Novel arterioplasty of severe left pulmonary artery stenosis using native main pulmonary artery

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Abstract

The literature describes multiple approaches for the repair of stenosed branch pulmonary arteries. Regardless of the technique, restenosis is undesirably and notoriously common. We describe a case of severe left pulmonary artery stenosis repaired with a novel technique in consideration of factors leading to restenosis. The native main pulmonary artery was transected and turned down to create a direct anastomosis with the left pulmonary artery. The child had a normal sized main pulmonary artery with tricuspid atresia and pulmonary atresia with ductus arteriosus feeding the severely stenosed left pulmonary artery. Our novel technique resulted in hemodynamically gratifying results with a tension free tissue-tissue anastomosis with potential for growth.

Introduction:

Stenosis of pulmonary arteries (PA) is a well-known anomaly associated with other congenital heart defects. It is presumed to occur due to extension of ductal tissue into the wall of the PAs. Although, bilateral branch PA stenosis is known, it more commonly affects the left pulmonary artery (LPA). Surgical repair of branch PA stenosis especially the LPA, regardless of the technique employed, carries a significant incidence of recurrence due to the residual ductal tissue and highlights the importance of removing it from the branch PAs which can sometimes compromise their length. This also makes direct tension-free anastomosis difficult to accomplish. We describe a novel way of enlarging the LPA by employing native, autologous pulmonary arterial tissue while excluding all ductal tissue from the anastomotic stoma.

***Case Report**:*

Institutional review board approval and informed consent from the parents and child were obtained for this case report. An 11-year-old girl, weighing 27 kgs, presented with exertional dyspnea and profound central cyanosis with resting saturations of 70% on room air. Her 2D echocardiography-color Doppler evaluation revealed tricuspid atresia, pulmonary atresia, normally related great arteries with severe LPA stenosis supplied by a stenotic patent ductus arteriosus (PDA). Her computed tomography angiogram confirmed the echo findings with the pulmonary end of the ductus showing significant narrowing and a bifid appearance with each limb supplying the respective branch PAs (Figures 1A, 2). The LPA measured less than 1 mm, 6 mm and 14 mm at its origin, mid segment and hilum respectively while the RPA was uniformly 13mms though its ductal communication was only around 1mm. The main pulmonary artery (MPA) was normal sized and continued as right pulmonary artery (RPA).

Steps of surgery:

The child underwent surgery for enlargement of LPA with creation of bidirectional Glenn (BDG) shunt as a first stage surgery in view of older age at presentation and dilated aortic root. The chest was opened with a midline sternotomy. The RPA pressure was measured directly prior to initiating cardiopulmonary

bypass and was found to be 14 mmHg. Subsequently, cardiopulmonary bypass was established with aorta – innominate vein – right atrial cannulation. The PDA was transfixed and divided at the aortic end.

The MPA was transected and its cardiac end was sutured in 2 layers. The pulmonary end of MPA was mobilized fully by freeing it from the entire ascending aorta. Two marker stitches were placed over the pulmonary end of the MPA to avoid torsion (Figure 1B). The LPA was transected beyond the origin at its mid portion and an incision was made into the pulmonary end of the LPA extending upto the hilum so as to create wide mouth of opening. (Figures 1B, 1C) Thereafter the pulmonary end of MPA was turned down alongside the opened LPA and both stomas were anastomosed using 6/0 continuous prolene sutures (Figures 1D, 3).

Thereafter the BDG shunt was performed in standard fashion. The pressures in the RPA and left atrium were measured after discontinuing bypass and were found to be 14 and 8 mmHg respectively. The patient made an uneventful recovery with resting saturations on room air of 85%. She was discharged on Aspirin 75mg daily. The morphology of pulmonary arterial confluence and branch PAs was evaluated after 6 months using a CT angiogram and was found to be satisfactory (Figure 4) with no evidence of residual or recurrent stenosis.

