therapy inappropriately (six patients) and a minority prescribed such medications for erectile dysfunction (four patients).

To address the possibility that SCC versus non-SCC outcomes were driven by these differences in medication use, analyses were performed of patients with available medication records. Adjustment for medication use resulted in a significant but modestly mitigated reduction in hospital admission (adjusted HR: 0.89, 95% CI: 0.82–0.97,  $p = 0.007$ ) for SCC patients (Table 1a). When subdivided by hemodynamic group, a significant yet mitigated reduction in hospitalizations was still seen for Cpc-PH (HR: 0.87, 95% CI: 0.76–0.99,  $p = 0.03$ ), but was nonsignificant in Ipc-PH.

To address the possibility that SCC versus non-SCC outcomes were driven by differences in access to large hospital facilities, we compared the SCC cohort to only non-SCC patients managed at other large hospitals. In this context, hospital admissions were again reduced in the SCC (HR: 0.86, 95% CI: 0.79–0.94,  $p < 0.001$ ) (Table 1b). When subdivided by hemodynamic group, this reduction of hospital admission was observed in both Cpc-PH (HR: 0.81, 95% CI: 0.71–0.93,  $p = 0.003$ ) and Ipc-PH (HR: 0.89, 95% CI: 0.80–0.98,  $p = 0.024$ ) groups. When adjusting for medications, improvement in admissions was only partially blunted, indicating that medications play a part of improving admissions, but do not explain the total benefit of the SCC.

## DISCUSSION

This retrospective, single-hospital system analysis elucidates crucial differences in care for PH-HFpEF patients provided at an SCC and offers evidence of improved hospitalizations. This improvement became more evident in PH patients with  $mPAP \geq 40$ , suggesting those with severe disease benefit robustly from referral. More aggressive treatment of volume status and comorbidities might improve symptoms and hospitalizations in SCC patients.<sup>6,7,10</sup> This is reflected in Table 1a, as adjustment for medications resulted in a mitigated improvement in hospitalizations related to SCC care. When comparing hospitalizations between the SCC and other large non-SCC hospitals in Table 1b, the adjustment for medication use was less apparent. This may be indicative of two important points. First, medical management at SCC significantly improves outcomes. Second, this improvement in hospitalizations is partially driven by medication adjustment and partially related to greater adherence to disease management guidelines by the SCC. Published

guidelines for managing both HFpEF and PH-HFpEF emphasize consistent volume optimization.<sup>6,11</sup> The pulmonary capillary wedge pressure at SCC was lower than non-SCC locations suggesting better control of volume status. Particularly in Cpc-PH patients known to carry worsened prognoses,<sup>4</sup> such improvements may be related to the SCC's high compliance with guideline-directed disease monitoring,<sup>6</sup> as reflected by the higher frequencies of RHC utilization for proper diagnosis and serial hemodynamic follow-up.<sup>12</sup> Additionally, spirinolactone use has been emphasized in HFpEF,<sup>10,13</sup> and more frequent use was noted in the SCC (Cpc-PH: 20% vs. 10%,  $p < 0.001$  and Ipc-PH: 16% vs. 11%,  $p < 0.001$ ). Notably, a small subset of patients with pulmonary vascular disease was prescribed vasodilators, which may provide benefit to selected Cpc-PH patients.<sup>14,15</sup> Conversely, of the vasodilators prescribed for non-SCC Ipc-PH patients, most were started inappropriately, potentially contributing to increased hospitalizations.

This study did not demonstrate a mortality benefit related to SCC care for PH-HFpEF patients as a whole or their hemodynamic subgroups. This may not be surprising, given the dearth of guideline-directed medications in PH-HFpEF and the challenges to manage many of the contributing comorbidities.

This study had limitations. While our SCC was a single-center, we compared to a variety of non-SCC locations within the UPMC system. Multicenter SCC trials will be needed to fully define benefits nationally or globally. Medication usage was only collected on yearly intervals which could affect the medication assessment. Finally, future work to subclassify these patients into more specific cohorts of PH-HFpEF could aid in identifying those who benefit the most from SCC care and potentially new therapies, including SGLT2 inhibitors.<sup>16</sup>

In conclusion, these findings offer quantitative proof to bolster efforts to refer PH-HFpEF patients to SCC with demonstrated improvement in hospitalizations, likely related to medication use and increased guideline-directed disease monitoring.

