

Left atrial unloading with an 8 mm septal cutting balloon to treat postcapillary pulmonary hypertension: a case report

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Abstract

We report the case of a 58-year-old female with severe postcapillary pulmonary hypertension (averaged mean pulmonary arterial pressure was 49 mmHg, pulmonary arterial wedge pressure 29 mmHg, and right atrial pressure 8 mmHg) due to heart failure with preserved ejection fraction. A left-to-right atrial shunt was created using an 8 mm cutting balloon, under transesophageal echocardiography guidance. Both pulmonary arterial and wedge pressure dramatically decreased after the procedure. Symptoms immediately improved and benefits were sustained at 6 months of follow-up. This case suggests that iatrogenic septal defect using a cutting balloon could be an option to treat symptomatic postcapillary pulmonary hypertension.

Keywords Pulmonary hypertension; Heart failure; Left atrium; Cutting balloon

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Introduction

Heart failure (HF) is a frequent syndrome associated with an increasing rate of admission to hospitals and a poor survival rate.¹ With a prevalence varying from about 40–80% among registries, cut-offs, phenotypes, and diagnostic tools, postcapillary pulmonary hypertension (PcPH) is one of the most frequent complications that worsens HF morbimortality.^{2,3}

Treating PcPH seems to be a serious option for HF patients at an early stage of the disease to improve their quality of life and to reduce symptoms mainly at exertion, as well as at advanced HF stages to reduce cardiovascular hospitalizations and death.^{2,4} Several randomized control trials have been performed using either endothelin-1 receptor antagonists or prostacyclin or phosphodiesterase type 5 inhibitors or soluble guanylate cyclase stimulators in PcPH patients; nevertheless, they failed to demonstrate routine use of vasodilation therapy for PcPH patients.⁴

At present, PcPH management is far from satisfactory mainly because research has focused on arterial vascular remodelling forgetting that PcPH is primarily related to an increase in left atrial (LA) pressure.

Case report

We report on the case of a 58-year-old female with a history of congenital aortic stenosis treated by aortic valve commissurotomy in 1972 then surgically converted to a Ross procedure in 2010 when her symptoms reoccurred. In 1998 and 2010, she underwent mastectomy, radiotherapy, and chemotherapy for bilateral breast cancer. In the context of chemotherapy, left ventricular (LV) ejection fraction was serially monitored. Her last examination reported biplane Simpson's rule LV ejection fraction above 50%, a 6 mm septal and posterior wall thickness, an early to late transmitral velocity E/A ratio of 2.9 with an averaged mitral septal, and lat-

eral early tissue Doppler velocities e' of 6.5 cm/s giving E/e' ratio of 24.5, which is consistent with the diagnosis of type III diastolic dysfunction.⁵ LA volume index was 33 mL/m², while right ventricular area was 21 cm² with normal longitudinal systolic function (tricuspid annular plane systolic excursion = 17 mm and systolic tricuspid tissue velocity = 11 cm/s). Due to a refractory New York Heart Association (NYHA) functional class III, and suspected signs of PcPH evaluated by echocardiography (i.e. tricuspid regurgitation peak velocity of 3.9 m/s), she has been referred to our HF-pulmonary hypertension centre.

Transoesophageal echocardiography (TEE) ruled out significant mitral regurgitation, while a computed tomography pulmonary angiography disregarded pulmonary venous stenosis or other lung disease including chronic thromboembolic pulmonary hypertension. Stiff LA syndrome was excluded by invasive right heart catheterization, with no gradient found between pulmonary arterial wedge pressure (PAWP) and LV end-diastolic pressure.

A right heart catheterization was performed at rest. PAWP, mean pulmonary arterial pressure (mPAP), and right atrial pressure (RAP) were 29, 49, and 8 mmHg, respectively. Cardiac index averaged 2.8 L/min/m², giving pulmonary vascular resistance (PVR) of 4.6 WU (Figure 1). Finally, heart failure with preserved ejection fraction (HFpEF) related to valvular and radiation-induced restrictive cardiomyopathy was the most likely cause of PcPH in this case.

Due to an important PAWP to RAP pressure gradient, it was decided to create a left-to-right atrial shunt. The procedure took place under general anaesthesia with TEE and haemodynamic invasive monitoring, both from right heart catheterization and arterial blood pressure recording. With the biplane transoesophageal modality, a first transeptal puncture was performed in the mid-fossa ovalis using an NRG-radiofrequency transeptal needle interfacing with

the Baylis Medical Radiofrequency Puncture Generator (RFP-100-115). By transoesophageal echocardiography, the interatrial septum was 2 mm thickened. Prior to introducing the cutting balloon, a 3 mm balloon was inflated in the septal orifice. A 6 mm and then an 8 mm cutting balloon was positioned in the hole, inflated and deflated every 5 s, and rotated clockwise every 60° (Figure 2). All materials were

Figure 2 Transoesophageal echo-guided procedure. (A) Echo-guided transeptal puncture in the mid-fossa ovalis; (B) advancement of a Schwartz catheter in the left atrium; (C) stabilization of the stiff guidewire positioned into the left ventricle; and (D) a fossa ovalis circular section with 2 consecutive cutting balloon inflations, with a 60° clockwise rotation obtaining 6 radial cuts.

