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CLINICAL RESEARCH

Hyperacute flash pulmonary oedema after transcatheter pulmonary valve implantation: The melody of an overwhelmed left ventricle



Œdème aigu du poumon flash après la mise en place d'une valve pulmonaire par voie percutanée

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Summary Percutaneous transcatheter Melody Valve implantation has achieved standard of care for the management of certain patients with right ventricular outflow tract dysfunction. With its widespread use, some rare and potentially fatal complications, such as right ventricular outflow tract rupture and coronary artery compression, have been reported. We report hyperacute flash pulmonary oedema after Melody Valve implantation for the first time in two patients and describe some possible predictors.
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Abbreviations: MRI, magnetic resonance imaging; TPV, transcatheter pulmonary valve.

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MOTS CLÉS

Implantation percutanée d'une valve pulmonaire ; Melody ; Cardiopathies congénitales ; Œdème pulmonaire

Résumé L'implantation d'une valve Melody par voie percutanée est désormais considérée comme une technique standard dans la prise en charge des patients ayant une dysfonction de la valve pulmonaire. Avec son utilisation croissante, des complications rares et potentiellement fatales telles que les ruptures de conduits ou les compressions coronaires commencent à être rapportées. Nous rapportons ici l'apparition d'un œdème du poumon hyperaiguë après valvulation pulmonaire chez deux patients et en décrivons les mécanismes possibles. © 2014 Elsevier Masson SAS. Tous droits réservés.

Background

Transcatheter pulmonary valve (TPV) replacement was reported in an animal model and then in humans for the first time in the year 2000 [1–3]. Percutaneous TPV implantation (Melody Valve; Medtronic, Minneapolis, MN, USA) is now an established therapy for the management of dysfunctional, incompetent and obstructed right ventricular outflow tract in eligible patients [4–6]. Excellent early, mid-term and even long-term success rates have been reported [7,8]. Coronary artery compression, conduit ruptures and pulmonary artery injury resulting from wire perforation and stent fracture are some procedural complications reported with TPV implantation [9–11]. We report unusual cases of hyperacute flash pulmonary oedema following uncomplicated TPV implantation in patients with standard indications and normal left ventricular systolic function.

Case 1

A 15-year-old male (weight 61 kg; height 165 cm) born with congenital stenotic bicuspid aortic valve underwent surgical aortic valve commissurotomy during the neonatal period. Subsequently, he underwent several palliative procedures, including percutaneous aortic dilation at 9 months and surgical mitral valve annuloplasty with cleft closure at 4 years. At 5 years of age he underwent a third balloon aortic valve dilation.

Owing to relentless progression despite balloon valvuloplasties, with high left ventricular pressures (190/0/18 mmHg), a transaortic valvular gradient of 80 mmHg and moderate insufficiency (aortic annulus 19 mm), he underwent the Ross procedure, with placement of a pulmonary homograft (16 mm), at 9 years of age; he also received optimised medical management with aspirin and beta-blockers.

Six years after the Ross procedure the patient presented with exertion dyspnoea and suprasystemic right ventricular systolic pressures of 125 mmHg, in contrast to a systemic pressure of 110 mmHg. Further evaluation showed severe calcific stenosis of the pulmonary homograft, with a maximum velocity of 6.8 m/s, a well-preserved left ventricular ejection fraction (70%), trivial mitral valve insufficiency, mild aortic valve insufficiency and poor exercise VO_2 max. Cardiac magnetic resonance imaging confirmed these

findings and cardiac catheterization showed a right ventricular end-diastolic pressure of 13 mmHg and a right ventricular systolic pressure of 110 mmHg, with 75 mmHg in the aorta during systole. Moreover, pulmonary pressure remained normal at 24/12 mmHg, with a mean of 16 mmHg. After ruling out possible coronary artery compression, the patient underwent dilation and stenting of the right ventricular outflow tract with a CP8Z34 stent mounted on a 20 mm BIB balloon (Numed Inc., Hopkinton, NY, USA). The immediate results were very gratifying, with a reduction in right ventricular to pulmonary artery pressure gradient. After 3 months, the patient underwent staged Melody Valve (22 mm) insertion. The Melody Valve was well deployed, with no complications or coronary artery compression and postprocedure haemodynamics showed a right ventricular pressure of 30/0/10 mmHg and a pulmonary artery pressure of 30/14 mmHg (mean 20 mmHg). The mean pulmonary capillary wedge pressures before and after Melody Valve insertion were 8 and 12 mmHg, respectively. During the 2-hour procedure, the patient received 1000 mL of serum and 80 mL of contrast dye. No volume expansion was needed during the catheterization.

Forty-five minutes after leaving the catheterization lab, while still in the recovery room, the patient developed sudden shortness of breath, coughing out massive amounts of pink frothy sputum, which made intubation very difficult, despite continuous suctioning. At this time, his systemic blood pressure was in the normal range (110/60/75 mmHg). Saturation dropped to 70% and a few litres of pink frothy fluid were suctioned in a brief period of time. The patient was finally intubated and ventilated using jet ventilation. During this time he coded with bradycardic cardiac arrest, but was successfully resuscitated with pressor support. A chest X-ray confirmed signs of severe cardiogenic pulmonary oedema (Fig. 1). After 5 days, the patient improved gradually with liberal diuresis and ventilation with positive end-expiratory pressure; he was extubated, watched for a day in the step down, and was subsequently mobilized and discharged from telemetry on aspirin and diuretics. Transthoracic echocardiography before discharge showed a well-seated transcatheter pulmonary valve with a reduction in right ventricular systolic pressure to 35 mmHg, a right ventricular to pulmonary artery maximum velocity of 2.3 m/s, good left ventricular systolic function and no signs of myocardial ischaemia. Cardiac magnetic resonance imaging (MRI) on subsequent follow-up at 3 years showed normal volumes and function of the right ventricle.

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