## ACKNOWLEDGMENTS

We thank M. Saul and O. Marroquin for expert guidance in extraction and analysis of clinical bioinformatic data from the electronic health record system at the University of Pittsburgh Medical Center. We thank SciVelo and the Department of Biomedical Informatics (University of Pittsburgh School of Medicine) for their data support. We acknowledge the Pulmonary Hypertension Association for offering the designation of Specialty Care Center to the UPMC Presbyterian hospital. We acknowledge the Clinical and Translational Science Institute for mortality data via NIH grant UL1

TR001857. National Institutes of Health (R01 HL124021, HL 122596, HL 138437); American Heart Association (18EIA33900027) (S. Y. C.).

### CONFLICT OF INTERESTS

S. Y. C. has served as a consultant for United Therapeutics and Acceleron Pharma; S. Y. C. is a director, officer, and shareholder in Synhale Therapeutics; S. Y. C. has held research grants from Actelion, Bayer, and Pfizer. S. Y. C. has filed patent applications regarding the targeting of metabolism in pulmonary hypertension. The remaining authors declare that there are no conflict of interests.

### ETHICS STATEMENT

The Institutional Review Board at the University of Pittsburgh Medical Center (UPMC) approved this study (PRO11070366), and this study was performed in accordance with the Helsinki Declaration.

### AUTHOR CONTRIBUTIONS

Stephen Y. Chan is the guarantor of this project, had full access to the data in the study, and takes responsibility of the content of the manuscript including the integrity of the data and the accuracy of the data analysis. Chad M. Kosanovich, Hongyang Pi, Mehdi Nouraie, and Stephen Y. Chan conceived and designed the study. Adam Handen, Erin Schikowski, and Yimin Chen performed clinical data collection from electronic health records. Floyd W. Thoma, Steve Koscumb, and Suresh Mulukutla obtained medication collection from the electronic health records. Mehdi Nouraie performed statistical analyses. Chad M. Kosanovich, Hongyang Pi, Mehdi Nouraie, and Stephen Y. Chan drafted the manuscript. All authors provided input on final letter revision.

### REFERENCES

- Lloyd-Jones D, Adams R, Carnethon M, De Simone G, Ferguson TB, Flegal K, Ford E, Furie K, Go A, Greenlund K, Haase N, Hailpern S, Ho M, Howard V, Kissela B, Kittner S, Lackland D, Lisabeth L, Marelli A, McDermott M, Meigs J, Mozaffarian D, Nichol G, O'Donnell C, Roger V, Rosamond W, Sacco R, Sorlie P, Stafford R, Steinberger J, Thom T, Wasserthiel-Smoller S, Wong N, Wylie-Rosett J, Hong Y, American Heart Association Statistics Committee and Stroke Statistics S. Heart disease and stroke statistics—2009 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation*. 2009; 119(3):480–6.
- Lam CS, Roger VL, Rodeheffer RJ, Borlaug BA, Enders FT, Redfield MM. Pulmonary hypertension in heart failure with preserved ejection fraction: a community-based study. *J Am Coll Cardiol*. 2009;53(13):1119–26.
- Vanderpool RR, Saul M, Nouraie M, Gladwin MT, Simon MA. Association between hemodynamic markers of pulmonary hypertension and outcomes in heart failure with preserved ejection fraction. *JAMA Cardiol*. 2018;3(4): 298–306.
- Assad TR, Hemnes AR, Larkin EK, Glazer AM, Xu M, Wells QS, Farber-Eger EH, Sheng Q, Shyr Y, Harrell FE, Newman JH, Brittain EL. Clinical and biological insights into combined post- and pre-capillary pulmonary hypertension. *J Am Coll Cardiol*. 2016;68(23):2525–36.
- Gerges C, Gerges M, Lang MB, Zhang Y, Jakowitsch J, Probst P, Maurer G, Lang IM. Diastolic pulmonary vascular pressure gradient: a predictor of prognosis in “out-of-proportion” pulmonary hypertension. *Chest*. 2013;143(3): 758–66.
- Galiè N, Humbert M, Vachiery JL, Gibbs S, Lang I, Torbicki A, Simonneau G, Peacock A, Vonk Noordegraaf A, Beghetti M, Ghofrani A, Gomez Sanchez MA, Hansmann G, Klepetko W, Lancellotti P, Matucci M, McDonagh T, Pierard LA, Trindade PT, Zompatori M, Hoeper M. 2015 ESC/ERS guidelines for the diagnosis and treatment of pulmonary hypertension: The joint task force for the diagnosis and treatment of pulmonary hypertension of the European Society of Cardiology (ESC) and the European Respiratory Society (ERS): Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPC), International Society for Heart and Lung Transplantation (ISHLT). *Eur Heart J*. 2015;46:903–75.
- McMurray JJ, Adamopoulos S, Anker SD, Auricchio A, Böhm M, Dickstein K, Falk V, Filippatos G, Fonseca C, Gomez-Sanchez MA, Jaarsma T, Køber L, Lip GY, Maggioni AP, Parkhomenko A, Pieske BM, Popescu BA, Rønnevik PK, Rutten FH, Schwitler J, Seferovic P, Stepinska J, Trindade PT, Voors AA, Zannad F, Zeiher A, ESC Committee for Practice G. AESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2012: the Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2012 of the European Society of Cardiology. Developed in collaboration with the Heart Failure Association (HFA) of the ESC. *Eur Heart J*. 2012;33(14):1787–847.
- Pi H, Kosanovich CM, Handen A, Tao M, Visina J, Vanspeybroeck G, Simon MA, Risbano MG, Desai A, Mathier MA, Rivera-Lebron BN, Nguyen Q, Kliner J, Nouraie M, Chan SY. Outcomes of pulmonary arterial hypertension are improved in a specialty care center. *Chest*. 2020;158(1):330–40.
- Sahay S, Melendres-Groves L, Pawar L, Cajigas HR, Pulmonary Vascular Diseases Steering Committee of the American College of Chest P. Pulmonary hypertension care center network: improving care and outcomes in pulmonary hypertension. *Chest*. 2017;151(4):749–54.
- Ponikowski P, Voors AA, Anker SD, Bueno H, Cleland J, Coats A, Falk V, González-Juanatey JR, Harjola VP, Jankowska EA, Jessup M, Linde C, Nihoyannopoulos P, Parissis JT, Pieske B, Riley JP, Rosano G, Ruilope LM, Ruschitzka F, Rutten FH, van der Meer P, ESC Scientific Document G. 2016 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: the Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure of the European Society of Cardiology (ESC) developed with the special contribution of the Heart Failure Association (HFA) of the ESC. *Eur Heart J*. 2016;37(2): 2129–200.