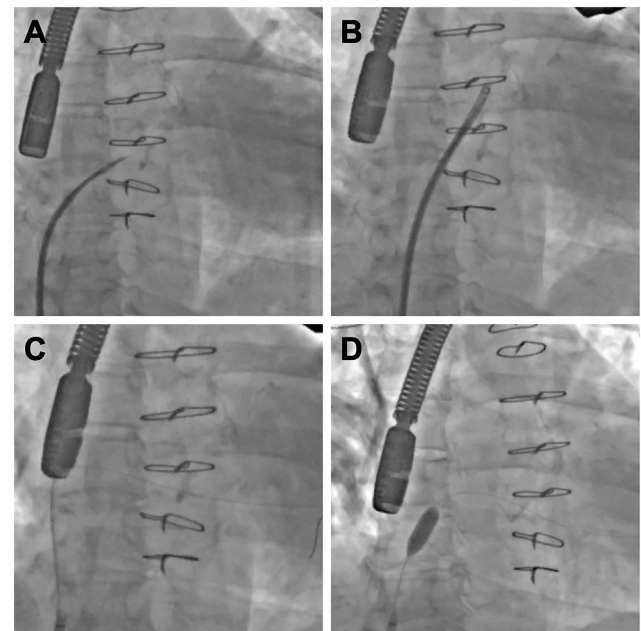
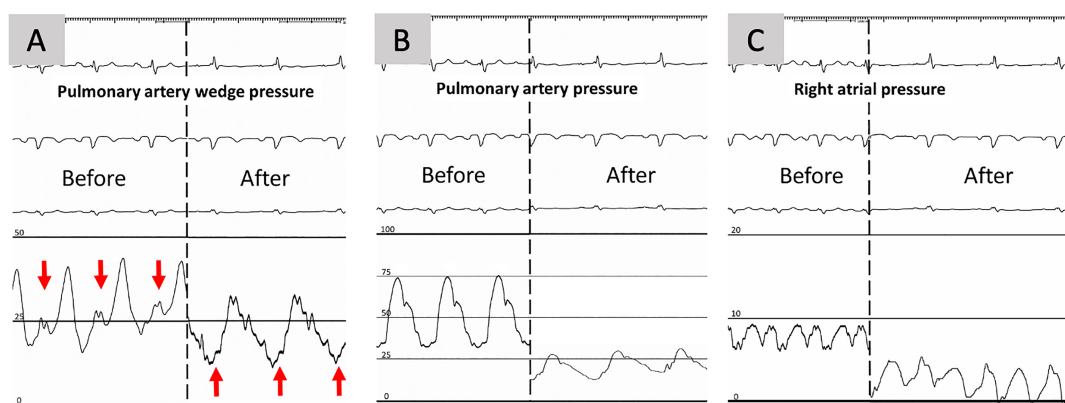


Figure 1 Right heart catheterization. (A) Pulmonary arterial wedge pressure (PAWP), (B) pulmonary artery pressure, and (C) right atrial pressure (RAP) before and after opening the interatrial septum. All measurements were taken at end-expiratory period. Note the significant drop in pulmonary artery pressure. Despite left-to-right atrial pressure and volume unloading, unexpected low RAP is observed in response to the left and downward shift of the right ventricular pressure-volume loop. Red arrows indicate PAWP.



removed once the procedure was completed. TEE confirmed a 6/7 mm septal defect with a unidirectional left-to-right shunt (Figure 3). Continuous-wave Doppler interrogation revealed a drop in the left-to-right atrial pressure gradient, while the patient's post-procedure right heart catheterization demonstrated an important pulmonary hypertension decrease when compared with the baseline (Figures 1 and 3).

At follow-up, the shunt was unidirectional and persistent, and the patient reported functional improvement, while transthoracic echocardiography was consistent with LV diastolic function enhancement. Baseline, 1 month, and 6 months of follow-up NYHA was 3, 2, and 2, respectively, while 6 min walking distance was 485, 486, and 450 m, respectively, and N-terminal pro-brain natriuretic peptide was 7273, 6160, and 5559 pg/mL, respectively.

