Hemodynamic rounds: Can we mimic a temporary pulmonary artery band in catheterization laboratory in corrected transposition of great arteries with severe tricuspid regurgitation?

Avinash Anantharaj, Kothandam Sivakumar
Department of Pediatric Cardiology, Institute of Cardiovascular Diseases, Madras Medical Mission, Chennai, Tamil Nadu, India

ABSTRACT

Right ventricular (RV) geometry is altered by septal shift after pulmonary artery banding. This may reduce tricuspid regurgitation (TR) and improve ventricular function in patients with corrected transposition of great arteries and systemic right ventricle. However, banding is risky in sick patients with severe RV failure. There are no predictive models in clinical practice to test this septal shift hypothesis before a risky surgery. A transcatheter model to mimic a pulmonary artery band is presented in corrected transposition of great arteries with failing right ventricle and severe TR.

Keywords: Corrected transposition, pulmonary artery banding, right ventricular geometry, septal shift, systemic right ventricle, transcatheter technique

INTRODUCTION

Natural history of congenitally corrected transposition of great arteries (CCTGA) in the absence of an associated ventricular septal defect (VSD) and left ventricular outflow tract obstruction is dependent on progressive tricuspid regurgitation (TR) and right ventricular (RV) failure.[1] When there is severe TR, prolonged survival is uncommon; the mean time from the occurrence of severe TR to onset of RV failure is 5 years.[1] As septal tricuspid papillary muscle chords attach to the interventricular septum, altered septal geometry causes systolic septal bowing leading to separation of the tricuspid leaflets.[2,3] Progressive RV dilatation and weak annular fibrous skeleton further aggravate the TR and RV failure.[4] Following anatomical repair or pulmonary artery banding (PAB), increase of the left ventricular pressure leads to the reduction of TR and improves RV function.[5] PAB is recommended in (a) prevention of RV failure before its occurrence, (b) correction of established RV failure, and (c) palliation of lesions not amenable for anatomic repair.[6] However, improvements are not uniformly observed in all patients, especially in older age group, dysplastic valves, and markedly dilated annulus.[7]

Based on the septal shift hypothesis, theoretic predictors of improvement of TR following PAB have been (1) sphericity index of both ventricles, (2) tethering distance of tricuspid valve, (3) coaptation height of tricuspid leaflets, (4) severity of TR, and (5) systolic pressure ratio between the two ventricles.[3] PAB remains a risky procedure in a sick patient with severe TR and failing RV. A less invasive temporary model to replicate PAB with a simultaneous hemodynamic assessment of ventricular pressure and pulmonary artery gradients would serve as an ideal tool to predict success in patients before PAB. No such transcatheter models of the creation of temporary PAB have been reported so far.

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Address for correspondence: Dr. Kothandam Sivakumar, Department of Pediatric Cardiology, Institute of Cardiovascular Diseases, Madras Medical Mission, 4-A, Dr. J. J. Nagar, Mogappair, Chennai - 600 089, Tamil Nadu, India. E-mail: drkumarsiva@hotmail.com

HEMODYNAMIC ROUNDS

A 13-year-old boy with situs inversus, dextrocardia, CCTGA, intact atrial septum, restrictive small muscular VSD underwent successful electrophysiological ablation of a peritricuspid concealed bypass tract for episodes of orthodromic atrioventricular reentrant tachycardia at 12 years of age. He had progressive TR and severe RV systolic dysfunction with New York Heart Association (NYHA) class II dyspnea. PAB to induce septal shift was considered to reduce TR severity and improve RV function, but his older age, dilated annulus, and RV dysfunction were considered as possible reasons for non response. To simulate a PAB in the catheterization laboratory, it was planned to partially balloon occlude the pulmonary artery. Hemodynamic data from all the chambers were continuously recorded using three venous and one arterial access under local anesthesia and conscious sedation [Table 1].