11. Yancy CW, Jessup M, Bozkurt B, Butler J, Casey DE Jr, Colvin MM, Drazner MH, Filippatos GS, Fonarow GC, Givertz MM, Hollenberg SM, Lindenfeld J, Masoudi FA, McBride PE, Peterson PN, Stevenson LW, Westlake C. 2017 ACC/AHA/HFSA Focused Update of the 2013 ACCF/AHA Guideline for the Management of Heart Failure: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Failure Society of America. *J Am Coll Cardiol.* 2017;70(6):776–803.
12. Deaño RC, Glassner-Kolmin C, Rubenfire M, Frost A, Visovatti S, McLaughlin VV, Gomberg-Maitland M. Referral of patients with pulmonary hypertension diagnoses to tertiary pulmonary hypertension centers: the multicenter RePHerral study. *JAMA Intern Med.* 2013;173(10):887–93.
13. Tsujimoto T, Kajio H. Spironolactone use and improved outcomes in patients with heart failure with preserved ejection fraction with resistant hypertension. *J Am Heart Assoc.* 2020;9(23):e018827.
14. Guazzi M, Vicenzi M, Arena R, Guazzi MD. Pulmonary hypertension in heart failure with preserved ejection fraction: a target of phosphodiesterase-5 inhibition in a 1-year study. *Circulation.* 2011;124:164–74.
15. Opitz CF, Hoeper MM, Gibbs JS, Kaemmerer H, Pepke-Zaba J, Coghlan JG, Scelsi L, D'Alto M, Olsson KM, Ulrich S, Scholtz W, Schulz U, Grünig E, Vizza CD, Staehler G, Bruch L, Huscher D, Pittrow D, Rosenkranz S. Pre-capillary, combined, and post-capillary pulmonary hypertension: a pathophysiological continuum. *J Am Coll Cardiol.* 2016;68(4):368–78.
16. Nassif ME, Qintar M, Windsor SL, Jermyn R, Shavelle DM, Tang F, Lamba S, Bhatt K, Brush J, Civitello A, Gordon R, Jonsson O, Lampert B, Pelzel J, Kosiborod MN. Empagliflozin effects on pulmonary artery pressure in patients with heart failure: results from the EMBRACE-HF trial. *Circulation.* 2021;143:1673–86.

**How to cite this article:** Kosanovich CM, Pi H, Handen A, Schikowski E, Chen Y, Thoma FW, Mulukutla S, Koscumb S, Nouraie M, Chan SY. Improved hospitalization rates in a specialty center for heart failure with preserved ejection fraction and pulmonary hypertension. *Pulmonary Circulation.* 2022;12:e12002.

<https://doi.org/10.1002/pul2.12002>