Novel arterioplasty using native main pulmonary artery to repair nonconfluent left pulmonary artery

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Abstract

There are multiple surgical approaches described to repair the left pulmonary artery (LPA). Regardless of the technique used, rate of restenosis is very high. We describe a case of discontinuous LPA which was treated by turning down the entire autologous main pulmonary artery (MPA) and creating a direct anastomosis between the MPA and LPA. This was in background of tricuspid and pulmonary atresia, hypoplastic right ventricle with atrial and ventricular septal defects and ductus arteriosus feeding the left pulmonary artery. This new technique resulted in a tension free tissue-tissue anastomosis with potential for growth and produced hemodynamically gratifying short-term results.

Introduction:

Stenosis or disconnection of the left pulmonary artery (LPA) 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 LPA. Although, bilateral branch pulmonary artery (PA) disconnection is known, it most commonly occurs unilaterally. Non-confluent PAs are supplied by 3 different sources viz. patent ductus arteriosus from aortic arch system, collateral or bronchial arteries from descending aorta or from a variation of persistent truncus arteriosus[1]. Surgical repair of LPA stenosis / disconnection, regardless of the technique employed, carries a significant incidence of recurrent stenosis due to the presence of ductal tissue and highlights the importance of removing all possible ductal tissue from the LPA which sometimes compromises the length of LPA. This also makes tension-free anastomosis impossible. In order to circumvent these problems, we describe a novel way of restoring the confluence of right pulmonary artery (RPA) with LPA by employing the native, autologous pulmonary arterial tissue itself after removing all ductal tissue to the best extent possible.

***Case Report**:*

Informed consent was obtained from the child and her parents for the purpose of publication of her data. An 11-year-old girl presented to our clinic with exertional dyspnea, severe central cyanosis and digital clubbing with resting saturations of 70% while breathing room air. Her 2D echocardiography-color Doppler evaluation revealed tricuspid atresia, pulmonary atresia, normally related great arteries, absence of confluence with severe LPA stenosis at its origin. Her computer tomography angiogram showed the MPA to be continuous with the RPA without any connection to LPA with only a chord of tissue between both ends measuring about 10 mm. The LPA was supplied by a patent ductus arteriosus. The hilar LPA measured 14mms. (Figure 1A, 2A).

Steps of surgery:

The patient was taken up for a first stage bidirectional Glenn shunt. The chest was opened with a midline sternotomy. Both lobes of the thymus were excised. Cardiopulmonary bypass was established with aorta –

innominate vein – right atrial cannulation. The ductus was transfixed with 2 sutures after division.

The MPA was then transected. The cardiac end was sutured off in 2 layers. MPA continued as RPA. The pulmonary end of MPA was dissected, mobilized fully and then separated from the aorta along its entire axis. 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 to each other with 6/0 continuous prolene sutures (Figure 1D, Figure 3).

Thereafter the bidirectional Glenn shunt was performed in standard fashion. She made an uneventful recovery with resting saturations on room air climbing to 85%. The LPA anatomy and the confluence were evaluated after 6 months using a CT angiogram and was found to be satisfactory (Figure 2B). She is awaiting rapid Fontan completion in view of her age.

Discussion:

There are several techniques described to create confluence and enlarge LPA. Enlargement with autologous pericardium (fresh / pre-treated), bovine pericardium, Gore-Tex patch / tube, Dacron grafts, homografts, resection with end to end anastomosis have been reported[2]. However, all these techniques have a high rate of restenosis which is largely attributed to extension of ductal tissue into the LPA, scarring, fibrosis and cicatrisation of autologous pericardium[3]. Other rare but well-known complications apart from re-stenosis are pulmonary artery-venous fistulas that require re-operation[4,5].

In our patient, the autologous tissue form the MPA was used to accomplish the reconstruction. The pulmonary end of the transected MPA was turned down as an extension to recreate tissue-tissue anastomosis between the pulmonary arteries. In our patient, there was a normal MPA above the atretic pulmonary valve and it continued as a normal RPA. There was fairly good length of MPA that continued as a normal RPA. It was critical that to avoid torsion while turning down the transected MPA towards the left hilum and hence two marker sutures were placed in the opposite directions for identification. Thus, transecting the MPA and turning down to left hilum and connecting it to transected LPA did not pose any difficulty and helped create a tension-free anastomosis. Being native autologous tissue, it is resistant to infection and calcification, free from autoimmune responses, has distensibility and potential for growth unlike pericardium, bovine pericardium, synthetic prosthetic materials. There is no loss of tissue with consequent tension across the anastomosis as well unlike in direct transection and anastomosis in the conventional way. Moreover, it just utilizes the MPA that is present in the area and has no clinical value in these subsets. However, this approach applies to very small subset of patients in whom there is a well formed MPA in the setting of disconnected LPA – not a frequent combination.

Conclusion:

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

Conflicts of Interest:

No conflicts of interest declared by any author

Author contribution statement:

Jineel 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

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

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Figure Legends:

Figure 1: Digital Illustration of pathology and operative correction

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

Fig1B: Illustration of incision sites as well as ligation and transection of patent ductus arteriosus. Lines illustrate the translocation of MPA from points A to A1 and B to B1.

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

Fig1D: Illustration of corrected flow and MPA-LPA anastomosis.

Figure 2: Computer Tomography (CT) imaging

Fig 2A: Pre-operative CT image showing non-confluence of LPA with MPA-RPA.

Fig 2B: Post-operative CT image showing good patency of MPA-LPA anastomosis 4 months later.

Figure 3: Annotated, intraoperative photograph of MPA-LPA anastomosis noting tension free anastomosis. RV and atretic pulmonary valve can be seen along with sutured cardiac end of MPA.

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