Discussion:

There are several techniques to create confluence and enlarge the branch PAs. These include enlargement with autologous pericardium (fresh / pre-treated), bovine pericardium, Gore-Tex patch / tube, Dacron grafts, homografts and resection with direct end to end anastomosis[1]. However, all of these techniques have a high rate of restenosis which is largely attributed to extension of ductal tissue into the branch PAs, scarring, fibrosis and cicatrization of autologous pericardium[2,3,4].

Our patient had an unusual presentation. Though the pulmonary arterial supply was sparse, the size of both the branch PAs at the hilum was generous. It is likely that the ductal flow during infancy and early childhood helped the growth of the branch PAs. Subsequently, the PDA gradually constricted along with the constriction of LPA origin resulting in progressive reduction of the pulmonary blood flow causing decreased effort tolerance and worsening cyanosis. Such late presentations are not uncommon in our part of the world.

The resection and end-to-end anastomosis or pericardial patch augmentation would have been conventional wisdom. Instead, we thought of recruiting the normal sized MPA which was lying in close proximity and had no clinical value. The pulmonary end of the transected MPA was turned down as an extension to create a tension free, tissue-tissue anastomosis between the branch PAs with the confluence. This was made possible due to the presence of a normal MPA above the atretic pulmonary valve. In order to avoid torsion while turning down the transected MPA towards the left hilum, two marker sutures were placed in opposing directions for identification after transection of MPA. Being native autologous tissue, it is resistant to infection and calcification, is free from autoimmune responses and has distensibility and potential for growth unlike other biological or prosthetic materials. However, the use of MPA to create a confluence is possible only in a select subset of patients with univentricular hearts or pulmonary atresia where it can be safely sacrificed as was the case in our patient.

***Conclusion**:*

Unlike all the other established techniques, our approach was simple and inexpensive and resulted in gratifying short-term outcome. We anticipate that this repair will be long-lasting since it involved direct tissue to tissue, tension free anastomosis.

Conflicts of interest:

No conflicts of interest declared by any author

Author contribution statement:

Jineel H Raythatha – Conceptualization; Formal analysis; Investigation; Methodology; Project administration; Visualization; Writing – original draft; Writing – review & editing

Bharat Vinayak Dalvi – Methodology; Supervision; Validation; Visualization; Writing – review & editing

Himanshu Choudhury – Methodology, Visualization, Writing – Reviewing & editing.

Krishnanaik Shivaprakasha – Conceptualization; Formal analysis; Methodology; Supervision; Validation; Visualization; Writing – review & editing

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Figure Legends:

Figure 1: Digital Illustration of operative correction

Fig1A: Illustrating pre-operative MPA-LPA non-confluence with 10mm gap. Note the atretic pulmonary valve with no flow from RV to MPA.

Fig1B: Illustrating incisions made and transection of PDA. Arrow lines illustrate the translocation of MPA from A to A1 and B to B1.

Fig1C: Illustrating rotational and translational alignment of transected MPA with LPA.

Fig1D: Illustrating corrected flow and MPA-LPA anastomosis.

Figure 2: Pre-operative Computer Tomography (CT) imaging

Fig 2A: Axial CT image showing severe stenosis (arrow) of LPA (L) with MPA continuing as RPA (R)

Fig 2B: 3D reconstructed CT image demonstrating continuation of MPA (M) as RPA (R) noting severe LPA (L) stenosis (arrow)

Fig 2C: 3D reconstructed CT image showing bifid PDA supplying LPA (L) and RPA (R).

Figure 3: Intra-operative photograph showing the MPA-LPA anastomotic bulb

Figure 4: Post-operative Computer Tomography (CT) Imaging

Fig 4A: Axial CT image showing good patency of MPA-LPA anastomosis and flow 6 months later.

Fig 4B: 3D reconstructed CT image demonstrating MPA-LPA anastomotic bulb (arrow)

Keys: Ao: Aorta; PDA: Patent Ductus Arteriosus; M: Main Pulmonary Artery; MPA: Main Pulmonary Artery, L: Left Pulmonary Artery; LPA: Left Pulmonary Artery; R: Right Pulmonary Artery; RPA: Right Pulmonary Artery; A-PV: Atretic Pulmonary Valve; A: Anterior; P: Posterior.