Giant right ventricle secondary to severe pulmonary hypertension

Kunal Kishor Jha,¹ Nuwadatta Subedi,² Durgesh Prasad Chaudhary,³ Manoj Lamsal⁴

¹Department of Critical Care Medicine, Geisinger Medical Center, Danville, Pennsylvania, USA

²Gandaki Medical College, Pokhara, Nepal

³Department of Internal Medicine, Norvic International Hospital, Kathmandu, Nepal

⁴Department of Internal Medicine, Tribhuvan University Institute of Medicine, Maharajgunj, Nepal

Correspondence to

Dr Kunal Kishor Jha,
kunaljhamd@gmail.com

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DESCRIPTION

A man aged 64 years presented to emergency department with chest pain and shortness of breath. On cardiac auscultation, he had loud P2 and parasternal heave; however, lung auscultation was normal. Bilateral lower limb pitting oedema was present. CBC, CMP and cardiac biomarkers were normal. D-dimer and CT chest were performed and it excluded pulmonary embolism. His EKG was suggestive of right ventricular strain. He was being treated for systemic and pulmonary hypertension, taking carvedilol 3.125 mg two times per day, furosemide 40 mg once daily, sildenafil 20 mg three times a day, treprostinil 0.5 mg two times per day, amlodipine 2.5 mg two times per day, atorvastatin 40 mg once daily, aspirin 81 mg once daily and clopidogrel 75 mg once daily. Trans-thoracic echocardiography showed his systolic function was normal, his estimated ejection fraction was 60–65% with markedly dilated right ventricle (RV), no valve defect was detected and peak pulmonary artery pressure (Ppa) was 80 mm Hg. On coronary angiography, there was 60%, 50% and 60% stenosis detected in the left main artery, left anterior descending and right coronary artery, respectively. On cardiac catheterisation, RV was massively dilated and hypokinetic, the left ventricle (LV) was normal in size and function with preserved ejection fraction (figure 1, videos 1 and 2), his pulmonary artery pressure was 76 mm Hg. On cardiac catheter haemodynamics, his mean right atrial pressure was 64 mm Hg, RV pressure was 66/20 mm Hg and pulmonary artery wedge pressure was 17/14 mm Hg. On echocardiography, tricuspid annular plane

systolic excursion was <0.9 cm, and TR jet velocity was 3.08 m/s. An apical four-chamber view shows dilation of RV (video 3). NT-proBNP value was 578 pg/mL. Symptomatic management with oxygen, intravenous fluid and home medications were continued. Owing to severe pulmonary hypertension and enlarged RV, atrial septostomy was offered. He refused to undergo surgery or consultation and went to another institution for further evaluation.

Normal RV have a thinner wall, are crescent shape and have greater compliance than the LV. RV enlargement is defined as the RV being greater than the two-thirds the size of the LV on the apical four-chamber view of echocardiography.¹ Pulmonary arterial hypertension (PAH) is associated with changes in the pulmonary vasculature and subsequently RV. The severity of symptoms and prognosis of PAH are strongly associated with RV function.²

In milder forms of PAH, the reduction in pulmonary vascular resistance (PVR) has beneficial effect on the RV, but these effects may not occur in severe PAH.² Although current pharmacological treatments of PAH reduce RV load, they do not reduce its pressure or output and RV failure may continue to progress, especially in those with severe PAH. Various haemodynamic factors such as mean Ppa, right atrial pressure, cardiac output and cardiac index are directly related to RV function and it has been identified as significant predictors of mortality.³ Despite its significant clinical implications, these factors and the effects of current PAH specific therapies on the RV have not garnered

Figure 1 Massively dilated right ventricle and normal left ventricle can be seen on right and left heart catheterisation, respectively.

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Video 1 Left ventricle on left heart catheterisation.

Video 2 Giant right ventricle on right heart catheterisation.

enough research interest.² Greater consideration for means of assessing the presence of overt and subclinical RV failure and strategies to support the RV in PAH is required.

Video 3 An apical four-chamber view showing right ventricle dilation.

Learning points

- ▶ Pulmonary arterial hypertension (PAH) is associated with changes in the pulmonary vasculature and subsequently right ventricle (RV). The severity of symptoms and prognosis of PAH are strongly associated with RV function.
- ▶ Although current pharmacological treatments of PAH reduce RV load, they do not reduce its pressure or output and RV failure may continue to progress.
- ▶ Greater consideration for means of assessing the presence of overt and subclinical RV failure and strategies to support the RV in PAH is required.

Contributors KJ compiled the case history and investigations, critically reviewed and prepared the manuscript. NS, DPC and ML analysed, critically reviewed the data and prepared the manuscript.

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