# Individualizing heart failure core measures and use of beta blockers in severe pulmonary hypertension-a teachable moment 

Waqas T Qureshi ${ }^{1 *}$, Thong Phan Nguyen ${ }^{1}$, Mouaz H Al-Mallah ${ }^{\mathbf{2}}$<br>${ }^{1}$ Division of Cardiovascular Medicine, Department of Internal Medicine, Wake Forest University School of Medicine, Winston Salem, NC, USA<br>${ }^{2}$ King Abdul-Aziz Cardiac Center, King Abdul-Aziz Medical City, Ministry of National Guard-8 Health Affairs, Riyadh 11426, Kingdom of Saudi Arabia


#### Abstract

A 73-year old African-American woman with history of hypertension, diabetes mellitus, liver cirrhosis, atrial fibrillation and diastolic heart failure presented with a one-week history of shortness of breath, increasing dyspnea, eight-pound weight gain, and progressive weakness. She had associated orthopnea, paroxysmal nocturnal dyspnea, and leg swelling. She denied fevers, cough, chills, hemoptysis, recent hospitalization or wheezing. Her initial vitals were: blood pressure $194 / 127 \mathrm{mmHg}$, heart rate 74 beats per minute (bpm), respiratory rate 22 and she was afebrile. Examination revealed elevated jugular venous pressure 15 cm above sternal notch at 45 degrees, loud P2, right ventricular heave but regular rate and rhythm. There was a holosystolic murmur grade II/VI present at the left lower sternal border consistent with regurgitation murmur, which increased with inspiration. Initial electrocardiogram showed sinus rhythm with occasional premature ventricular complexes with predominant rightward axis and echocardiogram demonstrated elevated right ventricular systolic pressure of $102 \mathbf{m m H g}$ indicative of severe pulmonary hypertension. The left ventricular ejection fraction was preserved.


Keywords: Heart failure; Pulmonary; Hypertension; Orthopnea; Dyspnea; Hemoptysis.
Accepted on March 13, 2017

## Story from the Front Lines

A 73-year old African - American woman with history of hypertension, diabetes mellitus, liver cirrhosis, atrial fibrillation and diastolic heart failure presented with a one-week history of shortness of breath, increasing dyspnea, eight-pound weight gain, and progressive weakness. She had associated orthopnea, paroxysmal nocturnal dyspnea, and leg swelling. She denied fevers, cough, chills, hemoptysis, recent hospitalization or wheezing. Her initial vitals were: blood pressure 194/127 mmHg , heart rate 74 beats per minute (bpm), respiratory rate 22 and she was afebrile. Examination revealed elevated jugular venous pressure 15 cm above sternal notch at 45 degrees, loud P 2 , right ventricular heave but regular rate and rhythm. There was a holosystolic murmur grade II/VI present at the left lower sternal border consistent with tricuspid regurgitation murmur, which increased with inspiration. Initial electrocardiogram showed sinus rhythm with occasional premature ventricular complexes with predominant rightward axis and echocardiogram demonstrated elevated right ventricular systolic pressure of 102 mmHg indicative of severe pulmonary hypertension. The left ventricular ejection fraction was preserved. Her initial brain natriuretic peptide was $764 \mathrm{pg} / \mathrm{dl}$. Treatment with intravenous diuretics was initiated with good response. Once euvolemia was achieved on the day of her planned discharge, her morning blood pressure was 140/93 mmHg . Since performance measures include use of betablocker in heart failure patients, just prior to discharge 120 mg of extended release propranolol once daily was initiated. The
patient subsequently developed sinus bradycardia with HR of 33 bpm later in the day and complained of severe dizziness. Intravenous glucagon was administered to reverse the beta blocking effects. However, the patient did not respond. A repeat dose did not improve the heart rate. A temporarytransvenous pacemaker was placed with the diagnosis of sick sinus syndrome was established. Permanent pacemaker was scheduled to be placed after an overnight fast. In the morning, the patient's blood pressure was 132/107 mmHg . During the procedure, just after administration of midazolam 2 mg for sedation, the patient developed pulseless electrical activity. She was resuscitated and was intubated. An emergent echocardiogram showed estimated right ventricular systolic pressure of 140 mm Hg with severe right ventricular hypokinesis. The temporarytransvenous pacemaker was replaced with a temporary external pacemaker with a pacing rate of 100 bpm . An epinephrine drip was started and the patient was admitted to the ICU. Inhaled epoprostenol and intravenous milrinone were administered to treat acute right ventricular failure and to reduce right ventricular afterload. Over the course of next few days, patient's hemodynamics improved and she had an unremarkable recovery.