A pigtail catheter placed in the left atrium after septal puncture for continuous pressure monitoring showed large "v" waves and elevated mean pressures indicating severe TR [Figure 1]. The elevated RV end diastolic pressure at 20 mmHg suggested a significant RV dysfunction [Figure 2]. Two 12 mm Tyshak valvuloplasty balloons (NuMed Inc., Hopkinton, NY, USA) were inflated just beyond pulmonary annulus across the origins of both branch pulmonary arteries to mimic a PAB and increase the left ventricular pressures to two-thirds systemic pressures [Figure 3]. This led to the reduction in the TR, as shown by reduced left atrial pressures and smaller "v" waves [Figure 4]. The reduction in RV end diastolic pressures indicated improved RV function too. This hemodynamic improvement, which was sustained for more than 20 min of balloon inflation predicted success in PAB strategy. The unaltered aortic and right atrial pressures indicated stable cardiac output during this temporary partial balloon occlusion caused by the 12 mm balloons on branch pulmonary arteries that measured 14 mm each. Fick oximetry run was not utilized for cardiac output assessment due to alterations in shunt ratio after balloon occlusion in the presence of restrictive VSD. Despite poor windows in the setting of dextrocardia, echocardiogram confirmed reduction in the severity of TR during this balloon occlusion and reduction of interventricular gradient across the VSD from 60 to 30 mmHg.

After midline sternotomy, PAB was done using Trussler's rule to increase the left ventricular systolic pressures to two-thirds systemic pressure. The systemic pressures and central venous pressures remained stable during the banding, which resulted in a 40-mmHg gradient across the band and reduction in TR severity. The postoperative course was uneventful. At a follow-up of 2 years, TR continued to remain mild, there were no recurrences of tachycardia, and he remained in NYHA class I without any cardiac medications.

Table 1: Hemodynamic data before and after pulmonary artery balloon occlusion

Site	Basal pressure (mmHg)	Final pressure (mmHg)
Right atrium	a-7, v-10, mean-6	Mean-7
Left atrium	a-17, v-22, mean-15	a-12, v-16, mean-7
Right ventricle	95 (systolic), end diastolic 20	90 (systolic), end diastolic 10
Left ventricle	40 (systolic), end diastolic 12	62 (systolic), end diastolic 10
Pulmonary artery	40/23/33	34/15/25
Ascending aorta	95/61/75	90/53/69

a: height of "a" wave, v: height of "v" wave

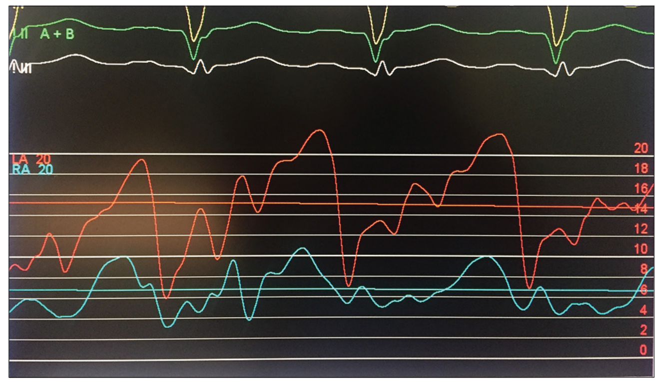

Figure 1: Left and right atrial pressures. Prominent "v" waves and deep v-y descent in left atrium indicated severe tricuspid regurgitation

DISCUSSION

PAB strategy is followed in some patients with CCTGA based on the septal shift hypothesis but is less often clinically practiced due to uncertainty of this concept in all real-world patients.[8,9] Older age, dysplasia of tricuspid leaflets, marked annular dilatation, and myocardial failure are reasons of non response after PAB.[7] Surgical attempts of testing this hypothesis involves gradual banding with monitoring of TR under unphysiological states that include inhalational anesthesia-induced depression and positive pressure ventilation.[10] In an older patient with severe TR and heart failure, no temporary transcatheter models have been published to mimic a PAB with complete hemodynamic assessment in spontaneous breathing without anesthesia. The long-term effect of PAB on TR can neither be predicted based on such surgical testing or our transcatheter models in this complex disease.