Discussion

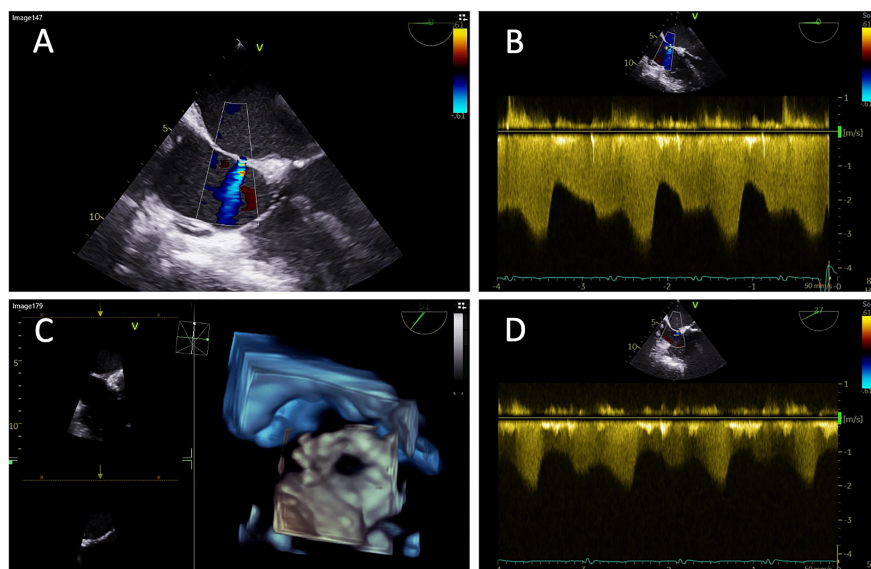
Patients with PcPH are defined as having both a PAWP > 15 mmHg and an mPAP > 20 mmHg.⁴ While PcPH—the most frequent form of pulmonary hypertension—negatively influences morbimortality,⁶ specific PcPH therapy does not exist.

Treatment of symptomatic PcPH due to HFpEF is still challenging, focused on PVR decrease. Several randomized controlled trials have tested specific pulmonary vasodilation therapies without any proven benefit.⁴ Yet, the main target remains the passive backward transmission of elevated LV

filling pressures to the pulmonary circulation. Unloading the LA with diuretics, or even better with LV assist devices, has proven to be the most efficient strategy for normalizing PVR, particularly in PcPH patients, as a bridge for heart transplantation.^{7–10} Further, in the Lutembacher syndrome, patients with mitral stenosis are less symptomatic because of a left-to-right atrial shunt. Thus, clinicians are now reconsidering interventional procedures to normalize left-sided filling pressures by creating an iatrogenic atrial septal defect. Permanent implants (e.g. the V-wave, Corvia's InterAtrial Shunt Device, and Occlutech's Atrial Flow Regulator) and 'leave nothing behind' procedures (e.g. Venus NoYA, atrial balloon septostomy, radiofrequency, and Vardi endocardial septostomy) are used to open the interatrial septum, with orifices varying from 5.1 to 14 mm. All have been investigated in HFpEF patients with provokable high LV filling pressure during exercise but not to treat PcPH.

Here, we report the first short-term results of an iatrogenic atrial septal defect created to decrease LA pressure to treat symptomatic PcPH due to HFpEF. To reach a consistent drop in pulmonary arterial pressure, the patient was strictly selected for high LA pressure > 25 mmHg at rest, important left-to-right atrial pressure gradient (PAWP-RAP) > 15 mmHg, and normal right ventricular function assessed by echocardiography. We used an 8 mm cutting balloon because in computer simulation, this diameter provides good results without overloading the right cavities.¹¹ Furthermore, the larger the pressure gradient between both atria, the greater the PAWP-RAP gradient reduction.¹²

Figure 3 Left-to-right atrial shunt visualized in transoesophageal echocardiography. (A) Atrial septal defect by transoesophageal echocardiography with colour Doppler; (B) transatrial pressure gradient immediately after the first dilatation and (D) decreased at the end of the procedure; (C) the yellow arrow shows the residual 6 × 7 mm hole.



Devices and ‘leave nothing behind’ procedures enabling a left-to-right atrial shunt are worth investigating in PcPH. To the best of our knowledge, no studies have explored this opportunity, likely because PcPH patients are often excluded from trials. ‘Leave nothing behind’ procedures like the cutting balloon have numerous advantages over other devices, including low cost, no risk of endocarditis, and, above all, the possibility for percutaneous closure in case of paradoxical embolism.

Conclusions

This case suggests that the use of an iatrogenic septal defect with a cutting balloon could be an efficient way to treat PcPH. Larger ongoing studies are needed to further investigate this possibility.

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Permission

A completed consent form has been filled in by the patient discussed in this case report.

Conflict of interest

None declared.

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