## Teachable Moment

Centers for Medicare and Medicaid services (CMS) launched the National Hearth Failure Project (NHF) in 1999 in an effort to improve quality of care for Medicare beneficiaries with heart failure. This project focused on four inpatient processes of care

Citation: Waqas T Q, Thong P N, Mouaz H Al. Individualizing heart failure core measures and use of beta blockers in severe pulmonary hypertension-a teachable moment. Currn Trend Cardiol. 2017;1(1):19-20.
[1]. The Joint Commission later on harmonized these measures as the measures of inpatient care that they utilize for hospital accreditation. As part of "pay for reporting" policy, hospitals had to submit these "core" measures for CMS reimbursement and for Joint Commission accreditation [2]. Later on, the American College of Cardiology and American Heart Association developed a broader group of performance measures similar to the ones identified by CMS [3]. Betablocker use at the time of discharge is an important part of these performance measures. Since the development of these performance measures, the relationship between performance measures and important patient outcomes has remained controversial [4]. There has been a consistent effort to improve use of beta-blocker and comorbidities may modify the risks associated with these relatively benign medications. In patients with severe pulmonary hypertension, bradycardia is poorly tolerated and could lead to acute decompensation of the right ventricle with acute rise in pulmonary artery pressure. The right ventricle poorly tolerates acute rises in pulmonary pressures. Patients with severe diastolic heart failure have withdrawal of beta-blocker therapy [5]. In addition, a case presentation revealed an episode of cardiac arrest following metoprolol [6]. Recently, a study of bisoprolol used in pulmonary hypertension patients showed no benefit, but instead decreased cardiac index and walking distance [7]. Pulmonary pressures in patients with diastolic heart failure should be considered as an important data point in decision making for medical management of heart failure. An echocardiogram with the aim to evaluate right ventricle is a necessary component for if done appropriately could help in providing information about pulmonary hypertension. This clinical case highlights the importance of consideration of right ventricle function and pulmonary pressures in decision making for medical management of heart failure. It also shows that the performance measures of heart failure are guidance for clinical practitioners and are not set in stone to be followed in all patients. In conclusion, beta-blocker use should be weighed cautiously against the long-term benefit in patients with diastolic heart failure and severe pulmonary hypertension. Hence, the performance measures are not applicable in all the heart failure patients.

## Acknowledgement

The authors have no financial conflicts. The authors will like to acknowledge and thank the patient who has read the manuscript and have approved its publication.

## References

1. Masoudi FA, Ordin DL, Delaney RJ, et al. The National Heart Failure Project: a health care financing administration initiative to improve the care of Medicare beneficiaries with heart failure. Congest Heart Fail. 2000; 6(6): 337-9.
2. Fonarow GC, Peterson ED. Heart failure performance measures and outcomes: real or illusory gains. JAMA. 2009; 302(7): 792-4.
3. Spertus JA, Eagle KA, Krumholz HM, et al. American College of Cardiology and American Heart Association methodology for the selection and creation of performance measures for quantifying the quality of cardiovascular care. Circulation. 2005; 111(13): 1703-12.
4. Hernandez AF, Hammill BG, Peterson ED, et al. Relationships between emerging measures of heart failure processes of care and clinical outcomes. American heart journal 2010; 159(3): 406-13.
5. Provencher S, Herve P, Jais X, et al. Deleterious effects of beta-blockers on exercise capacity and hemodynamics in patients with portopulmonary hypertension. Gastroenterology. 2006; 130(1): 120-6.
6. Peacock A, Ross K. Pulmonary hypertension: a contraindication to the use of $\{b e t a\}$-adrenoceptor blocking agents. Thorax. 2010; 65(5): 454-5.
7. van Campen JS, de Boer K, van de Veerdonk MC, et al. Bisoprolol in idiopathic pulmonary arterialhypertension: an explorative study. The European respiratory journal. 2016; 4: 5.

## *Correspondence to

Waqas T Qureshi
Department of Internal Medicine
Wake Forest University School of Medicine
United States of America
E-mail: Wqureshi@wakehealth.edu