An issue encountered in the creation of this temporary model is vulnerability of the conduction tissues during manipulation of catheters and balloons across the left ventricular outflow tract. We positioned two simultaneous balloons to occlude the pulmonary arteries beyond the

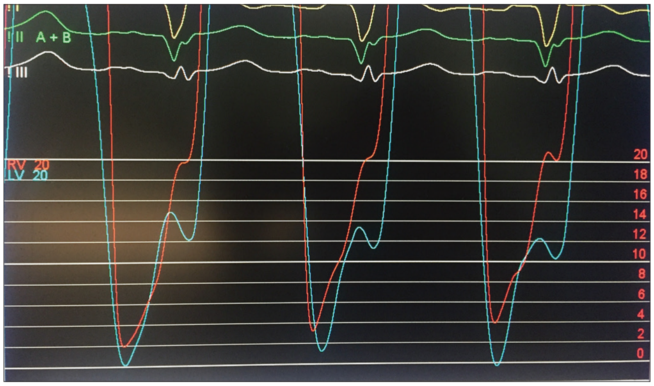

Figure 2: Left and right ventricular pressures. Elevated end diastolic pressure in the right ventricle indicated significant right ventricular dysfunction

Figure 3: Balloon occlusion mimics pulmonary artery banding. A pigtail catheter advanced through a transseptal Mullins sheath monitored left atrial pressures, and the side arm of the Mullins sheath monitored right atrial pressures. A 12 mm Tyshak II balloon advanced through another Mullins sheath into the right pulmonary artery with the tip of Mullins sheath in the left ventricle to monitor ventricular pressures. Another 12 mm Tyshak II balloon was advanced to the origin of the left pulmonary artery. An arterial sheath with pigtail catheter measured right ventricular and aortic pressures

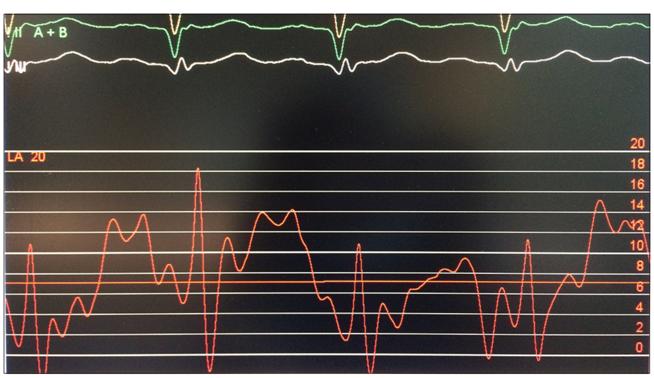

Figure 4: Left atrial pressures after balloon occlusion. There was considerable reduction in "v" waves and left atrial mean pressures indicating significant reduction in tricuspid regurgitation

annular level, thereby avoiding conduction disturbances and pulmonary incompetence. The balloon occlusion should be partial to allow the entire cardiac output through the pulmonary arteries, and this was confirmed by lack of fall of aortic pressures and lack of elevation of the right atrial pressures. Utilizing long Mullins sheaths enabled us to monitor simultaneously the pressures in ventricles and atria, aorta, and pulmonary arteries. Distal pulmonary artery pressures were measured through the lumen of the balloon catheters after guidewire withdrawal. Avoidance of inhalational anesthesia avoided the depression of the systemic pressures and spontaneous respiration maintained hemodynamics through the respiratory cycles. Intraprocedural echocardiogram despite the poor windows in the setting of dextrocardia confirmed reduction in the TR. A transesophageal echocardiogram could have added more insights into the mechanism and severity of TR but at the cost of added sedation and positive pressure ventilation. The hemodynamic improvement on this temporary model gave us the confidence to proceed to PAB and the success of this strategy on TR was shown on the 2-year follow-up.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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